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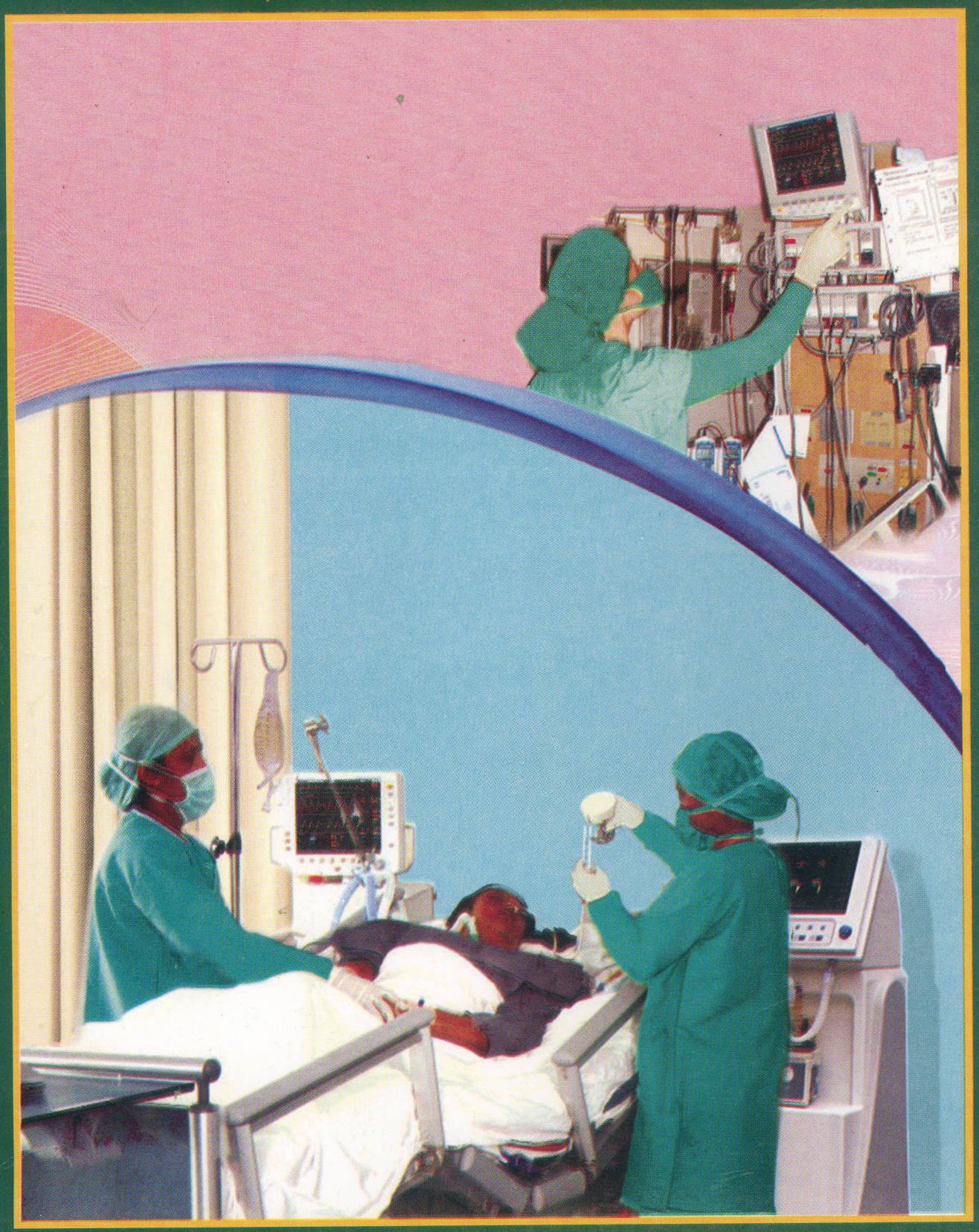
School of Health Sciences

BNS-032
Nursing Management in Critical Care Conditions



CARE OF CRITICALLY ILL PATIENTS WITH PROBLEM OF RENAL AND ENDOCRINE SYSTEM

5

"शिक्षा मानव को बन्धनों से मुक्त करती है और आज के युग में तो यह लोकतंत्र की भावना का आधार भी है। जन्म तथा अन्य कारणों से उत्पन्न जाति एवं वर्गगत विषमताओं को दूर करते हुए मनुष्य को इन सबसे ऊपर उठाती है।"

— इन्दिरा गांधी

"Education is a liberating force, and in our age it is also a democratising force, cutting across the barriers of caste and class, smoothing out inequalities imposed by birth and other circumstances."

– Indira Gandhi

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Block

5

CARE OF CRITICALLY ILL PATIENTS WITH PROBLEM OF RENAL AND ENDOCRINE SYSTEM

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BLOCK INTRODUCTION .

In Blocks 1-4 you have studied the care of critically ill patients with problems of Gastrointestinal, Respiratory, Cardiothoracic and Nervous System. Block 5 deals with problems of renal and endocrinal system that are critical and require intensive Nursing Care. You have already learnt about the various diseases, in basic nursing programme, so emphasis has been given on specific nursing assessment, diagnosis and intervention based on pathology and clinical manifestation of acute and life threatening conditions.

The block has been organized in following 4 units:

Unit 1 Relates to Care of patients with renal transplantation.

Unit 2 Deals with Acute Tubular Necrosis and Bladder Trauma.

Unit 3 Describes Diabetic Ketoacidosis, Hyperglycemic Hyperosmolar nonketotic syndrome and Hypoglycemia.

Unit 4 Focuses on – Thyroid crisis, Myxedema coma, Adrenal crisis, Syndrome of inappropriate hypersecretion of anti diuretic hormone (SIADH).

UNIT 1 CARE OF PATIENTS WITH RENAL TRANSPLANTATION

Structure

1.0 Introduction

1.1 Objectives

1.2 Review of Anatomy and Physiology of the Renal System

1.3 Definition, Indications and Contraindications of Renal Transplantation

- 1.3.1 Definition
- 1.3.2 Indications for Renal Transplantation
- 1.3.3 Contraindications of Renal Transplantation

1.4 Advantages of Renal Transplantation

1.5 Assessment before Renal Transplantation

1.6 Classification of Donors and their Eligibility

1.7 Organ Donation

- 1.7.1 Cadaver Organ Donation
- 1.7.2 Live Organ Donation

1.8 Transplant Surgery

1.9 Management of Patient Undergoing Renal Transplantation

- 1.9.1 Preoperative Nursing Management
- 1.9.2 Post Operative Management
- 1.9.3 Post Operative Health Education and Home Care
- 1.9.4 Complications of Renal Transplantation

1.10 Let Us Sum Up

1.11 Answers to Check Your Progress

1.0 INTRODUCTION

As a GNM / B.Sc. Nurse you have learnt that a patient with chronic renal failure will most often end up with End Stage Renal disease (ESRD). ESRD occurs when both the kidneys of the patient are no longer able to function or keep the patient alive even with the best supportive measures available. The Management of ESRD is lifetime dialysis or renal transplantation. You have already learnt that only dialysis alone can not perform all the functions of the Kidney. Thus transplantation becomes the only viable option. In this unit we shall discuss about definition, indications, contraindication and advantages of renal transplantation. We shall also focus on asseessment, classification of donor and eligibility and management of a patient with renal transplant.

1.1 OBJECTIVES

After completing this unit, you will be able to:

- explain the meaning indications contraindication and advantages of renal transplantation;
- discuss the classification of donors and their eligibility;

- provide care to patients with End Stage Renal disease;
- explain the pre and post operative nursing management of patients with renal transplantation; and
- educate the patient about the complications of renal transplantation and post transplantation home care.

1.2 REVIEW OF ANATOMY AND PHYSIOLOGY OF THE RENAL SYSTEM

As you have learnt in GNM that Urinary tract consist of four organs i.e., kidneys, ureters, bladder and urethra.

Kidneys are located retroperitoneally in posterior aspect of abdomen. They lie between 12th Thoracic vertebrae to 3rd lumber vertebrae. Each kidney has three major parts (i) Cortex (ii) Medulla and (iii) Pelvis. Functional unit of kidney is nephron. Each kidney contains more than one million nephrons. Nephron is a microscopic structure consisting of a glomerulus (Bowman's) capsule and tubular system that empty into renal pelvis and the ureter: (Fig. 1.1)

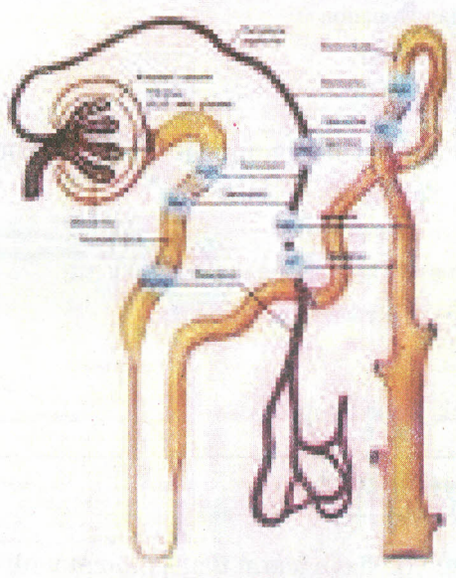

Fig. 1.1: Structure of Nephron

The function of normal kidneys include following:

- Excretion of nitrogenous waste products.
- Maintenance of water balance of the body.
- Regulation of acid base balance and electrolyte balance of the body.
- Excretion of drugs, poisons and unwanted chemicals from the body.
- Regulation of blood pressure of the body.
- Erythropoiesis (make blood)
- Making active Vitamin D which is necessary for bone function.

Urine is formed by nephron by three processes (i) Filtration (ii) Reabsorption and (iii) Secretion.

Filtration is the passage of liquid through a filtering membrane as a result of pressure differential. Reabsorption takes back fluids and other substances through body tissues. Secretion involves the active transport of certain chemicals from the bloodstream in to the tubules. Body regulates fluid and electrolyte balance with these processes.

Kidney also maintains acid-base balances by excreting phosphoric, ketouric acid, and sulfuric acid as they are formed. In the presence of acid base imbalances, kidneys excrete either hydrogen or bicarbonate ions to restore balance. Kidneys also produce rennin which helps in blood pressure regulation.

1.3 DEFINITION, INDICATIONS AND CONTRAINDICATIONS OF RENAL TRANSPLANTATION

In this section we shall focus on definition, indications and contraindication of renal transplantation.

1.3.1 Definition

Kidney transplantation is a surgical procedure to remove a healthy functioning kidney from a living or brain-dead donor and implant it into a patient with nonfunctioning kidneys.

1.3.2 Indications for Renal Transplantation

- End Stage Renal Diseases (ESRD) is one of the main indication for renal transplantation. (*See appendix 1 flowchart of ESRD*)
- Renal transplantation is also indicated whenever bilateral nephrectomy or removal of a solitary functioning kidney is necessary (usually occurs in the case of trauma/or negligence.

1.3.3 Contraindications of Renal Transplantation

- Life expectancy of less than 1 year.
- Recent malignancy.
- Active infection.
- HIV infection.
- Chronic untreated infection.
- Uncontrolled psychiatric disorder.
- Psychiatric disorders that impair the ability to informed consent to adhere to the treatment regimen.
- Active substance abuse.

- ABO blood group mismatch.
- Severe Cachexia
- Severe or uncorrectable
  - coronary disease
  - peripheral vascular disease
  - pulmonary disease
  - cardio myopathy
- Active peptic ulcer diseases.

1.4 ADVANTAGES OF RENAL TRANSPLANTATION

You need to know that the renal transplant has many advantages over dialysis and if you provide care to patient with renal transplant, you can explain this to the patients.

- Renal transplantation patients develop many physiological changes related with renal failure as compared with the dialysis. In other words there is reversal of physiologic changes caused due to renal failure.
- **Higher survival rate:** Patients who undergo kidney transplantation survive longer than those patients who are on periodic dialysis.
- **Better quality of life:** The patient with renal transplantation have better quality of life and are able to live a more independent life because they don't have to receive periodic dialysis.
- **Fewer dietary restrictions:** The patient with renal transplantation have fewer dietary restrictions than patient on dialysis.
- **Cost effective:** It is actually less costly than long term dialysis treatment.

1) Renal transplant is one time management option and is more Cost effective in the long run as compared to periodic dialysis treatment.

Check Your Progress 1

i) Define kidney transplantation. List the indications for renal transplantation.

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ii) What are the advantages of renal transplantation?

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iii) State the contraindication of renal transplantation.

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1.5 ASSESSMENT BEFORE RENAL TRANSPLANTATION

You need to do a detailed body assessment to identify any co-morbid conditions before renal transplantation of the patient which will help to ensure the transplantation success. You need to assess and rule out the problems related with various body systems and functioning and take appropriate measures to correct the problems.

Remember

Prior to Kidney Transplant

If a patient has undergone kidney transplant earlier but transplant has failed so a failed prior kidney transplant patient is not a contraindication to a transplantation.

Age

Advanced age is not a contraindiction to renal transplantation.

Obesity

Obesity increases the risk of postoperative complications and mortality after surgery. You must encourage the obese patient to achieve ideal body weight before transplantation. The large body size results in physiologic changes that have been linked to hyper filtration of the nephron which becomes a risk factor for progression and may cause subsequent pre mature failure of the graft.

Diabetes

Management of the diabetic patient is very important

The success of Renal Transplantation (RT) in Diabetes Mellitus (DM) patient depends on effective diabetic control. Which should be achieved before transplant.

Cardiovascular disease (CVD)

CVD is a major cause of mortality in renal transplant recipients. So evaluation of cardiac status at regular intervals, especially in diabetics and those with increased risk factors for coronary ischemia is very important.

Infections

You have already learnt that the presence of any active infection is a contradiction to transplantation. You must therefore screen the patient properly for the presence of occult infection such as dental abscess and urinary tract infection. Patient with ESRD carry an increased risk of mycobacterial disease and immuno suppressive medication used to prevent organ rejection can lead to reactivation of latent tuberculosis and disseminated diseases.

i) Hepatitis Virus

Of the five major hepatitis virus A, B, C, D and E only B and C have been linked to chronic liver disease in renal transplant patients. HIV infection is highly prevalent in haemodialysis patients.

ii) Malignancy

Many occult tumors have been discovered at the time of transplantation and therefore older patients who have been on the list for lengthy period should be evaluated at regular intervals.

iii) Gastro Intestinal Conditions

Dialysis patients have a high incidence of upper gastro intestinal disease. You have already learnt that Active peptic ulcer disease is a contraindication to transplantation. Acute pancreatitis also results in high morbidity and mortality after transplantation.

iv) Pulmonary Disease

Evaluation of pulmonary status of patients before transplantation should include a forced expiratory volume and assessment of pulmonary risk factors. A screening of chest radiograph is a standard practice and any suspicious lesion should be extensively evaluated. Smoking increases the risk of post transplant malignancy and cardiovascular disease.

v) Psychological Issues

Psychiatric evaluation should be directed to determine the patient's ability to provide informed consent, to adhere to the post transplant immuno suppressive regimen and to the potential for the immuno suppressive regimen to interfere with psychiatric therapy. So a thorough evaluation must be taken.

vi) Immunologic evaluation of transplant recipient

There are three categories of immunologic evaluation in selection of appropriate donor or in determining the prognosis of transplant patients.

- Typing and Antigen Matching
- Serum Screening
- Cross Matching.

Typing and Antigen Matching

The goal of typing and matching histocompatibility antigen between the recipient and donor is to minimize the number of incompatibilities presented by the graft and thus to decrease the likelihood of rejection.

Serum Screening and Cross Matching

The Recipient and donor Human Leukocyte Antigens (HLA) cross match is the most important immunologic test done before transplantation. Six antigens in common are considered a perfect match. Cross matching permits the clinician to determine whether there are preexisting antibodies against donor histocompatibility antigen at the time of transplant or in recent achieved serum samples for the recipient.

ABO Blood Group Matching

In virtually all cases, kidney donors must be ABO blood group (red blood cell antigen) compatible with recipients to ensure successful transplantation. The natural antibodies against A and B blood group bind to vascular endothelial cells which express these antigens.

Cellular Assay for Histocompatibility Testing

The mixed lymphocyte culture is an assay that has historically been obtained to confirm that a halotype matched donor has no incompatibility at the **HLA- DR locus. HLA- DR** serotyping has been technically difficult and **error rites** scan approach 15% to 20%. A negative mixed lymphocytic synthetic culture index ensures the clinician of a positive HLA identified between familial donor-recipients pairs.

1.6 CLASSIFICATION OF DONORS AND THEIR ELIGIBILITY

Donors can be classified in three groups:

a) **Related living donor:** Brother, Sister, Father or mother is the best possible source of kidney donor who match the patient closely.

b) **Other living donors:** Spouse and relatives like cousin, aunt, uncle, friend.

c) **Cadaveric donors:** A clinically brain dead person who is less than 65 years and free of systemic disease, malignancy and / or infections can also be a suitable donor.

Eligibility or requisite for a donor

You have to play a vital role in predonation counseling. You must explain that nephrectomy is a major surgery involving risks of trauma or disease damaging the remaining kidney in the future. You must ensure that—

- Donor must have normal renal function of both kidneys.
- No malignant-disease or generalized infection or abdominal/renal trauma should be present.
- Potential donor must have negative Hepatitis B antigen and negative HIV antibody.
- Must have no problem in ventilation, respiration or cardiac rhythm.
- Donor must have normal BP.
- Donor must have excellent health with two properly functioning kidneys.
- Serum for ABO blood group, tissue specific antigen, Human leukocyte Antigen (HLA) suitability, mixed lymphatic synthetic culture index (histocompatibility).
- Complete emotional wellbeing of the donor should be determined.
- Complete understanding of the patient about donation process and outcome is necessary.

Check Your Progress 2

i) Classify the different types of donors for renal transplantation.

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ii) Enumerate the special evaluation you will perform before Renal Transplantation..

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iii) Enumerate the requisites for a donor in renal transplantation.

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1.7 ORGAN DONATION

In this section we shall discuss about Cadaver organ donation and live organ donation.

1.7.1 Cadaver Organ Donation

Since 2001 the number of families donating cadaver organs and the number of successful transplant have increased and yet the number of people on transplant waiting lists continue to grow and a critical shortage of available organ exists. The situation will continue to worsen and will continue to be primary impediment to success in organ transplantation. The potential cadaver donor is estimated to be 60% to 80%.

Cadaver Donation

i) Cadaver Donor's Evaluation:

- o Identify potential cadaver donor.
- o Inform organ procurement agency.
- o Establish brain death.
- o Obtain consent of donation from family.
- o Prepare cadaver donor with appropriate haemodynamic and respiratory support.
- o Maintain stable electrolyte balance and urine output.
- o Obtain medical history to rule out high risk illness.
- o Screen laboratory reports of.

Blood type

- HLA screening
- Complete blood count
- Serum chemistry
- Blood
- Sputum
- Urine culture
- Urine analysis
- Chest X ray
- Viral Serology
- Administer pre operative antibiotics and solumedrol.
- Repeat blood, sputum, urine culture (from each ureter and blabber).
- Harvest kidney.

ii) Cadaver Donor harvesting and Preservation

The donor kidneys are obtained through a block nephroureterectomy including a segment of aorta and inferior vena cava. Before cross matching aorta is canulated and organs flashed with a preservative solution (University of Wisconsin Solution). The harvest is then completed and organs stored in cold ice bath virtually eliminating warm ischemia. **Warm ischemia** refers to the period between donor asystole or lack of blood flow to the kidneys and the beginning of cold storage. **Cold ischemia** refers to the time in cold storage or machine perfusion. **Warm ischemia** for longer than 20 minutes and **Cold ischemia** longer than 24 hours are associated with increased risk for delayed graft function. In the process of harvesting cadaver kidney is flushed with a cold intracellular solution and placed in an ice bath for storage until transplantation. The time of ice bath varies.

1.7.2 Live Organ Donation

Live organ donation is not as frequently performed as cadavaric organ transplantation. Individuals who are willing to donate to a family member, a friend or as an anonymous donation must meet strict evaluation and assessment criteria.

i) Live Donor Evaluation

- Identification of interested family member.
- Potential donor schedules appointment with donor nephrologists.
- Complete history and physical examination.
- Blood type and HLA determination.
- Screening lab analysis – complete blood count, serum chemistry.
- Viral serology – HBV, HCV, CMV, PT and PTT, glucose tolerance if diabetic.
- Pregnancy test.
- Urine analysis with examination of sediment and urine culture – 2 times.
- 24 hours urine collection for protein and creatinine excretion – 2 times.
- ECG.
- Chest X ray
- PFT.
- Intravenous pyelogram.
- Arteriogram.
- Repeat cross match before donation.

Living unrelated donors become a major source of organ donation for kidney transplantation. The graft survival rate for spouse donor was superior than that of cadaver donors and identical to living related one halo type donor.

Check Your Progress 3

i) List the points you will keep in mind for evaluation of Cadaver Donor.

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ii) List the points you will keep in mind for evaluation of a Live Donor.

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iii) Name the solution used to flush the donor kidney for preservation.

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1.8 TRANSPLANT SURGERY

The potential recipient must be evaluated for operative suitability immediately before surgery. This includes immediate management of volume status and electrolyte abnormalities (generally with dialysis) and an aggressive search for active infection such as UTI, peritonitis associated with peritoneal catheter, or evidence of coronary ischemia. Before operation, the patient needs to be re-examined. A thorough preoperative medical evaluation is done to screen for any illness. It is advantageous to dialyse the patient within 12 hours of the transplant surgery to optimize volume status and correct any electrolyte imbalance.

The surgery usually takes three hours. The donated kidney is usually implanted in the right or left lower quadrant of the recipient, (Fig 1.3) determined by the

anatomy of the donated kidney, through a "hockey stick" type shape incision extending from the iliac crest to the symphysis pubis.

1.9 MANAGEMENT OF PATIENT UNDERGOING RENAL TRANSPLANTATION

Nursing care for the patient preparing for renal organ transplantation includes the following measures:

1.9.1 Preoperative Nursing Management

- Collaborating with the organ procurement agency, the organ transplant coordinator, and the surgical team to ensure a timely and successful graft placement.
- Assisting in dialysis, which may be performed before surgery, depending on electrolyte and fluid status.
- Maintaining the patient as nothing by mouth (NPO) to help minimize nausea and vomiting immediately post-operatively and prevent intraoperative aspiration.
- Performing a thorough nursing assessment
    - o Baseline vital signs and weight should be documented for later comparison; and
    - o Extremity pulses should be assessed and documented for later comparison.
- Reviewing current medical history and implementing the following medical orders:
    - o Delivering appropriate medication administration (some facility protocols dictate immunosuppressive coverage and antibiotic coverage preoperatively),
    - o Obtaining blood tests to make sure that the patient has electrolytes within normal limits, is infection free, and is the appropriate cross match for the organ donation,
    - o Initiating telemetry and obtaining an electrocardiogram (ECG),
    - o Obtaining a chest radiograph and forwarding the results to the surgical team,
    - o obtaining vascular access, and
    - o Skin prepariton, which may include clipping hair in the surgical area (Bartucci & Schanbacher, 2001; Burrows – Hudson et. al. 2005).

Investigation

As a preparation for transplant – both the donor and recipient undergo an in depth reassessment and several investigations which include –

- Urine analysis — Routine, Microscopic and 24 hours urine test for creatinine
- Blood test
- Human Leucocyte antigen (HLA)

- Renal ultrasound
- Vital studies
- Kidney Biopsy
- Intravenous Pyelogram
- Renal arteriogram

Fumigation - Preparation of the room to be fumigated by 2% Bacillocid.

Preoperative Implementation

- Verify histocompatibility test of identical twin or family member.
- Administer immunosuppressive medication to recipient as prescribed for 2 days before transplantation.
- Maintain protective isolation.
- Verify that haemodialysis of the recipient is completed 24 hours before the transplant.
- Ensure that patient is free from any infection.
- Assess renal function studies.
- Encourage discussion of feelings of both donor and recipient.

1.9.2 Post Operative Management

The nurse will be at the bedside for 24 hours for close monitoring. Reverse Barrier nursing is an important aspect of postoperative care.

Remember

- Kidney begins to function immediately or it may delay for few hours.
- Haemodialysis is performed until adequate kidney function is established.
- Monitoring of vital signs is crucial.

In the 1st post operative hour the vital signs are recorded every 15 minutes. In the next 2 hours it is recorded every ½ hour and then one hourly.

- Monitoring intake and output is done hourly.
  - monitor I/V fluids closely for fluid overload.
  - start oral fluids as prescribed.
- Monitoring of daily lab studies, urine for specific gravity, blood studies daily weight, pulse oximetry, BUN and creatinine levels are required to be measured accurately.
- Maintaining the patient in semi fowler position is desirable.
- It is important to note that the urine is pink and bloody initially but gradually returns to normal within several days to weeks.
- Monitoring for gross haematuria and clots are important which are not expected and needs to be notified to the surgeon if they occur.

- Monitoring the three way bladder irrigation if prescribed is done to prevent clot formation.
- The Foley's catheter should be removed as soon as possible to prevent infection.
- Maintain protective isolation precaution and monitor infection. Reverse barrior nursing should be need.
- Monitor bowel sounds and initiate a diet as prescribed when bowel sound return.
- Maintain good oral hygiene to monitor stomatitis, bacterial and fungal infection.
- Encourage deep breathing and coughing exercise.
- All staffs need to maintain strict aseptic technique while doing dressing.
- Administer medications as prescribed which may include – antifungal, antibiotics, immunosuppressive agents and corticosteroids.
- Continuous Assessment for organ rejection is important by monitoring fever, malaise, raised WBC.
- Promoting a healthy live donor and recipient relationship is the nurses' mandate.
- Monitoring is required to assess the patient and recipient for depression.

1.9.3 Post Operative Health Education and Home Care

Post operative health education and home care begins from the admission of the patient and post operatively after the renal transplantation. Instructions that you must provide to your clients**:include –**

- Avoid prolonged period of sitting.
- Recognise the signs and symptoms of infection and rejection.
- Avoid contact sports in which the kidney may be injured.
- Avoid exposure to people with infection.
- Use medication as prescribed and maintain immunosuppressive therapy lifelong.
- Dietary restrictions – restrict sodium and calories, excess proteins should be avoided so as to reduce formation of waste products.
- Maintain weight.
- Record and report weight gain/ loss of more than ½ -1 kg within a period of few days to a week.
- Daily intake output chart to be maintained.

1.9.4 Complications of Renal Transplantation

The following complications are possible after a renal transplantation:

i) Graft rejection

Rejection is always a major risk of transplantation. Body recognises the transplanted kidney as non-self.

Graft rejection is assessed by:

- Fever, malaise, elevated WBC count, graft tenderness, signs of deteriorating renal function, acute hypertension and anaemia.
- Hyperacute rejection – occurs immediately after surgery to 48 hours post operatively when removal of rejected kidney is necessary.
- Acute rejection – occurs within 6 weeks of surgery but sometimes it may occur as late as 2 years. So high doses of corticosteroid, monoclonal antibiotics may be administered.
- Chronic rejection – occurs slowly, months to years after transplant. Hence immunosuppressive medication must continue.

ii) Urologic and Vascular Complications

These complication include the development of a lymphocele (pocket of lymph accummulation), compression of the iliac vein causing ipsilateral leg swelling, haematoma leading to pain, infection and urethral stenosis or occlusion.

- Urine leak from the anastomotic site may occur in the early post operative period or at the onset of post transplant diuresis. Pseudo aneurysms secondary to infection, arterial thrombus or venous thrombus could also occur.
- Post Operative bleeding may occur when the small vessels in the renal helium has increased perfusion.

iii) Homodynamic Instability

There is a delayed functioning of the transplanted kidney due to prolonged ischemic time. This leads to intravascular volume problems.

iv) Hypertension

The etiology of hypertension post renal transplant may be due to retained native kidneys, parenchymal disease of transplanted organ, effects of immunotherapy, renal artery stenosis and weight gain.

v) **Electrolyte and Acid base imbalance:** Electrolyte imbalance of hyper kalaemia hyperphosphatemia and hyponatraemia may persist or can be after exacerbated after renal transplantation. Surgical tissue destruction along with the impaired renal function increases the hyperkalaemia state. After surgery impairment of hydrogen ion leads to metabolic acidosis.

vi) **Renal dysfunction:** Recipients of cadaver kidneys with preservation times of more than 24 hours may experience delayed graft function. Acute tubular necrosis is a common complication and may last from several days to weeks Most patients can be supported by dialysis until their urine output increases and their serum creatinine and BUN levels begin to normalize.

vii) **Infection:** Patients with renal transplantation are very susceptible to infection because of the higher amounts of immuno suppression received, the alterations in normal defenses of the body due to surgery and the impact of ESRD. Co-morbidities may compound the effects of the immune response.

Check Your Progress 4

i) Write down the preoperative nursing management of renal transplantation in points.

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ii) List down the different preoperative implementation done before Transplantation.

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iii) List down the special points in post operative management.

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iv) List down the different complications patient may develop after transplantation.

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v) List down the health education given to a patient after Renal Transplantation.

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1.10 LET US SUM UP

This unit discussed about definition of renal transplantation, indications, contraindications, advantages, classification of donors and their prerequisites, management of patient preoperatively and postoperatively, complications of renal transplantation, and special instructions given to a renal transplant patient. Keeping in mind all the above points- individualized care should be given to the transplant- patient.

1.11 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

i) • Kidney transplantation is a surgical procedure to remove a healthy functioning kidney from a living or brain-dead donor and implant it into a patient with nonfunctioning kidneys.

ii) • ESRD, bilateral nephrectomy or when removal of solitary functional kidney is indicated.

iii) • Reversal of physiologic changes due to renal failure.
- Higher Survival Rate
- Distinctly better quality of life
- Fewer Dietary Restriction
- Cost effective- less costly than long term dialysis treatment.

iv) Contraindication to Renal Transplantation
- Life expectancy- less than one year
- Recent malignancy

  Severe or uncorrectable
  - Plum onary disorders
  - Severe cardiomyopathy
- Active peptic ulcer diseases.
- Active infection
- HIV infection
- Chronic Untreated infection
- Uncontrolled psychiatric disorder

- Psychiatric disorders that impair ability to give informed consent or adhere to treatment regimen
- Active Substance abuse
- ABO Blood Group Mismatch
- Severe cachexia
- Severe or uncorrectable coronary artery disease (CAD).
- Severe or uncorrectable pulmonary disease and periferal vascular diseases (PUD).

Check Your Progress 2

i) Related living donor

- Other living donor
- Cadaver donor

ii) Special evaluation for:

- Diabetic Patient
- Cardio Vascular Disease
- Infectious
- ABO blood group matching
- Cellular assay
- Malignancy
- Gastro Intestinal disease
- Genito Urinary Disease
- Pulmonary Disease
- Psychological issues
- Immunologic evaluation

iii) Eligibility or prerequisite for a donor

- Donor must have normal renal function of both kidneys.
- No malignant-disease or generalized infection or abdominal/renal trauma is present.
- Potential donor must have negative Hepatitis B antigen and to negative HIV antibody.
- No problem in ventilation, in respiration or cardiac rhythm.
- Donor must have normal BP.
- Donor must have in excellent health with two properly functioning kidneys.
- Serum for ABO blood group, tissue specific antigen, Human leukocyte antigen (HLA) suitability, mixed lymphatic synthetic culture index (histocompatibility).
- Complete emotional wellbeing of the donor is determined.
- Complete understanding of the donation process and outcome is necessary.

Check Your Progress 3

i) Cadaver Donor's Evaluation:

- Identify potential cadaver donor.
- Inform organ procurement agency.
- Establish brain death.
- Obtain consent donation from family.
- Preparation of donor with appropriate haemodynamic and respiratory support.
- Maintain stable electrolyte and urine output.
- Obtain medical history to rule out high risk illness.
- Screen lab examination
  - o Blood type
  - o HLA screening
  - o Complete blood count
  - o Serum chemistry
  - o Blood
  - o Sputum
  - o Urine culture
  - o Urine analysis
  - o Chest X ray
  - o Viral Serology
- Administer pre operative antibiotics and solumedrol
- Repeat blood, sputum, urine culture (from each uretor and bladder)
- Harvest kidney

ii) Live Donor Evaluation

- o Identification of interested family member.
- o Potential donor schedules appointment with donor nephrologists.
- o Complete history and physical examination.
- o Blood type and HLA determination.
- o Screening lab analysis - complete blood count, serum chemistry.
- o Viral serology - HBV, HCV, CMV, PT and PTT, glucose tolerance if diabetic.
- o Pregnancy test.
- o Urine analysis with examination of sediment and urine culture - 2 times.
- o 24 hours urine collection for protein and creatinine excretion - 2 times.
- o ECG.
- o Chest X ray
- o PFT.

- o Intra venus pyologram.
- o Arteriogram.
- o Repeat cross match before donation.
- o Hospitalization of donor.

iii) Donated organ is flushed with preservative solution namely University of Wisconsin Solution.

Check Your Progress 4

i)
- Verify histocompatibility test
- Administer immunosuppressive medication 2 days prior operation
- Strict Isolation
- Haemodialysis to complete 24 hours prior to surgery
- Ensure patient is free from infections
- Assess renal function studies
- Give psychological support
- Prepare the part
- Hospital clothing to be put on
- Identification wrist band to be worn by patient.
- Keep file ready with all required paper
- Give premeditation
- Accompany the patient OT
- Reassure relatives

ii)
- Monitor vitals and give proper position with all drainage tubes
- Perform Haemodialysis until kidney function is established
- Monitor intake-output chart hourly
- Monitor daily lab studies
- Note the color consistency and amount of urine
- Prepare for discharge health teaching.

iii)
- Kidney begins to function immediately or it may delay for few hours.
- Haemodialysis is performed until adequate kidney function is established.
- Monitor vital signs.
- Monitor intake output every hourly.
- Monitor daily lab studies, urine for blood specific gravity, daily weight, pulse oximetry, BUN, creatinine level.
- Maintain the patient in semi fowler's position.
- Note that the urine is pink and bloody initially but gradually returns to normal within several days to weeks.
- Monitor for gross haematuria and clots, which are not expected and notify the surgeon if they occur.

- Monitor the three way bladder irrigation if prescribed to prevent clot formation.
- Note that Foley catheter to be removed as soon as possible to prevent infection.
- Maintain protective isolation precaution and monitor infection.
- Monitor I/V fluids closely for fluid overload.
- Start oral fluids as prescribed.
- Monitor for Bowel sounds and initiate diet as prescribed when bowel sound returns.
- Maintain good oral hygiene to prevent stomatitis bacterial and fungal infections.
- Encourage deep breathing and coughing exercises.
- Maintain strict aseptic technique while doing dressing.
- Administer medication as prescribed which may include - antifungal, antibiotics, immunosuppressive agents and corticosteroids.
- Assess for organ rejection by fever, malaise - WBC count etc.
- Promote live donor and recipient relationship.
- Monitor patient and recipient for depression.

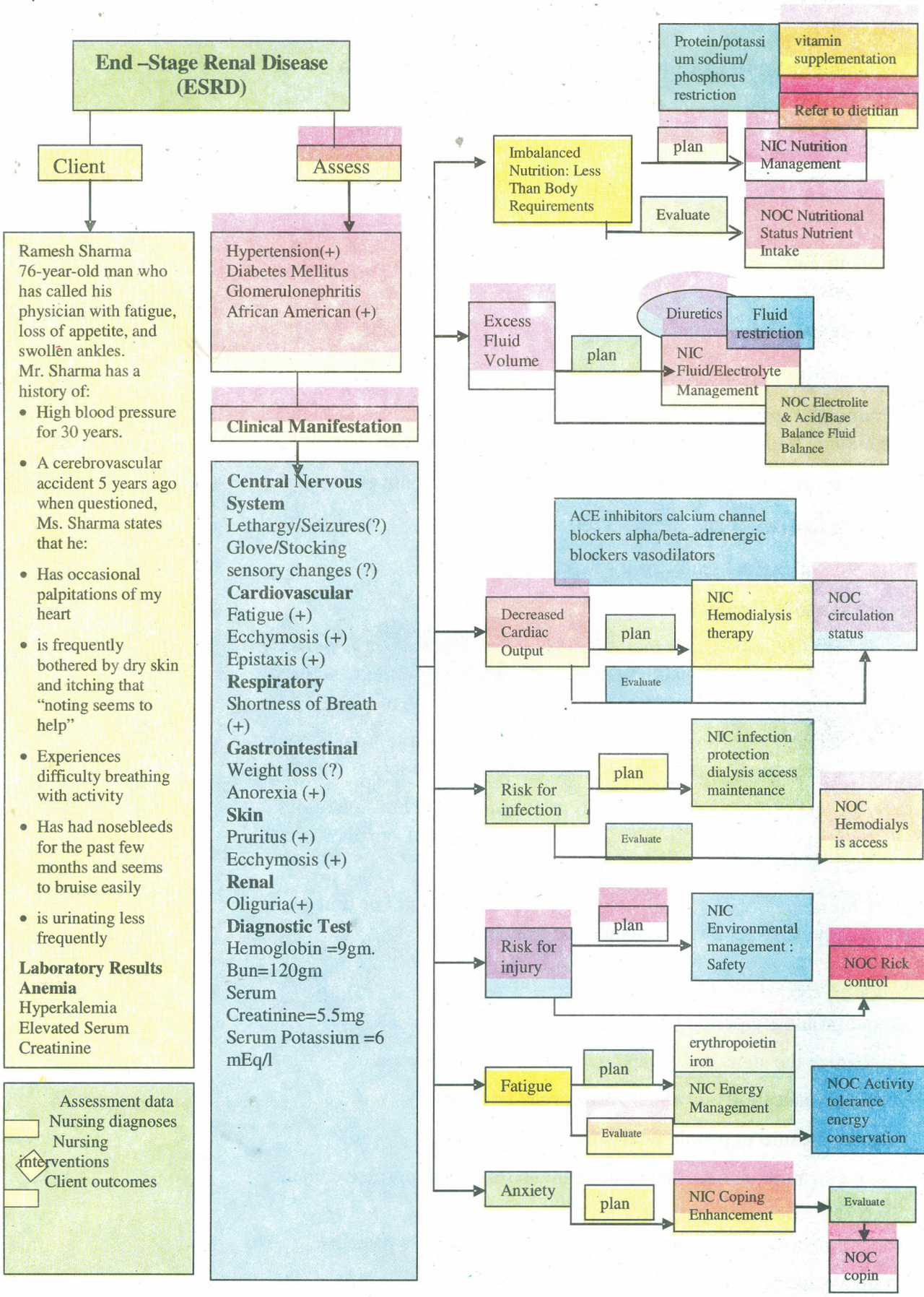
iv) • Post Operative Complications

- o Graft rejection - is the major complication. Assessment is done by fever, malaise, elevated WBC count, graft tenderness, signs of deteriorating renal function, acute hypertension, anemia.
- o Hyperacute rejection - occurs immediately after surgery to 48 hours post operatively so removal of rejected kidney is done.
- o Acute rejection - occurs within 6 weeks of surgery but sometimes it may occur as late as 2 years. So high doses of corticosteroid, monoclodal antibiotics may be administered.
- o Chronic rejection - occurs slowly months to years after transplant. So immunosuppressive medication to continue.

v) Post Operative Health Education and home care.

- o Avoid prolonged period of sitting.
- o Recognize the signs and symptoms of infection and rejection.
- o Avoid contact sports in which kidney may be injured.
- o Avoid exposure to people with infection.
- o Use medication as prescribed and maintain immunosuppressive therapy lifelong.
- o Dietary restriction- restrict sodium and calories, excess proteins.
- o Maintain weight.
- o Daily intake output chart to maintain.

Appendix A – Flow chart of End stage renal disease



UNIT 2 ACUTE TUBULAR NECROSIS (ATN) AND BLADDER TRAUMA

Structure

2.0 Introduction

2.1 Objectives

2.2 Definition and Causes of Acute Tubular Necrosis

2.3 Pathophysiological Changes and Clinical Features

2.4 Diagnostic and Therapeutic Management in Acute Tubular Necrosis

2.5 Nursing Process in Acute Tubular Necrosis

2.6 Bladder Trauma

- 2.6.1 Concept and Definition
- 2.6.2 Pathophysiological Changes and Signs and Symptoms
- 2.6.3 Diagnostic Tests
- 2.6.4 Medical Management and Surgical Management
- 2.6.5 Nursing Management

2.7 Let Us Sum Up

2.8 Answers to Check Your Progress

2.0 INTRODUCTION

In the previous unit 1 you have already learnt about renal transplant and care of patient undergoing renal transplant. We shall now look at two important condition from the point of critical care nursing namely, Acute Tubular Necrosis (ATN) and Bladder trauma.

Further, this unit will cover the definition, Pathophysiological changes and therapeutic management of ATN thus helping you to develop the nursing process.

This unit also details about bladder trauma and its management along with the diagnostic tests which help you to care for your patient with bladder trauma.

2.1 OBJECTIVES

After completing this unit, you will be able to:

- describe the structure of urinary system;
- state the clinical features and causes of Acute Tubular Necrosis;
- apply nursing process in management of the patient with Acute Tubular Necrosis;
- explain the causes and signs and symptoms of a patient with bladder trauma; and
- describe the management of patient with bladder trauma based on nursing process.

2.2 DEFINITION AND CAUSES OF ACUTE TUBULAR NECROSIS

As you know nephrons are the basic unit of the kidney and they are especially susceptible to injury from ischemia and nephrotoxins. In Acute Tubular Necrosis (ATN) destruction of tubular epithelial cells causes an abrupt and progressive decline of renal function.

Definition

Acute renal failure with tubular necrosis is a disease affecting both kidneys characterized by anuria or oliguria with urine of low specific gravity and rapidly developing uremia.

Causes

The causes of ATN are divided to two categories as follows:

- Ischemic acute Tubular necrosis.
- Toxic Acute Tubular necrosis.

Ischemic acute tubular necrosis occurs as a result of vasodilatation associated with sepsis and when hypotension or a low cardiac output episode is prolonged, it further deteriorates by renal hypoperfusion or dehydration.

Toxic ATN occurs due to administration of radio opaque dye during an interventional or diagnostic radiological study which further causes the injury to renal endothelium.

Acute tubular necrosis also occurs as a complication of various conditions which are associated with marked hypotension, arteriolar constriction, severe water and salt depletion.

Sometimes acute tubular necrosis may develop following general circulatory disturbances. For example, it may occur following incompatible blood transfusion or during acute hemolytic crises.

It may even be seen in patients with obstructive jaundice, severe infection or septicemia.

Nephrotoxic drugs like sulphonamides, cephaloridines, poisonous substances and radiologic contrast media which produce renal parenchymal necrosis may also be responsible.

2.3 PATHOPHYSIOLOGICAL CHANGES AND CLINICAL FEATURES

Acute Tubular Necrosis (ATN) ends in renal failure. Severe shock, water and salt depletion are accompanied by vasoconstriction. Renal ischemia may result from hypotension and different arteriolar vasoconstriction. If ischemia is severe and of long duration, focal necrosis of renal tubules occurs and may be aggravated by intravascular glomerular thrombosis. Ischemia lasting more than 2 hours causes severe and irreversible damage to tubules causing patchy cellular necrosis and sloughing. You must remember that the glomerular filtration rate is significantly

reduced as a result of ischemia, activation of rennin-angiotensin system and tubular obstruction by cellular debris which raises the pressure in the glomerular capsule and leads to oliguria and finally to azotemia.

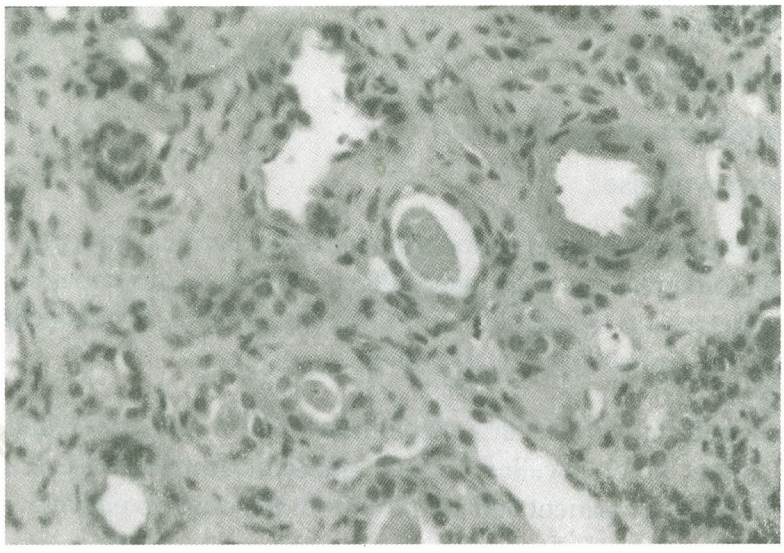


Fig. 2.1: Acute Tubular Necrosis

Look at flow chart below for quick understanding of Pathophysiology of Acute Tubular Necrosis (ATN).

Pathophysiology of ATN in a cascade form

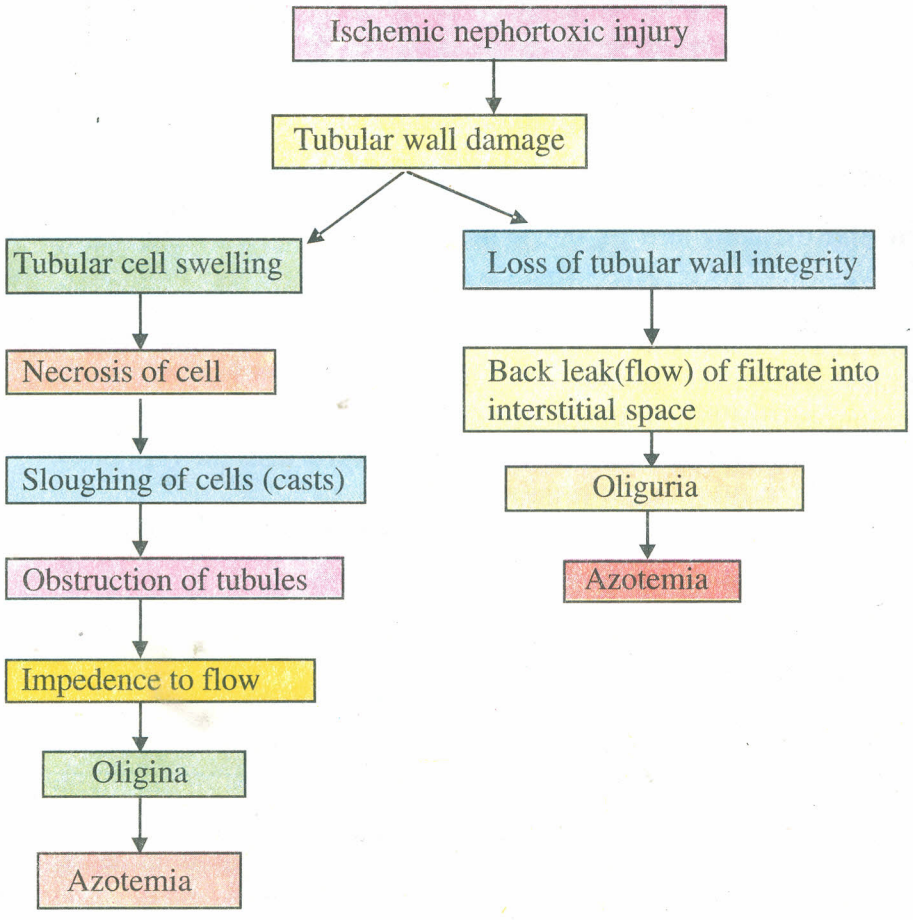


Fig. 2.2: Flow chart of pathophysiology in Acute Tubular necrosis

Clinical features

The clinical features of ATN are same as in renal failure. In the clinical feature of ATN, there are 4 phases; as given below:

- **onset phase,**
- **oliguric phase,**
- **diuretic phase,** and the
- **recovery phase.**

In the **onset phase** or initial period from the time of critical injury to cell damage. Here the glomerular filtration rate (GFR) decreases due to impaired renal blood flow and decreased glomerular ultra filtration pressure. If treatment is given in this phase the damage can be alleviated.

Thus, you will find that the urine violume of patient is greatly reduced to 200 to 500 cc/ 24 hrs which is called **oliguric phase** or **anuric phase**. It lasts for 5-8 days in nanoliguric patient and 10-16 days in the oliguric patient.

You may find that urine contains protein, cast, RBC & WBC and the specific gravity is 1.010 early in the course of disease. This phase may continue for several days to weeks.

At first patient may feel well but after sometime anorexia, nausea vomiting may occur which may be followed by mental confusion and later muscular twitching, fits, drowsiness, coma and bleeding episodes occur.

At this stage you need to be alert for the main dangers to life as indicated by:

- Pulmonary oedema
- Potassium intoxication
- Occurrence of severe systemic infection.

Oliguric phase usually lasts for one to three weeks and may include a period of anuria.

The third phase is **diuretic phase**. In this phase you will find that there is uncontrolled water and sodium loss and sometimes flaccid paralysis due to loss of potassium occurs in the absence of treatment. The concentration of blood urea ceases to rise and then gradually falls. This phase lasts for 7-14 days.

The last phase of ATN is the **recovery** or **convalescent phase**. During this phase renal function slowly returns to normal with a GPR that is 70-80% of normal with in a period of 1-2 yrs.

Check Your Progress 1

i) Define Acute Tubular Necrosis.

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ii) List the two main cases of Acute Tubular Necrosis (ATN).

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iii) List the signs and symptoms of Acute Tubular Necrosis.

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2.4 DIAGNOSTIC AND THERAPEUTIC MANAGEMENT IN ACUTE TUBULAR NECROSIS

Diagnosis of ATN relies more on blood serum analysis rather than urine analysis. Serum electrotype levels of sodium, potassium, calcium, magnesium, phosphate chloride is done. Artery blood gas is also assessed as these patients develop metabolic acidosis which is the LANDMARK of acute renal failure. Blood serum levels of BUN and serum creatinine is also **above increased.** If the patient is producing adequate quantity of urine, creatinine decreases fractional excretion of sodium and urinary sodium is also evaluated.

A CT may also be preformed for evaluating THE ANATOMIC status of the kidney in the critically ill patient.

Therapeutic management includes:

Prevent or minimize renal lesion, by treating underlying cause urgently and the appropriate solution is administered to treat the deficit

Manage oligric phase: Therapeutic management during the **oliguric phase** is to minimize the need for renal function until healing occurs and this is achieved by simple dietary restrictions.

- **Water and electrolyte balance-** This is maintained by replacing the obligatory losses of water through the skin and lungs estimated to be about 600 ml of water per day and by giving no electrolytes since none are being lost from the body. Only in febrile patient extra allowances of water is required to replace the fluid and a further supplement equal to the volume of urine passed each day should be added.
- **Proteins and calories:** Dietary protein is restricted to about 20gm/day. In case of vomiting, oral treatment is avoided and carbohydrate should be given in the form of 10 percent glucose through a large peripheral vein. When diuresis starts the concentration of blood urea tends to remain constant for a few days and then begins to fall. When this occurs you must guide your patient to consume a light diet containing 45gm protein per day and ample of fruits.
- **The intake of fluid** is also increased by the volume of previous days urinary output. On average 3 gm sodium chloride and 2gm sodium bicarbonate is needed for each liter of urine passed. Oral administration of potassium chloride is also required.

As the blood urea concentration return to normal value and renal function improve, a normal diet may be resumed.

- In case of **elevation of plasma potassium**, attempts should be made to reduce it by
  - o Using sodium or calcium charged resin which removes potassium from the body,
  - o By giving 20 units of soluble insulin subcutaneously and intravenous infusion of 50gm glucose,
  - o Controlling metabolic acidosis is done by intravenous administration of isotonic sodium bicarbonate.
- **Haemodialysis in certain patients may be anticipated especially in those.**
  - o Suffering from severe infection or massive tissue damage,
  - o Where Blood has acumulated in one of tissue spaces,
  - o Who have Rapid rise of blood urea and potassium,
  - o Daily heamodialysis may be required for several weeks before renal functions starts.

Great care for infection control and for prescribing drugs which are excreted by kidney are also exercised.

2.5 NURSING PROCESS IN ACUTE TUBULAR NECROSIS

The nurse plays a vital role in managing a patient with ATN s/he asseses the patient and the formulates the nursing diagnosis that will enable you to care for the patient with ATN. The nursing diagnosis may include –

a) Excess Fluid Volume related to inability to excrete water

b) Activity intolerance related to anemia

c) Risk of injury related to bleeding tendency

d) Risk for infection related to impaired immune system

e) Altered Nutrition related to restricted diet, anorexia, nausea, vomiting

f) Risk of injury related to acute confusion

g) Sexual dysfunction

h) Knowledge deficit.

After identifying the various nursing diagnosis we will now go on to put down nursing interventions and their rational so as to manage this patient with ATN.

Your nursing care plan and implementation will be according to the Nursing diagnosis identified in your client. Here are some standard interventions to help you as a guideline:

Nursing Intervention	Rationale
Excess Fluid Volume: • Monitor daily weight at same time	Weight gain represents retention of fluid
• Monitor intake- output chart	Monitors degree of fluid retention
• Assess for and report shortness of breath, tachycardia, crackles in lungs, frothy sputum, heart irregularities, hypotension, cold clammy skin	These symptoms of heart failure may accompany fluid overload
• Watch for distended neck vein	Right sided heart failure causes distended neck vein due to venous congestion
• Monitor vital signs , BP	Increase in Blood pressure s reflects fluid volume excess
• Monitor for edema	It is a symptom of fluid overload
• Monitor serum protein albumin level	Low serum protein albumin contributes to edema
• Maintain sodium and fluid restrictions as ordered. Teach patient importance of each	Sodium and fluid cause fluid retention in patients with renal failure
• **Activity Intolerance** Assess for pale mucous membrane, skin color, chest pain and dyspnea	These are signs of anemia
• Monitor for Hb and Haematocrit	Low hemoglobin indicates anemia
• Watch for signs of bleeding	Bleeding worsens anemia
• Administer blood transfusion and Epogen as prescribed	Erythropoietin stimulates production of RBC by bone marrow
• Rest and Quiet environment	Rest period decreases demand of oxygen in body
• **Risk for injury related to bleeding** Observe for and report blood in stool, bleeding from mucus membrane	Bleeding must be recognized to prevent complication
• Monitor Hb, vitals	Low Hb indicates blood loss
• Teach patient to prevent injury to self and report if any symptoms of bleeding	Injury can cause bleeding
• **Risk for infection.** Monitor for s/s of infection and report promptly to physician	Early recognition of infection and prompt treatment help to prevent complications
• Protect patient from any source if infection including visitors or roommates carrying infections	Exposure to pathogen increases risk of infection as their immunity is low
• Maintain skin integrity	Intact skin prevents infection
• Standard hand hygiene practices	It helps to control infection
• Do culture for any suspected site of infection as prescribed by physician	Culture identifies pathogens and guides treatments
• Teach patient s/s of infection	Early reporting indicates early treatment

• **Altered Nutrition** Monitor weekly weight and serum protein albumin level	Weight & lab result gives information about nutritional status
• Initiate calorie count-consult dietitian	This gives information about adequacy of diet
• Provide frequent oral hygiene	Oral hygiene enhances appetite
• Offer frequent small feeds and dietary Supplement	Oral care enhances appetite.
• Offer medications as prescribed for nausea before meal	Small feeds are better tolerated, nausea should be controlled
• Ensure bowel movement daily	Constipation interferes with appetite
• **Risk of injury related to acute confusion.** Assess and report decrease in alertness, loss of memory, delirium, muscle tremors, twitches of hands	These are symptoms of uremic encephalopathy. If present dialysis may be initiated
• Observe for restless leg, numbness, tingling, burning pain in extremities	There are signs of neuropathy and makes a patent prone to get injury
• Administer medications as prescribed	Anti depressant and anti convulsant may help neuropathy
• Protect from injury and falls	Patient with numbness is at risk for fall
• Ambulate the patient at least three times per day	Ambulation prevents calcium mobilization from bones.
• **Sexual dysfunction** Assess problems related to sexual function	Patient may not share this problem unless discussed
• Offer counseling and include partner as desired by patient	Professional counselor may be contacted
• **Knowledge deficit.** Assess patient and family's knowledge of disease and treatment regimen	A baseline arrangement provides direction for instruction
• Consult dietitian, clinical specialist, social worker if needed	Specialist may be consulted for proper management

Note: The above given interventions are just the guide. Kindly assess the patient before you do these nursing intervention.

Check Your Progress 2

i) Describe the therapeutic management of a patient with Acute Tubular Necrosis (ATN).

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ii) State Nursing diagnosis of Acute Tubular Necrosis (ATN).

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iii) Write nursing intervention of a patient with Acute Tubular Necrosis (ATN).

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2.6 BLADDER TRAUMA

2.6.1 Concept and Definition

You must have worked in the Trauma and Emergency unit? During that posting you might have come across with cases of Bladder trauma often resulting from automobile accidents, (when the seat belt compresses the bladder). A bladder distended with urine can rupture with a direct blow to the lower abdomen. The bladder may also be punctured by a bullet, knife, bony splinter from a fractured pelvis, or internal medical instrumentation. When the bladder ruptures urine spills into the peritoneal cavity. The peritoneal urine accumulation from a ruptured bladder can lead to peritonitis and pelvic cellulitis.

Definition

It is defined as blunt or penetrating injury to the bladder that may cause bladder rupture.

Bladder Trauma may be classified as follows:

i) Contusions

ii) Intra peritoneal injury

iii) Extra peritoneal injury

iv) Combined injuries.

You will come across this condition more commonly among males rather than females.

2.6.2 Pathophysiological Changes and Signs and Symptoms

Contusions and Intra peritoneal rupture may be secondary to blow and kick or fall on fully distended bladder. Extra peritoneal rupture is caused by fractured pelvis or is secondary to major trauma or surgical damage.

Intra peritoneal rupture is associated with sudden agonizing pain in hypogastria often accompanied by syncope, haematuria, distended abdomen and peritonitis. Bladder injury usually produces hematuria and pain low in the abdomen or pain referred to the shoulder. The client may also have trouble in voiding. Manifestation of peritonitis may develop as well. Fever is usually present as the peritonitis and pelvic cellulitis continue to develop. If the client has had an injury or blow to the abdomen, suspect bladder injury as the cause of manifestation. The physical finding for bladder trauma also includes lower abdominal bruising, distension and pain.

2.6.3 Diagnostic Tests

Diagnostic tests include an intravenous pylogram (IVP) with lateral view or a CT scan with the bladder full and empty, a cystogram and a voiding cystourethrogram. If blood is flowing from the meatus, urethral disruption may be present. In this case, catheterization should be avoided until the urethra is evaluated. This allows assessment of both bladder integrity and the bladder's ability to empty.

2.6.4 Medical Management and Surgical Management

Medical Management

The first treatment for suspected bladder injury is insertion of an indwelling or suprapubic catheter to monitor for hematuria or urine production and to keep the bladder decompressed during healing. Any injury other than a simple contusion or a very small perforation requires surgical repair.

Surgical Management

Clients with bladder injuries usually require surgical intervention. After a urethral or suprapubic catheter has been inserted, surgical repair of the damaged bladder wall is performed. The extravascated urine in the perivesical area is drained. It is important to maintain urinary drainage through a patent catheter to promote healing and to avoid potential development of fistulae or leakage.

Extraperitonial rupture of the bladder may be managed conservatively with catheterization and antibiotics for 7 to 10 days but unresolved extravasations may require surgical management.

Check Your Progress 3

i) Explain the Therapeutic surgical management of a patient with bladder trauma.

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2.6.5 Nursing Management

You must immediately assess a suspected bladder injury if the client has had blunt trauma of the lower pelvis or abdomen. Closely monitor the client's urine output for both amount and the presence of hematuria. Report any decrease in urine output in relation to fluid intake to the physician immediately. Careful catheter insertion is necessary for the client with suspected bladder trauma.

Nursing Management of the Surgical Client

Your actions for Nursing Management depends on the type of injury. Extra peritoneal injury can be managed by catheter drainage of the bladder. Intra peritoneal injury requires laparotomy with bladder repair, peritoneal lavage followed by suprapubic and bladder drainage.

Post operative management includes, maintaining urinary drainage to prevent tension on the sutures in the bladder. A penrose drain is left in place to allow drainage of any urine remaining in the pelvis. This may necessitate dressing changes. Since the patient may be discharged with an indwelling or supra pubic catheter, you will have to teach catheter care to the client and the relatives. Assess the client's self care abilities to determine a possible need for assistance at home. If the patient or significant others cannot care for the catheter, arrange for a home health visit.

Follow up care is essential after discharge to assess healing. A cystogram may be done before the catheter is removed. If a suprapubic catheter has been placed, the client can begin bladder training before the catheter is removed. If the client has a urethral catheter, the catheter is removed before the client can attempt to void. If client do not void within 4 to 6 hours after removal, the catheter should be reinserted.

Nursing Process

The diagnosis made by nursing personnel can be applicable in care of a patient with bladder trauma. After assessment you may arrive at various nursing diagnosis as follows:

- Risk of injury – related to surgical procedure
- Risk of infection
- Risk of impaired skin integrity
- Pain related to surgical intervention
- Constipation
- Anxiety.

Your nursing care Plan and Intervention will be according to the nursing diagnosis identified in your patient. A guideline to help you is as follows:

A) Risk for injury to surgical procedure

- Support catheter while moving
- Support dressing while coughing and deep breathing exercise
- Change dressing if soaked
- Watch for bleeding or leakage of urine
- Avoid kinking of tubes

B) Risk for infection

- Monitor temperature, pulse, respiration, BP
- Administer antibiotic as prescribed
- Maintain hygiene (bed bath, changing clothes and linen)
- Keep the dressing clean by changing dressing daily
- Observe wound for any sign of infection
- Empty urinary bag when it is filled 3/4th
- Encourage the patient to take more oral fluids

C) Risk of impaired skin integrity

- Keep the dressing clean and dry
- Observe wound carefully
- Watch for any leakage around the suprapubic catheter
- You must keep your patient clean and dry
- Change position frequently and give back care 4 hourly
- Monitor intake and output
- If patient does not void within 4-6 hours after removal of catheter – it is to be reinserted again.

D) Pain in the surgery area

- Watch for any signs of bleeding or infection of wound
- Pain killer to be given as prescribed
- Maintain cool and calm environment
- Diversion therapy some times are helpful

E) Constipation related to immobilization, decreased physical activity and poor oral intake

- Encourage patient to take adequate fluids
- Small and frequent meals which are easily digestible.
- Roughage to be introduced in meals
- Laxative or stool softeners are helpful.

F) Anxiety

1) Teach patient about care of indwelling and suprapubic catheter on discharge
2) Explain about treatment to patient and family
3) Assess patient's self care activities to determine the possible need for assistance at home.
4) Clear doubts of patients and family
5) Bladder training programme can be conducted if suprapubic catheter has to be removed.
6) Watch for urine output when the catheter is removed (urine color amount, consistency)
7) Teach the patient about the change of color of urine for four days after surgery

Check Your Progress 4

i) Write the common nursing diagnosis you will make in a patient with bladder trauma.

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2.7 LET US SUM UP

In this unit you have learnt about concepts definitions, pathophysiological changes, nursing process and therapeutic management of the acute tubular necrosis and bladder trauma. The high mortality accompanying acute renal failure with tubular necrosis and bladder trauma has been greatly reduced by the measures described in this unit. Prognosis depends upon the speed and efficiency with which the therapeutic measures are implemented.

2.8 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

i) Acute tubular necrosis is a complication of various conditions which are associated with marked hypotension, arteriolar constriction, severe water and salt depletion.

ii) The causes of ATN are divided to two categories as follows:

- Ischemic acute Tubular necrosis.
- Toxic Acute Tubular necrosis.

Ischemic acute tubular necrosis occurs as a result of vasodilatation associated with sepsis and when hypotension or a low cardiac output episode is prolonged, it further deteriorates by renal hypoperfusion or dehydration.

Toxic ATN occurs due to administration of radio opaque dye during an interventional or diagnostic radiological study which further causes the injury to renal endothelium.

iii) Initially urine volume is greatly reduced 200 to 500 cc/24 hr. which is oliguric phase.

- Urine contains RBC, WBC, Cast, protein
- Anorexia, nausea vomiting.
- Mental confusion, muscular twitching
- Fits, drowsiness, bleeding
- Pulmonary oedema, potassium intoxication

Check Your Progress 2

i) **Prevent or minimize renal lesion**, by treating underlying cause urgently and the appropriate solution is administered to treat the deficit

Manage oligric phase: Therapeutic management during the **oliguric phase** is to minimize the need for renal function until healing occurs and this is achieved by simple dietary restrictions.

- **Water and electrolyte balance:** This is maintained by replacing the obligatory losses of water through the skin and lungs estimated to be about 600 ml of water per day and by giving no electrolytes since none are being lost from the body. Only in febrile patient extra allowances of water is required to replace the fluid and a further supplement equal to the volume of urine passed each day should be added.
- **Proteins and calories:** Dietary protein is restricted to about 20gm/day. In case of vomiting, oral treatment is avoided and carbohydrate should be given in the form of 10 percent glucose through a large peripheral vein. When diuresis starts the concentration of blood urea tends to remain constant for a few days and then begins to fall. When this occurs you must guide your patient to consume a light diet containing 45gm protein per day and ample of fruits.
- **The intake of fluid** is also increased by the volume of previous days urinary output. On average 3 gm sodium chloride and 2gm sodium bicarbonate is needed for each liter of urine passed. Oral administration of potassium chloride is also required.

As the blood urea concentration return to normal value and renal function improve, a normal diet may be resumed.

- In case of **elevation of plasma potassium**, attempts should be made to reduce it by
  - o Using sodium or calcium charged resin which removes potassium from the body,
  - o By giving 20 units of soluble insulin subcutaneously and intravenous infusion of 50gm glucose,
  - o Controlling metabolic acidosis is done by intravenous administration of isotonic sodium bicarbonate.
- **Haemodialysis in certain patients may be anticipated especially in those.**
  - o Suffering from severe infection or massive tissue damage,
  - o Where Blood has acumulated in one of tissue spaces,
  - o Who have Rapid rise of blood urea and potassium,
  - o Daily heamodialysis may be required for several weeks before renal functions starts.

Great care for infection control and for prescribing drugs which are excreted by kidney are also exercised.

ii) Common nursing diagnoses

- Excess fluid volume related to inability to excrete water
- Activity intolerance
- Risk of injury
- Risk of infection
- Altered nutrition
- Acute confusion
- Sexual dysfunction
- Knowledge deficit

iii)

Nursing Intervention	Rationale
Excess Fluid Volume: • Monitor daily weight at same time	Weight gain represents retention of fluid
• Monitor intake- output chart	Monitors degree of fluid retention
• Assess for and report shortness of breath, tachycardia, crackles in lungs, frothy sputum, heart irregularities, hypotension, cold clammy skin	These symptoms of heart failure may accompany fluid overload
• Watch for distended neck vein	Right sided heart failure causes distended neck vein due to venous congestion
• Monitor vital signs , BP	Increase in Blood pressure s reflects fluid volume excess
• Monitor for edema	It is a symptom of fluid overload
• Monitor serum protein albumin level	Low serum protein albumin contributes to edema
• Maintain sodium and fluid restrictions as ordered. Teach patient importance of each	Sodium and fluid cause fluid retention in patients with renal failure
• **Activity Intolerance** Assess for pale mucous membrane, skin color, chest pain and dyspnea	These are signs of anemia
• Monitor for Hb and Haematocrit	Low hemoglobin indicates anemia
• Watch for signs of bleeding	Bleeding worsens anemia
• Administer blood transfusion and Epogen as prescribed	Erythropoietin stimulates production of RBC by bone marrow
• Rest and Quiet environment	Rest period decreases demand of oxygen in body
• **Risk for injury related to bleeding** Observe for and report blood in stool, bleeding from mucus membrane	Bleeding must be recognized to prevent complication
• Monitor Hb, vitals	Low Hb indicates blood loss

• Teach patient to prevent injury to self and report if any symptoms of bleeding	Injury can cause bleeding
• **Risk for infection.** Monitor for s/s of infection and report promptly to physician	Early recognition of infection and prompt treatment help to prevent complications
• Protect patient from any source if infection including visitors or roommates carrying infections	Exposure to pathogen increases risk of infection as their immunity is low
• Maintain skin integrity	Intact skin prevents infection
• Standard hand hygiene practices	It helps to control infection
• Do culture for any suspected site of infection as prescribed by physician	Culture identifies pathogens and guides treatments
• Teach patient s/s of infection	Early reporting indicates early treatment
• **Altered Nutrition** Monitor weekly weight and serum protein albumin level	Weight & lab result gives information about nutritional status
• Initiate calorie count-consult dietitian	This gives information about adequacy of diet
• Provide frequent oral hygiene	Oral hygiene enhances appetite
• Offer frequent small feeds and dietary Supplement	Oral care enhances appetite.
• Offer medications as prescribed for nausea before meal	Small feeds are better tolerated, nausea should be controlled
• Ensure bowel movement daily	Constipation interferes with appetite
• **Risk of injury related to acute confusion.** Assess and report decrease in alertness, loss of memory, delirium, muscle tremors, twitches of hands	These are symptoms of uremic encephalopathy. If present dialysis may be initiated
• Observe for restless leg, numbness, tingling, burning pain in extremities	There are signs of neuropathy and makes a patent prone to get injury
• Administer medications as prescribed	Anti depressant and anti convulsant may help neuropathy
• Protect from injury and falls	Patient with numbness is at risk for fall
• Ambulate the patient at least three times per day	Ambulation prevents calcium mobilization from bones.
• **Sexual dysfunction** Assess problems related to sexual function	Patient may not share this problem unless discussed
• Offer counseling and include partner as desired by patient	Professional counselor may be contacted
• **Knowledge deficit.** Assess patient and family's knowledge of disease and treatment regimen	A baseline arrangement provides direction for instruction
• Consult dietitian, clinical specialist, social worker if needed	Specialist may be consulted for proper management

Check Your Progress 3

Therapeutic and Surgical Management

- Extra peritoneal injury can be managed by catheter drainage of bladder
- Intra-Peritoneal injury requires laprotomy with bladder repair, peritoneal lavage followed by supra pubic and bladder drainage

Check Your Progress 4

After assessment nursing diagnoses will be as follows:

- Risk of injury- related to surgical procedure.
- Risk of infection
- Risk of impaired skin integrity
- Pain related to surgical intervention
- Constipation
- Anxiety.

UNIT 3 DIABETIC KETO ACIDOSIS, HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC SYNDROME AND HYPOGLYCEMIA

Structure

3.0 Introduction

3.1 Objectives

3.2 Diabetic Ketoacidosis

- 3.2.1 Definition and Causes
- 3.2.2 Pathophysiology
- 3.2.3 Assessment, Clinical Manifestation and Diagnosis Tests
- 3.2.4 Nursing Diagnosis and Management

3.3 Hyperglycemic Hyperosmolar Nonketotic Syndrome

- 3.3.1 Definition and Causes
- 3.3.2 Pathophysiology
- 3.3.3 Assessment Clinical Manifestation and Diagnosis
- 3.3.4 Nursing Management of a Patient with Hyperglycemic Hyperosmolar Nonketotic Syndrome

3.4 Hypoglycemia

- 3.4.1 Definition and Causes
- 3.4.2 Phathophysiology
- 3.4.3 Assessment
- 3.4.4 Clinical Manifestation
- 3.4.5 Immediate Management
- 3.4.6 Nursing Intervention
- 3.4.7 Prevention of Hypoglycemia

3.5 Let Us Sum Up

3.6 Glossary

3.7 Answers to Check Your Progress

3.0 INTRODUCTION

In the previous unit 2, you have learnt about the management of the patients with acute tubular necrosis and bladder trauma. In this unit we will discuss about critically ill patients with problems of endocrinal system such as Diabetic Ketoacidosis(DKD), Hyperglycemic, hypersomolar non-ketotic syndrome (HHNS) and hypoglycemia.

While working in causality, you must have seen known cases of diabetic patients coming in unconscious state. These patients may either have DKA, HHNS or hypoglycemia. All these three conditions are critical illnesses that require immediate management. Patients with these acute complications require immediate identification, careful monitoring and prompt nursing and medical interventions so as to prevent irreversible brain damage or even death.

When studying about DKA, HHNS and Hypoglycemia we will also study various assessments, nursing diagnosis and interventions needed for patients suffering from each of these disorders.

3.1 OBJECTIVES

After completing this unit, you will be able to:

- explain the meaning of Diabetic Ketoacidosis, Hyperglycemic Hyperosmolar Nonketotic Syndrome and Hypoglycemia;
- describe pathophysiology, signs and symptoms and assessment; and
- discuss nursing management based on nursing process of clients with Diabetic Ketoacidosis, Hyperglycemic Hyperosmolar, Nonketotic Syndrome and Hypoglycemia.

3.2 DIABETIC KETOACIDOSIS

In this section we shall discuss about definition and causes of diabetic ketoacidosis (DKA)

3.2.1 Definition and Causes

Diabetic Ketoacadosis (DKA) is an acute metabolic condition that occurs in uncontrolled hyperglycemia due to an absence or markedly inadequate amount of insulin. It is a life threatening complication of diabetes mellitus. Three main clinical features of DKA are:

- Hyperglycemia blood glucose levels greater than 250 mg/dl
- Dehydration and electrolyte loss
- Acidosis arterial ph below 7.3 and serum bicarbonate level below 18 mg/dl.

Causes

Diabetic Keto Audosis (DKA) is most often seen in type 1 diabetics who are dependent on insulin. It is rarely seen in type II diabetes. The major causes are:

- Infection
- Interruption of Insulin administration or change in the type of insulin
- Major emotional crisis or life cycle changes
- Decrease in usual amount of exercise
- Increased food intake over a period of time
- Undiagnosed diabetes presenting in DKA
- DKA may also be precipitated by acute pancreatitis
- Diabetes causes of DKA are starvation, ketoacidoxis and alcoholic ketoacidosis.

3.2.2 Pathophysiology

Due to lack of insulin, the amount of glucose entering the cells is reduced and the liver increases glucose production (gluconeogensis). Both these factors lead to hyperglycemia. In search of energy from non-carbohydrate sources, the body begins to break down large amount of fat to obtain glucose. Fat and protein are broken down into free fatty acids and glycerol. The glycerol is released and converted into glucose. The fatty acids are further processed and converted to acetylCo enzyme A (acetylCo A) by liver. AcetylCo A unites with a substance called oxalocalate before it is transferred to peripheral tissues to be used for energy production. If the cycle were allowed to progress normally, the eventual outcome would be CO_2, H_2O and energy (as ATP and creatinine phosphate).

As lipolysis increases, free fatty acids are released and their oxidation results in excess amounts of AcetyCo A. AcetyCo accumulates because it is in excess amounts relative to the available oxaloacetate. This results increase in number of Ketone bodies or Ketoacids (acetoacetic acid and beta hydroxybutyric acid). The protein breakdown ultimately leads to a negative nitrogen balance leading to coma Acetone is found in patient's urine and is detectable on the breath. It is a spontaneous breakdown product of acetoacetic acid.

Hyperglycemia results is osmotic diuresis leading to dehydration and marked electrolyte loss. Sodium, Potassium, chloride, magnesium and phosphate are apt to be lost. Ketonemia results in metabolic acidosis. Compensatory mechanisms occur in buffering substances, the renal system and the lungs. The respiratory system is stimulated and the patient begins to breathe deeply and rapidly in an effort to blow off excess carbonic acid as water and CO_2 (you may recall that this type of respiration is called Kussmaul's respiration).

The kidneys increase their excretion of the H^+ ions by the secretion of ammonia, which is then excreted as ammonium chloride. The kidneys also excrete Ketones.

The pathophysiology of DKA is simplified in the flow chart given below:

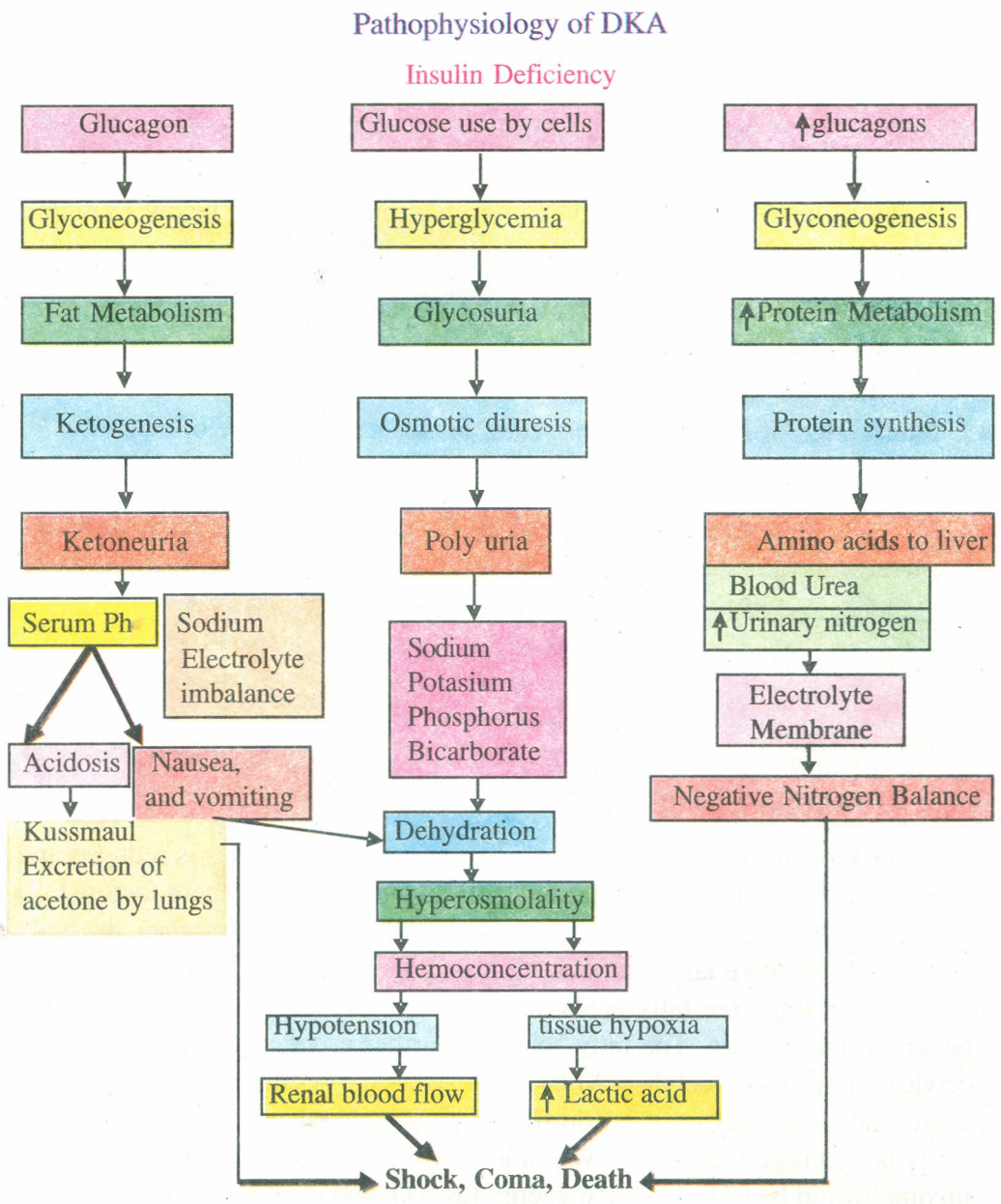

Fig. 3.1: Flow chart showing pathophysiology of DKA

3.2.3 Assessment, Clinical Manifestation and Diagnostic Tests

The diagnosis of DKA is usually made without much difficulty. If the patient is a known diabetic, the process is easier. Unique causes of coma in diabetes are hypoglycemia (insulin reaction, insulin shock) hyperosmolar nonketotic coma, lactic acidosis and DKA. A blood glucose test will quickly rule out hypoglycemia. Hyperosmolar coma is similar to DKA, but it lacks acidosis component. While in DKA hyperglycemia with lactic acidosis may be present.

The diagnosis of DKA should be based on the patient's clinical picture, history and laboratory findings. You may find your patient presenting with the clinical manifestations mentioned here.

Clinical manifestations

You will find the patient with DKA acutely ill. Significant dehydration is manifested by dry skin with poor skin turgor, thirst, soft eyeballs and wrinkled corneas. Mucus membranes in the conjunctival and oropharyngeal areas are dry. Polyuria and polydipsia are present. Anticipate orthostatic hypotension or even hypotension with weak rapid pulse. When you auscultate the lungs, a pleural friction rub may be present which may disappear on rehydration. The acidosis leads to mental changes ranging from confusion to coma and GI symptoms such as anorexia, nausea, vomiting and abdominal pain. Assessment will also reveal acidotic breath (fruity odor) and hyperventilation (Kussmaul's respiration). Fever, flushed face and diminished deep tendon reflexes are some other signs that you may identify in your patients.

Diagnostic tests

Many diagnostic tests are helpful which include following:

- Blood glucose level which may vary from 300 to 800 mg/dl. The severity of DKA is not necessarily related to the blood glucose levels.
- Arterial blood gases reveal an acidotic arterial pH.(6.8 to 7.3) values. The PaO_2 may be normal. A low $PaCO_2$ level (10 to 30 mm Hg).reflects respiratory compensation. The bicarbonate concentration will be reduced to below 18mEq//l
- Accumulation of ketone bodies is reflected in blood and urine ketone measurements.
- Sodium, potassium levels may be low, normal or high depending on amount of water loss.
- Elevated levels of creatinine, BUN, hematocrit may also be seen.

3.2.4 Nursing Diagnosis and Management

DKA is life threatening, your prompt assessment, early diagnosis and proactive management is important.

Based on the assessment data, you may formulate the following nursing diagnoses:

- Deficient fluid volume related to osmotic diuresis
- Deficient knowledge about prevention of further episodes of DKA
- Ineffective breathing pattern (Kussmaul respiration) related to lactic acidosis

- Ineffective tissue perfusion-cerebral, related to acidosis, cerebral edema
- Ineffective tissue perfusion -peripheral, renal, related to dehydration.

Other Collaborative problems may include

- Fluid overload, pulmonary edema following rehydration therapy:
  - o Hyperglycemia due to insufficient insulin to meet metabolic needs
  - o Ketoacidosis
  - o Hypokalemia
  - o Hypoglycemia following unmonitored insulin infusion
  - o Cerebral edema

The chief components of nursing and medical management include following:

- Insulin therapy,
- Fluid replacement, electrolyte replacement,
- Antiacidosis therapy, and
- Management of the symptoms.

So your major goals for these patients may include–

- Controlling hyperglycemia,
- Bringing blood glucose below 250 mg/dl,
- Maintenance of fluid and electrolyte balance,
- Reversal of acidosis and maintenance of acid base balance,
- Prevention of complications namely respiratory and renal.

Let us explain each one of these in detail as follows:

Controlling Hyperglycemia

This can be achieved by Insulin therapy

Insulin is perhaps the cornerstone of treatment. You already must be aware that Insulin may be given in large doses or small doses intermittently or in continuous infusion.

Generally insulin is given in continuous infusion to decrease blood glucose at a fairly predictable level of about 75 to 100 mg/dl/hour. For this about 5 units to 10 units/hours of regular insulin is given by use of infusion pump. The client may receive an IV bolus of regular insulin (0.15 u/kg).

Your close and accurate monitoring of the patient's blood glucose level is imperative, blood sign is maintained at about 250 to 300 mg/dl. The danger of developing hypoglycemia necessitates decreasing the insulin dose. The patients in DKA are treated with regular insulin, once the initial hyperglycemic crisis is over, the patients will likely be returned to other kinds of insulins. You will carefully evaluate the patient during the insulin therapy. Blood glucose evaluations are done every hour. After the first few hours of treatment, insulin may be given on a sliding scale. Insulin is inactivated when the pH is above 7.5. Thus, some inactivation may be seen when bicarbonate therapy is used.

Fluid Replacement

The average fluid deficit is 3L to 5L of water in an adult. Rapid rehydration is needed to maintain vascular tone and metabolic functioning. Isotonic normal saline solution (0.9%) is the initial fluid of choice despite its hyperosmolarity. Generally, 1L to 2L is given in first 2 hours or so up to 10 L of fluid is the average volume to be administered in first 12 hours. After first few hours 0.45% normal saline or 5% dextrose water may be infused instead of normal saline lactated Ringer's solution may be used to minimize chloride load and avoid hyperchloremia, Alkalinizing solutions, such as sodium bicarbonate and sodium locate are occasionally used for severe acidosis.

Electrolyte Replacement

Losses of sodium, potassium, chloride magnesium, phosphate and calcium are seen in patients with DKA. Sodium and chloride are replaced with 0.45% or 0.9% normal saline intravenous solution given rapidly. Calculation of potassium replacement is difficult. In DKA, the total body amount of potassium is deficient. However, the patent may present with high, low or even normal serum potassium level. When insulin and fluid administration are instituted, potassium is rapidly moved back into the cell and a life threatening hypokalemia can arise in a period of 1 to 4 hours of treatment. Potassium replacement should be carefully evaluated using serum potassium level. If the level is high initially, no potassium is needed.

If the potassium level is normal, Potassium chloride (20 mEq - 40 mEq/L) may be given after the initial hydration therapy. Potassium should only be given with extreme care to any patient with anuria. The electrocardiogram is a useful guide to the patient's potassium level. You may review here the ECG changes associated with potassium disturbances.

After the acute phase is over, the oral route of administering potassium is safe. Once the patient can tolerate liquids, orange juice, beef broth can be given.

Reversing Acidosis

The acidosis that occurs in DKA is reversed with insulin, which inhibits fat break down; thereby stopping acid buildup. Alkali Therapy to counteract the metabolic acidosis is usually reserved for severe acidosis only. A Ph of less than 7 indicates acidosis severe enough for sodium bicarbonate therapy. Bicarbonate administration should be done cautiously as it may contribute to lactic acidosis. When 2, 3 DPG (diphosphoglycerate, a molecule of red blood cell that releases oxygen) is low, a sudden rise in pH may reduce oxygen release to the tissues and predispose the patient to lactic acidosis.

Management of Symptoms

Other medical and nursing treatment of DKA may be instituted depending upon the needs of individual patients. The treatment includes dextran or plasma in severe hypovolemic shock. Vasopressors may be used in peripheral vascular collapse. Antibiotics are prescribed if an infection is present. Gastric intubation is used if vomiting or distension is present.

Collaborative care guide for the patient with Diabetic Ketoacidosis is illustrated in Table 3.1.

Table 3.1: Collaborative Care Guide For DKA

Outcomes	Interventions
OXYGENATION	
Arterial blood gases are normal	Chest physiotherapy, deep breathing, coughing exercises
No evidence of acute respiratory failure	Observe for RR, depth and for Kussmaul respiration Monitor ABG, pulse oximetry, end tidal volume prepare for intubation
There is no evidence of atelectasis Or pneumonia	Auscultate breath sounds, chest physiotherapy
PERFUSION	
Blood pressure, HR,& hemodynamic parameters are normal	Recording of vitals hourly. Look for neck vein distension,CVP, PAWP
Patient is free from dysrythmia hyperkalemia	Monitor ECG, treat hypoxia, acidosis,
FLUIDS	
Evidence of rehydration without complications	Infuse fluids, monitor serum osmolality, urine output neurological status and vital signs, assess BUN, urine ketones, serum glucose, serum electrolytes
MOBILITY	
Patient is free from injury	Implement seizures and fall precautions, assess neurologic status hourly. Practice range of motion exercises.
SKIN INTEGRITY	
Skin will remain intact	Reposition frequently, use pressure reduction mattress.
NUTRITION	
Calories and nutritional needs met	Provide parenteral nutrition/ full liquid are diet. Progress to diabetic diet. Assess bowel sounds 2 hourly administer antiemetics as advised. Give frequent oral hygiene
COMFORT/ PAIN CONTROL	
Patient has minimal pain	Use objective pain scale.

Check Your Progress 1

a) List down any four signs and symptoms of diabetic ketoacedosis.

i) .....

.....

.....

ii) .....

.....

.....

.....

iii) .....

.....

.....

iv) .....

.....

.....

b) Fill in the blanks:

i) Insulin is given through ..... route in the initial management of a patient with DKA.

ii) The fluid of choice in initial fluid replacement therapy for DKA is .....

iii) 5% Dextrose solution I/V should be started when blood glucose level reaches .....

3.3 HYPERGLYCEMIC HYPEROSMOLAR NON-KETOTIC SYNDROME (HHNS)

3.3.1 Definition and Causes

Hyperglycemic hyperosmolar non-ketotic syndrome (HHNS) is a complication of diabetes mellitus(DM) characterized by extreme hyperglycemia (600 to 2000 mg/dl), profound dehydration, mild or undetectable ketonuria and absence of acidosis. HHNS commonly occurs in older clients with DM type 2. Mortality with HHNS is greater than with DKA (10% to 40%). This is because clients are older and commonly have significant medical problems.

The major difference between HHNS and DKA is the lack of ketonuria with HHNS. This is because some residual ability to secrete insulin remains in type 2 DM and therefore mobilization of fats for energy does not occur.

Causes

HHNS may occur secondary to extreme stress associated with severe medical illness, pancreatitis, trauma, sepsis, burns or pneumonia or following peritoneal dialysis, certain drugs such as corticosteroids, thiazide diuretics, sedatives and sympathomimetics may cause adverse effects.

3.3.2 Pathophysiology

In the absence of adequate insulin, there is considerable hyperglycemia. This increases the osmotic pressure and water is drawn from the interstitial spaces and cells. This results in osmotic diuresis and eventually cells become dehydrated. Over a period of time you will find the patient's mental alertness diminishes with reduced sensation to pain. This is called obtundation. The flow chart 3.3 given below gives a simplified sequence of events which occur in HHNS.

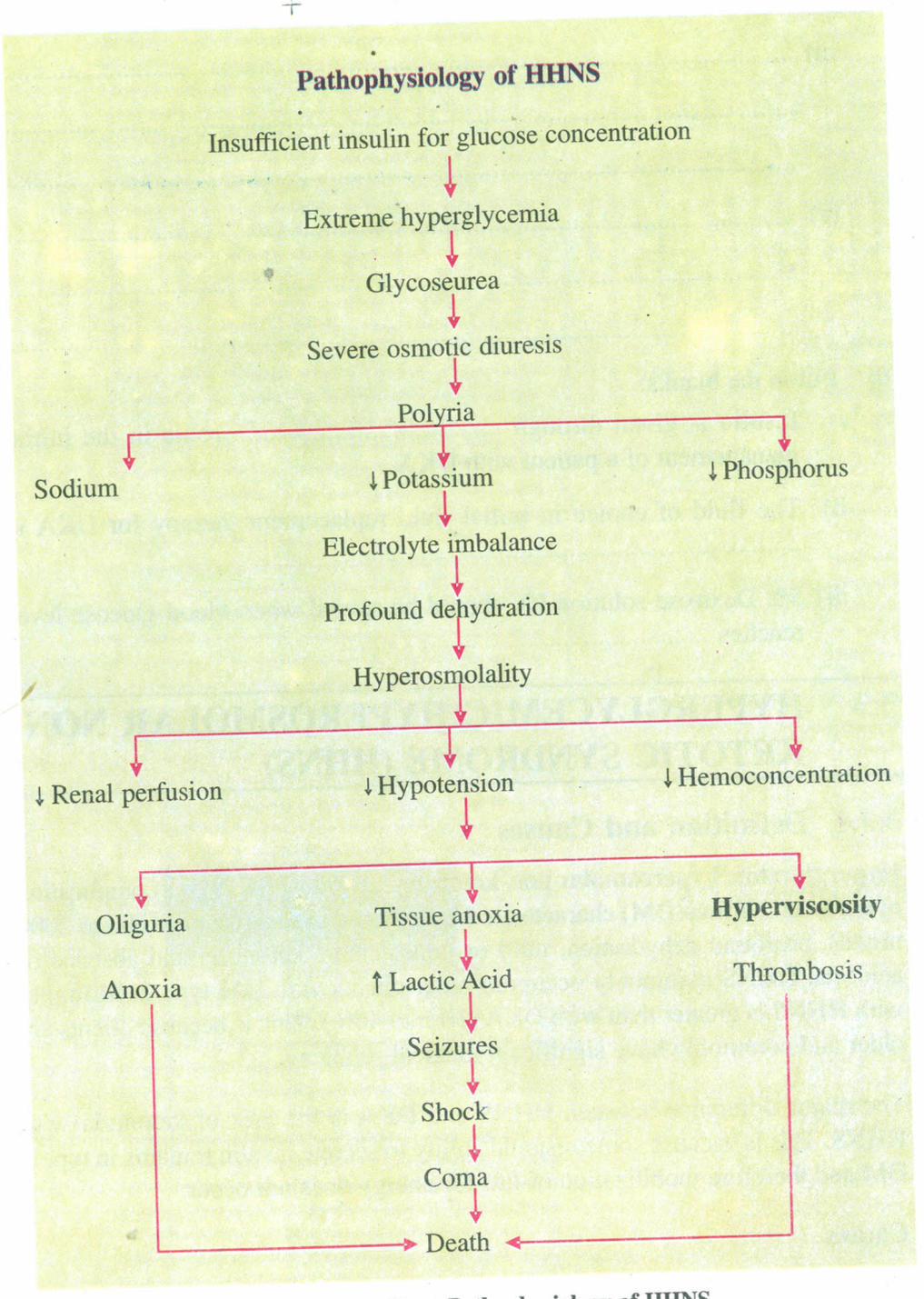

Fig. 3.2: Flow Chart Pathophysiology of HHNS

3.3.3 Assessment Clinical Manifestation and Diagnosis

History and Physical Examination

Patient has history of being drowsy for days with decreased food and fluid intake. There are 4 major clinical features:

- Severe hyperglycemia
- No or only slight ketosis
- Profound dehydration (10% - 15% loss of body water)
- Hyperosmolarity and raised BUN

The onset is gradual, usually more than 5 days

Clinical Manifestation

Sign and Symptoms may include:

- Excessive thirst
- Altered level of consciousness (or confusion)
- Dehydration
- Tachycardia
- Hypotension
- Rapid respiration **without** Kussmaul's breathing
- Warm flushed skin
- Toxic appearance.

Diagnosis

Various Laboratory studies are carried out to diagnose HHNS

Blood glucose	:	>- 600mg/dl
Serums Na	:	High
Serum osmolarity	:	> 310 to 320 mOsm/Kg
Bicarbonate	:	Normal or > 10 m Eq/l

Patient may have azotemia, hyperkalemia and lactic acidosis as well. Comparison of signs and symptoms of DKA and HHNS in Table 3.2 will guide to a quick assessment.

Diffraction Between of Diabetic Ketoacidosis (DKA) and hyperglycemic Hyperosmolar Non-Ketotic Syndrome (HHNS)

The difference between the DKA and HHNS is given in Table 3.2 below:

Table 3.2: Comparison of Signs and Symptoms of Diabetic Ketoacidosis (DKA) and hyperglycemic Hyperosmolar Non-Ketotic Syndrome (HHNS)

Signs and symptoms

	DKA	HHNS
Onset	Gradual or sudden, usually less than 2 days	Gradual, usually more than 5 days
Previous history of diabetes mellitus	85% (15% have new onset)	60%
Type of diabetes mellitus	Type 1	Type 2
Age of patient	Usually younger than 40 years	Usually older than 60 years
Mortality risk	1% - 15%	20% - 40%
Drug history	Insulin	Steroids, thiazides, oral agents

Physical signs	Dehydration, Kussmaul's respirations, mental status changes, "fruity breath," febrile at times	Dehydration, obtundation, hypothermia, toxic appearance, Kussmaul's respirations absent
Glucose level	Mean, 600 mg/dL Range, 250 – 1200 mg/dL	Mean, 1100 mg/dL Range, 400 – 4000 mg/dL
Ketones	Present	Absent
Osmolarity	Mean, 320 mOsm/L	Mean, 400 mOsm/L
Arterial pH	Mean, 7.07	Mean, 7.26
Bicarbonate	Markedly low [< 10 mEq/L]	Normal or > 10 mEq/L
Anion gap	> 12 mEq/L	< 12mEq/L

3.3.4 Nursing Management of a patient with Hyperglycemic Hyperosmolar Nonketotic Syndrome

Based on the individual assessment of clients you may arrive at some of the following nursing diagnosis:

- Deficient fluid volume related to osmotic diuresis.
- Impaired gaseous exchange related to low cardiac output, dehydration.
- Ineffective tissue perfusion – cerebral, related to cerebral edema.
- Ineffective tissue perfusion - peripheral, renal, related to dehydration.
- Risk of intravascular thrombosis related to hemoconcentration and hyperosmolarity.
- Risk of fluid overload during rehydration therapy.

Other related problems may include

- Hyperglycemia
- Hyperkalemia
- Cerebral edema

Goal of the therapy include

- immediate management of blood glucose level,
- reversal of hyperosmolar state,
- fluid replacement and correction of dehydration,
- correction of electrolytes imbalances
- prevention of complications associated with hyperosmolar states and rehydration therapies.

Therapy for HHNS is directed at following:

- correcting the volume depletion,
- controlling hyperglycemia and
- identifying the underlying cause of HHNS and treating it,
- Rehydration is cautiously done owing to the fragile state of the patient.

Isotonic saline or hypotonic saline is given initially. Some patients may require 9-12 liters of fluid overall. Patients require homodynamic monitoring especially

elderly patients with cardiac and renal disease. Low doses of insulin along with the fluid replacement are given by continuous infusion (0.1 u/kg/hour). The insulin is stopped when blood glucose level reaches 250 - 350 mg/dl rather a 5% Dextrose is started. At this point insulin can be administered subcutaneously. As in DKA, Potassium, sodium and chloride may be administered, intravenously after carefully monitoring of their level. The underlying cause of HHNS should be investigated and treated if possible. For example, chest infection should be treated aggressively with antibiotics, chest physiotherapy, turning and deep breathing and coughing exercises, exogenous sources of glucose (tube feeding, peritoneal dialysis, and medication) should be removed.

3.4 HYPOGLYCEMIA

This is not an uncommon occurrence in hospitals and I am sure you must have come across hypoglycemic episodes especially among patients who are on Insulin Therapy.

3.4.1 Definition and Causes

Hypoglycemia is also known as insulin reaction or hypoglycemic reaction. It is a well recognized complication among patients with type-1 diabetes mellitus and can also be seen in patients with type 2 diabetes treated with insulin or oral agents. The precise blood glucose level at which patients have manifestations of hypoglycemia varies, but they usually do not occur until the blood glucose level is less than 50 to 60 mg/dl.

It is observed that people with type-1 diabetes who maintained strict intensive therapy for their diabetes experienced a threefold greater incidence of severe hypoglycemia than those patients with less strict treatment protocols.

Mild hypoglycemia causes unpleasant symptoms and discomfort; however severe hypoglycemia can lead to life threatening complications such as seizures, coma and even death if not reversed. Prolonged or recurrent hypoglycemia, although uncommon, has the potential to cause permanent brain damage.

Causes

Hypoglycemic reaction may result from the following:

- An overdose of insulin or secondary to administration of sulfonylurea
- Omitting a meal or eating less food than usual
- Overexertion without additional carbohydrates compensation
- Nutritional and fluid imbalances caused by nausea and vomiting
- Alcohol intake
- Severe liver disease
- Severe sepsis
- Stress
- Drug effects
    - o Ethanol

- Salicylates
- Quinine
- Haloperidol
- Insulin
- Sulfonylurea
- Allopurinol
- Sulfonamides
- Clofibrate
- Beta adrenergic agents

3.4.2 Pathophysiology

Brain depends on glucose supplied by the circulation due to lack of its ability to burn long chain free fatty acids, lack of glucose stores in the brain and unavailability of ketones. The brain recognizes its energy deficiency when blood glucose level falls abruptly to about 45 mg/dl. The term neuroglycopenia refers to the degree of hypoglycemia sufficient to cause brain dysfunction resulting in personality changes and intellectual deterioration. The exact level at which symptoms occur varies widely from person to person. Further in clients with type-1 DM in the first 2-5 years of diabetes, the secretion of glucagons becomes deficient. Later the secretion of epinephrine may become impaired secondary to sub-clinical neuropathy. Both these hormones promptly increase blood glucose in the event of hypoglycemia. But now this prompt help is not available. Combination of these abnormalities makes the patients with type-1 DM susceptible to frequent development of hypoglycemia

3.4.3 Assessment

Occasional reactions happen in even the most stable insulin dependent diabetic patient. As long as the reactions are mild they can usually be tolerated and do not require change in regimen.

History and physical examination

Ask about food intake, exercise. Note the problems with insulin dosage or administration. Look for the flaws and inconsistencies in prescription and mismatched syringes and insulin units or reporting. Ask for the history of alcohol intake or drug intake.

As the blood sugar level falls below normal, the manifestations are elicited. These are of two types as mentioned below:

3.4.4 Clinical Manifestations

i) Adrenergic manifestation. These are mild reactions due to increasing epinephrine like –

Shakiness

Irritability

Nervousness

Tachycardia, palpitations

Tremors

Hunger

Diaphoresis

Pallor

Paresthesis

ii) Neuroglycopenic manifestations

Headache

Mental illness

Inability to concentrate

Slurred speech

Blurred vision

Confusion

Irrational behavior

Lethargy

Loss of consciousness

Coma

Seizure

Death

3.4.5 Immediate Management

Main goal of management is to return the blood glucose to normal levels, prevent permanent cerebral damage and prevent further hypoglycemia attacks.

3.4.6 Nursing Interventions

Management of hypoglycemia depends on the severity of the reaction. For mild hypoglycemia give 10-15 gm of simple carbohydrate in the form of 40oz orange juice 60 oz, regular cola, 6-8 oz milk, or 4tsp sugar granulated. If it is not sufficient to relieve the symptoms, repeat the intake. For moderate hypoglycemia give 20-30 gm of carbohydrates. Injection glucagon may be given S/c or I/V by a family member at home. Glucagon injection is effective in most cases but is followed by complaints of nausea and vomiting. At this point family must seek medical assistance. Usually a patient who reaches hospital with hypoglycemia is usually having severe hypoglycemia and has altered level of consciousness. Immediately give 10-25gm of I.V. Glucose (as 50% or 25% dextrose) over 1-3 minutes. This is followed by 5-10gm/hr of 5% Dextrose infusion until the patient is fully recovered and able to eat.

The response to IV glucose is almost immediate i.e. 1-2 minutes at most. (Failure to respond fully in appropriate time indicates that not enough glucose has been given or that the diagnosis is incorrect or that the hypoglycemia has been long and severe enough to produce persistent, although not necessarily, permanent cerebral dysfunction.

3.4.7 Prevention of Hypoglycemia

You role in educating patients and family members about timely recognition and quick action is crucial.

You need to ensure that all patients must understand the following:

Why hypoglycemia occurs

When are the peak effects of their drugs or insulin so as to predict high risk times for hypoglycemia?

- Early manifestations of hypoglycemia.
- The danger of severe or repeated reactions.
- The importance of early intervention.
- How to prevent hypoglycemia.

Teach all people with diabetes to report hypoglycemic reactions to their health care provider for adjusting their regimen. They should always carry identification and medical alert information with them at all the time.

Check Your Progress 2

a) The expanded form of HHNS is .....

b) HHNS is clinically seen in type ..... DM patients.

c) Patient with HHNS will have excessive loss of ..... due to hyperosmolar state.

d) Manifestations resulting in decreased CNS functioning due to hypoglycemia are called .....

3.5 LET US SUM UP

You have studied that diabetes mellitus can result in major life threatening conditions like DKA, HHNS and hypoglycemia. While DKA is seen in DM Type-1, HHNS is seen in Type-2 DM. Hypoglycemia is found mostly in type1 DM but can be seen in both type of patients.

Fluid and electrolyte replacement, and controlling hyperglycemia by insulin infusion are cornerstone of Management of DKA and HHNS whereas in Hypoglycemia the patient should be given oral glucose/sugar if s/he is consciousness or I/V glucose if patient is confused or obtunded.

3.6 GLOSSARY

Hyperglycemia	:	Blood glucose level more than normal i.e. above 120 mg/dL.
Lipolysis	:	Breakdown of fats for energy production.
Ketonuria	:	Presence of Ketone bodies in the urine.
BUN	:	Blood urea Nitrogen
Neuroglycopenic	:	Manifestations associated with lack of manifestations glucose availability to the brain.

3.7 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

a) Dehydration, hyperventilation, hypotension, polyuria.

b) i) intravenous infusion

ii) normal saline

iii) 250 mg/dl.

Check Your Progress 2

i) Hyperglycemic hyperosmolar nonketotic syndrome.

ii) Type 2 DM.

iii) Fluid.

iv) Neuroglycopenic manifestations

UNIT 4 THYROID CRISIS, MYXEDEMA COMA, ADRENAL CRISIS AND SYNDROME OF INAPPROPRIATE HYPERSECRETION OF ANTI DIURETIC HORMONE

Structure

4.0 Introduction

4.1 Objectives

4.2 Thyroid Crisis

- 4.2.1 Definition and Causes
- 4.2.2 Precipitating Factors
- 4.2.3 Pathophysiology
- 4.2.4 Assessment Clinical Features Diagnostic Studies
- 4.2.5 Nursing Management of Patient with Thyroid Crisis

4.3 Myxedema Coma

- 4.3.1 Precipitating Factors
- 4.3.2 Pathophysiology
- 4.3.3 Assessment and Diagnosis
- 4.3.4 Nursing Management of the Patient with Myxedema Coma

4.4 Adrenal Crisis

- 4.4.1 Definition and Causes
- 4.4.2 Pathophysiology
- 4.4.3 Assessment, Clinical Manifestation and Laboratory Studies
- 4.4.4 Nursing Process of the Patient with Adrenal Crisis

4.5 Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)

- 4.5.1 Definition and Causes
- 4.5.2 Pathophysiology
- 4.5.3 Assessment and Investigations
- 4.5.4 Management of Patient with SIADH

4.6 Let Us Sum Up

4.7 Glossary

4.8 Answers to Check Your Progress

4.0 INTRODUCTION

In the previous unit 3 you have learnt about some of the endocrinal emergencies namely, DKA, HHNS and hypoglycemia. In this unit we will discuss some other critical conditions due to the disorder of thyroid gland, adrenal gland and pituitary

gland. These include thyroid crisis, myxedema coma, Adrenal Crisis and syndrome of inappropriate antidiuretic hormone secretion (SIADH).

Endocrine disorders have multisystem effects. At the same time, acute illnesses may lead to hypofunction of neuroendocrine system. Patients with acute illness who are at risk of endocrinal dysfunction may have a pre-existing endocrinal disorder. However, many a times these are not recognized until acute illness develops. For this reason endocrinal dysfunction should be considered in assessment and management of all critically ill patients.We shall focuss on definition, causes, pathophysiology, assessment, clinical features, diagnosis and management including nursing management of patients with thyroid crisis, myxedema coma, Adrenal Crisis and syndrome of inappropriate antidiuretic hormone secretion (SIADH).

4.0 OBJECTIVES

After completing the unit, you will be able to:

- explain the pathophysiological principles involved in the endocrinal diseases like thyroid crisis, myxedema coma, adrenal crisis, and syndrome of inappropriate antidiuretic hormone secretion;
- distinguish causes, history and clinical manifestations presented in each disorder;
- discuss the laboratory studies that are useful in diagnosing each of the diseases;
- analyze the similarities and differences in caring for patients with endocrine crisis; and
- describe the Nursing role in assessing and managing the patients with endocrine crisis.

4.2 THYROID CRISIS

We shall discuss about definition, causes, preciptating factors and other aspects of thyroid crisis as follows:

4.2.1 Definition and Causes

Thyrotoxic crisis (you would have heard the terms thyroid storm, thyrotoxicosis) is a severe form of hyperthyroidism often associated with physiological or psychological stress. When the thyroid state worsens critically, it is called thyroid crisis or thyrotoxicosis.

It is a potentially fatal acute episode of thyroid over activity characterized by high fever, severe tachycardia, delirium, dehydration and extreme irritability. These patients must be admitted to the intensive care unit for supportive measures, antithyroid medications, steroids and continuous nursing care. Now why do these critical conditions occur? Read on to find out.

4.2.2 Precipitating Factors

There are many precipitating factors that lead to a thyroid crisis some of them are given below:

- Undiagnosed or untreated hyperthyroidism
- Infections
- Thyroid ablation
- Metabolic catastrophes
- Thyroid medication over dosage
- Labour and delivery
- Inadequate preparation for thyroid surgery.
- Metastatic thyroid cancer
- Trauma
- Coexistent medical illness (e.g., Myocardial infarction, pulmonary disease).

4.2.3 Pathophysiology

The exact cause of thyroid crisis is poorly understood. But you will find any of the above mentioned factors lead to a sudden release of large quantities of thyroid hormones (TH). This results in hypermetabolism (as TH directly influences metabolic rate). This in turn leads to increased heat production, increased nutrient and oxygen consumption, negative nitrogen balance, a state of nutritional deficiency and weight loss and a catabolic state. There is alteration in fluid and electrolyte balance. Raised thyroid hormone also causes adrenergic hyperactivity. It leads to extreme tachycardia and palpitations and increased cardiac output. The many different endocrine, reproductive, gastrointestinal, integumentary and ocular manifestations are caused by increased circulating levels of thyroid hormones and by stimulation of sympathetic nervous system.

Another mechanism that may contribute to thyroid crisis is excessive lipolysis and fatty acid production. With excessive lipolysis, increased fatty acids are oxidized and produce an over abundance of heat, which is difficult to be balanced by vasodilatation.

4.2.4 Assessment, Clinical Features and Diagnosis Studies

You have to assess the patients by taking history, performing physical examination and observing science and symptoms. Refer Fig. 4.1 for palpation of thyroid glands. Ask any precipitating factors that have been listed above. Look for clues like exposure to iodine, current use of thyroid hormone, anterior neck enlargement, thyroid enlargement, protrusion of one or both eyes (exopthalmos) or family history of thyroid disease. This may indicate presence of Grave's disease (most common form of hyperthyroidism). Look for the signs and symptoms mentioned below:

Clinical manifestations

Look for signs and symptoms of hyperthyroidism that affect all body systems and these may include–

- Sweating
- Heat intolerance
- High grade fever (above 104° F)
- Tachycardia
- Anxiety and nervousness
- Tremors, restlessness
- Palpitations
- Dysrythmia
- CNS abnormalities like agitation, delirium, seizures or coma

Diagnostic Studies

In the diagnosis of thyroid crisis the following results will be found:

Serum total T_4	elevated
Serum free T_3	elevated
Serum free T_4	elevated
Serum TSH	extremely low
Electrolyte imbalances due to dehydration	
Serum Ca^{++}	elevated
Serum K^+	decreased
Serum glucose	elevated
Radio iodine uptake test	elevated
ECG	atrial fibrillation and supraventricular tachycardia, ventricular dysrythmia

4.2.5 Nursing Management of Patient with Thyroid Crisis

The main nursing diagnosis may include–

- Deficient Fluid volume related to hypermetabolic state
- Imbalanced nutrition, less than body requirement
- Hyperthermia related to hypermetabolic state
- Decreased cardiac output related to hypermetabolic state and heart failure
- Impaired cerebral tissue perfusion related to hypermetabolic state and heart failure
- Activity intolerance related to exhaustion
- Potential for injury related to altered mental status

Collaborative problems may include

- Cardiac Dysrhythmia
- Cardiogenic shock
- Angina or myocardial infarction
- Hyperglycemia and metabolic acidosis.

Planning and Nursing Interventions

The management goals for thyroid crisis are fourfold:

1) Treating the precipitating factors
2) Controlling excessive thyroid hormone release.
3) Inhibiting the thyroid hormone biosynthesis.
4) Treating the peripheral effects of thyroid hormone.

The patient must be admitted in ICU for supportive measures and continuous nursing care.

- Treatment Includes Anti-thyroid drugs to control thyroid release or biosynthesis. These are Propythiouracil and methimazole. Propythiouracil is preferred because it blocks conversion of T_4 to T_3 and binds iodine. But it can be given only orally. Iodine solutions such as sodium iodide (give IV) or potassium iodide orally are given to block the release of thyroid hormone. Emergency removal of excess of circulating hormone replacement therapy can be accomplished by plasmapharesis, dialysis or hemoperfusion adsorption.

Beta blockers like propranolol are used to decrease the symptoms due to adrenergic stimulation Digoxin, deltiazime and diuretics may be used in combination to treat cardiogenic shock.

- Oxygen is delivered to meet additional Metabolic requirements.
- Monitor heart rate and rhythm every hour.
- Monitor blood pressure and respiratory rate every hour.
- Put the patient on continuous cardiac monitoring to identify any dysrythmia or myocardial ischemia.
- Monitor body temperature every hour. Patient may be given tepid bath or covered with a cooling blanket. Acetaminophen is recommended for temperature control. Aspirin is not given as it increases free T_3 & T_4 levels.
- Assess the neurological status hourly. If patient is unconscious, make airway patent by side lying position and airway placement.
- As patient can have seizures, provide well padded side rails and prevent injury.
- Nutritional needs are increased therefore administer glucose-containing solutions, nutritional support and vitamin supplementation and sedation.

Evaluation

Thyroid storm is a potentially fatal condition. The patient with thyrotoxic crisis should recover when nursed in ICU. Patient should be free from cardiac symptoms, hypermetabolic state and show normal temperature. s/he should recover from CNS deterioration.

Check Your Progress 1

i) List the precipitating factors for thyroid crisis.

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ii) State the signs and symptoms of a patients with thyroid crisis.

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iii) List the nursing diagnosis in patients with thyroid crisis.

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iv) The metabolic rate is abnormally ..... in thyrotoxic crisis.

v) Adrenergic hyperactivity may lead to ....., ..., ..., (any three) in patients with thyrotoxicosis.

4.3 MYXEDEMA COMA

Myxedema Coma is a rare, life threatening emergency caused by extreme hypothyroidism, usually seen in patients with undiagnosed or undertreated hypothyroidism. Though rare you need to know this condition as these cases are nursed in the critical care units.

In Myxedema (moderate hypothyroidism) you will see a dry, waxy type of swelling with abnormal deposits of mucin in the skin and other tissues. The edema is of the non pitting type and is common in the pretibial and facial areas. It is commonly diagnosed in hypothyroid women in their 60s.

Myxedema coma (severe hypothyroidism) is a major complication of hypothyroidism. Although it is a rare condition, its mortality rate is very high i.e., almost approaching 100%. The condition is characterized by a drastic decrease in metabolic rate, hypoventilation leading to respiratory acidosis, hypothermia and hypotension. The patient has coma, pericardial and pleural effusion, megacolon with paralytic ileus and seizures. Death can result if severe hypoxia and hypercapnia are not reversed.

4.3.1 Precipitating Factors

Myxedema coma can occur in those untreated hypothyrodism patients who experience stress. The stressors include :

- Infection
- Respiratory failure
- Heart failure
- Cerebral vascular accident
- Trauma
- Prolonged exposure to cold
- Metabolic disturbances
- Surgery
- Seizures
- Drugs (like phenothiazines, barbiturates, opioids, anesthetics).

4.3.2 Pathophysiology

Hypothyroidism usually affects all body systems. A low basal metabolic rate (BMR) and decreased energy metabolism and heat production are characteristic. In myxedema, connective fibers are separated by an increased amount of protein and mucopolysaccharides. This binds water, producing non pitting, boggy edema, especially around the eyes, hands and feet. It is also responsible for thickening of the tongue and the Laryngeal and Pharyngeal mucous membranes, resulting in slurred speech and hoarseness.

4.3.3 Assessment and Diagnosis

You will assess the patient with myxedema as follows:

a) History and Physical Examination

Refer the previous section 4.3. for the physical examination of the thyroid gland. The history should include evaluation of the precipitating factors and also the clinical features.

Myxedema coma is a rare manifestation of hypothyroidism. Among signs and symptoms of hypothyroidism you will find clients complaining of fatigue, weakness, decreased bowel sounds, decreased appetite, weight gain, and you may find ECG changes. Signs and symptoms of Myxedema coma are:

- Severe depression of the sensorium
- Hypothermia
- Hypoventilation
- Hypoxemia
- Hyponatremia
- Hypoglycemia
- Hyperreflexia
- Hypotension
- Bradycardia

b) Investigations

• T_4 and free T_4	Decreased
• **Serum Sodium**	Decreased
• Serum Potassium	Increased
• TSH	Markedly Increased

ABG :

• $PaCO_2$	Increased
• PaO_2	Decreased
• Chest X-ray	Pleural Effusion
• ECG	Bradycardia, a prolonged PR interval, and decreased amplitude of the P wave and QRS complex. Heart block may develop.

4.3.4 Nursing Management of the Patient with Myxedema Coma

The nursing diagnosis include following:

- Ineffective breathing related to decreased CNS functioning
- Decreased cardiac output related to persistent bradycardia
- Hypothermia

- Risk for injury – pleural effusion, respiratory failure
- Risk for impaired skin integrity related to Edema and dryness secondary to infiltration of fluid into interstitial spaces
- Risk for injury related to altered sensorium.

Nursing interventions

A multisystem approach is used in treating this emergency. Mechanical ventilation is used to control hypoventilation, hypercapnia, and respiratory arrest. Intravenous hypertonic normal saline and glucose correct the dilutional hyponatremia and hypoglycemia. Fluid administration plus vasopressor therapy may be necessary to correct hypotension.

Pharmacological therapy includes the administration of thyroid hormone and corticosteroids. T_4 300 to 500 µg can be given intravenously in the beginning. Subsequent dose may include 100 µg daily. Alternatively T_3 25 µg 8 hourly can be given IV for first 24 to 48 hours. Hormone replacement should be given slowly and the patient should be monitored continuously during treatment to avoid sudden increased metabolic demand and resultant myocardial infarction. Ensure that you replace Fluids and establish rewarming of the patient to avoid complications.

Some of the nursing measures that you need to take are given below:

- Monitor the patient's intake and output. Urine output should increase significantly during thyroid therapy. Monitor the client's weight daily.
- Assess the patient carefully for manifestations of thyrotoxicosis, such as tachycardia, increased appetite, diarrhea, sweating, agitation, tremor, palpitations, and shortness of breath.
- Notify the physician at once so that the dosage can be reduced.
- Rewarming should be done gradually using blankets and socks. Mechanical devices should not used.
- Monitor for neurological status and changes in level of consciousness
- Prevent injury by using side rails and protect during seizures
- Prevent aspiration by giving side lying position and other complications due to immobility
- Assess Cardiovascular and respiratory functions. Avoid complications related to mechanical ventilation.
- Monitor fluid administration to avoid fluid overload
- Abdominal distension may require Ryle's tube aspiration and smaller fluid administration.

As the patient recovers, your interventions should focus on patient self care and education. Patient follow-up includes a thorough investigation of how the severe Hypothyroidism occurred and how it can best be avoided in the future. Patient teaching, family follow-up, medical alert activation and involvement of community supports may be necessary for this complex patient.

Evaluation

It is expected that after successful treatment, the patient will demonstrate no evidence of heart failure, edema or skin impairment; temperature and urine output will be normal; and no further evidence of myxedema will be seen.

Check Your Progress 2

i) In hyperthyroidism the ..... hormones are released in large quantities.

ii) Metabolic rate is ..... in myxedema coma.

iii) Intravenous T_4 hormone may be prescribed in patients with .....

4.4 ADRENAL CRISIS

The Adrenal medulla is situated above each kidney. It is actually a sympathetic nerve ganglion that has sceretory cells. Stimulation results in the release of catecholamines, which although not essential for life, play in the physiologic response.

Adrenal crisis also called acute Adrenal insufficiency, is a rare but life threatening dysfunction of the adrenal cortex. Adrenal crisis may occur in patients having pre existing adrenal insufficiency and is presently under stress without appropriate hormone replacement. The stressors include surgery, pregnancy, infection, dehydration, anorexia, fever or emotional upheaval.

Adrenal insufficiency may be either primary (directly involving the adrenal gland and also called Addison's disease) or secondary (due to hypothalamic-pituitary disease).

Primary adrenal insufficiency is caused by hypofunction of the adrenal glands.

4.4.1 Definition and Causes

Advenal insufficient may be primary or secondary due to,

a) Primary adrenal insufficiency

- Autoimmune adrenalitis
- Infection with Mycobacterium Tuberculosis
- Bilateral hemorrhage of the glands secondary to sepsis
- Shock
- Metastatic malignancies
- Acquired immunodeficienciency syndrome
- Fungal infections
- Surgical adrenalectomy
- Sarcoidosis

b) Secondary adrenal insufficiency may be due to

- Abrupt withdrawal of exogenous adrenocorticotropic hormone (ACTH)
- Complication of cortisol therapy
- Pituitary infarction, surgery or irradiation

4.4.2 Pathophysiology

Acute adrenal insufficiency or adrenal crisis occurs when there is a change in the chronic condition or massive hemorrhage. In addition to the pre-existing hypoadrenalism, an infection, trauma, surgical procedure, or some extra stress occurs, precipitating acute adrenal crisis in the patient. The patient is therefore unable to meet the requirements for the increased metabolic needs as necessary for stress or illness. As the patient struggles to survive, s/he quickly depletes cortisol stores and may require exogenous replacement.

Management

Addisonian crisis is an emergency condition that requires careful monitoring in the critical care unit. Thereafter the rate of recovery is patient specific. The immediate goal of therapy is to administer the needed hormones and restore fluid and electrolyte balance. Hydrocortisone, 100 mg intravenously, is administered immediately, followed by 100 mg every 6 to 8 hours. Fluid resuscitation is also started immediately with normal saline and 5% dextrose solutions. The rate of fluid and electrolyte replacements is dictated by the degree of volume depletion, serum electrolyte levels, and clinical response to therapy. Associated medical or surgical problems may indicate the need for invasive blood pressure and hemodynamic monitoring.

The overall desired outcome of treatment is prevention of the morbidity and mortality associated with the addisonian crisis. Untreated, addisonian crisis is fatal. The cause of the crisis must be determined and treated immediately. Hypotension and electrolyte imbalance need to be corrected quickly. Rapid rehydration is essential. An isotonic solution usually corrects the volume depletion, salt depletion, and hypotension. The patient may also need oxygen, vasopressors, or volume expanders. Sodium polystryrene sulfonate (Kayexalate) may be administered orally or as an enema in combination with sorbitol. Kayexalate is a resin that releases sodium ions in exchange for potassium ions.

4.4.3 Assessment Clinical Manifestations and Laboratory Studies

Assessment includes following:

History and Physical Examination is of primary importance

Symptoms of adrenal insufficiency are the same for primary and secondary disease. Because adrenal insufficiency affects both glucocorticoids and mineralocrorticoids, many body functions are affected, including glucose metabolism, fluid and electrolyte balance, cognitive state, and cardiopulmonary status.

Clinical Manifestations

- Aldosterone deficiency
  - o Hyperkalemia

- Hyponatremia
- Hypovolemia
- Elevated (BUN)

- Cortisol deficiency
  - Hypoglycemia
  - Decreased gastric motility
  - Decreased vascular tone
  - Hypercalcemia
- Generalized signs and symptoms
  - Anorexia
  - Nausea and vomiting
  - Abdominal cramping
  - Diarrhea
  - Tachycardia
  - Orthostatic hypotension
  - Headache, lethargy
  - Fatigue, weakness
  - Hyperkalemic electrocardiographic changes
  - Hyperpigmentation.

Laboratory Studies

Laboratory values in acute conditions of glucocorticoid and mineralocorticoid deficiency show deranged serum levels like hyponatremia, hyperkalemia, decreased serum bicarbonate levels, and elevated blood urea nitrogen (BUN). Metabolic acidosis may occur because of dehydration. Hypoglycemia is usually present. Other abnormal laboratory findings include anemia and lymphocytosis with eosinophilia. In primary adrenal insufficiency, the patient presents with chronically elevated ACTH levels. ACTH levels are normal or decreased in the patient with secondary adrenal insufficiency.

In primary adrenal insufficiency, repeated injections of ACTH (Or Cortrosyn) do not cause a rise in cortisol levels because the adrenal gland is dysfunctional. In secondary adrenal insufficiency, ACTH injections cause a normal but delayed response.

A computed tomography (CT) scan and MRI of the adrenal glands and the head may be done to detect tumors or other pathology of the adrenal and pituitary gland.

4.4.4 Nursing Process in the Patient with Adrenal Crisis

The various nursing diagnosis that you as a critical care nurse can formulate are as follows:

- Fluid volume deficit related to Aldosterone deficiency
- Electrolyte disturbances (hyperkalemia, hyponatremia) related to Aldosterone deficiency.
- Hypoglycemia related to cortisol deficiency.
- Risk for injury related to altered mental status.
- Infection related to decreased inflammatory response secondary to cortisol deficiency.

Nursing interventions

- Monitor signs and symptoms of electrolyte imbalance (hyponatremia and hypercalcemia) and respiratory and cardiovascular function.
- Look for changes in blood pressure, heart rate and rhythm, skin color and temperature, capillary refill time, and central venous pressure (CVP).
- Watch for orthostatic hypotension, bradycardia and dysrhythmias.
- Monitor neuromuscular signs, such as weakness, twitching, hyperreflexia, and paresthesia.
- Replace fluid and electrolytes as prescribed according to the degree of volume depletion, serum electrolyte levels.
- Evaluate Hypoglycemia and correct with intravenous (IV) glucose (5% dextrose [D_5] solution IV or IV glucose bolus).
- Replace Steroids as prescribed. Hydrocortisone 100 mg is administered as an intravenous (IV) bolus followed by 100 mg IV every 8 hours for 24 hours. The IV hydrocortisone is tapered as the patient's status dictates.
- Provide emotional support and quiet environment to assist the patient emotionally through the physiological crisis.

With rapid, efficient intervention, addisonian crisis usually resolves within 12 hours. The client's condition stabilizes, and the convalescent period begins. Once the acute crisis is over, patient education is a goal of care. You must give importance to Patient education because the ultimate prognosis depends on the patient's ability to understand and follow through with self-care. Self-care includes knowing the medication regimen, stress factors and their effect on the disease, and the signs of impending crisis; wearing a medical alert tag, bracelet, or carrying a wallet card; and taking medication as prescribed.

Evaluation

The outcome of treatment for addisonian crisis is easily evaluated, usually within 12 hours of treatment. By this time, the client should no longer exhibit any manifestations of addisonian crisis or death would have occurred.

4.5 SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION (SIADH)

SIADH is a condition characterized by excess of Antidiuretic hormone production or secretion from posterior pituitary and other ectopic sources. It produces severe fluid and electrolyte imbalances and adverse neurological changes.

ADH as you already know is synthesized in the hypothalamus and stored in the posterior pituitary. It is released when stimulated by specific conditions and causes the renal tubules to reabsorb more water and sodium.

4.5.1 Definition and Causes

There are multiple causes of SIADH. They are as follows:

a) **Malignancies**
- Bronchogenic carcinoma
- Pancreatic carcinoma
- Prostate or thymus cancer

b) **Central Nervous System (CNS) Causes**
- Neoplasm
- Hemorrhage
- Trauma
- Infection

c) **Pulmonary Causes**
- Mechanical ventilation
- Chronic obstructive pulmonary disease (COPD)
- Respiratory failure
- Lung abscess

d) **Medications**
- Nicotine
- Morphine
- Chlorpropamide
- Antineoplastics
- Tricyclic antidepressants
- Anesthetics
- Clofibrate

e) **Other Causes**
- Human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS)
- Senile atrophy
- Idiopathic.

4.5.2 Pathophysiology

In SIADH, there may be either increased secretion or increased production of ADH. This increase in ADH occurs despite the fact that the initial osmolality is normal. As a result, it causes an increase in total body water. SIADH is considered whenever the patient experiences hypotonic hyponatremia, the hallmark of the disorder.

The secretion of ADH is considered "inappropriate" if it continues despite the decreased osmolality of the plasma. The normal feedback system regulating the release and inhibition of ADH fails, and ADH secretion continues. The circulating ADH acts on the renal tubules, causing reabsorption of water that is inconsistent with the body's needs. Other reasons for the continued secretion of ADH are also lacking. There is no hypokalemia and edema; cardiac, renal, and adrenal function are normal; and there is normal or expanded plasma and extracellular fluid (ECF) volumes. Bronchogenic and pancreatic carcinoma result in secretion of ADH, independent of normal physiological controls.

Key features of SIADH you will sees are water retention, hyponatremia, hypo-osmolality. why does this happen? A continual release of ADH causes water retention from renal tubules and collecting ducts; extra-cellular fluid volume increases with dilutional hyponatremia; hyponatremia suppresses renin and aldosterone secretions causing a decrease in proximal tubule reabsorption of Sodium (Na^{+}).)

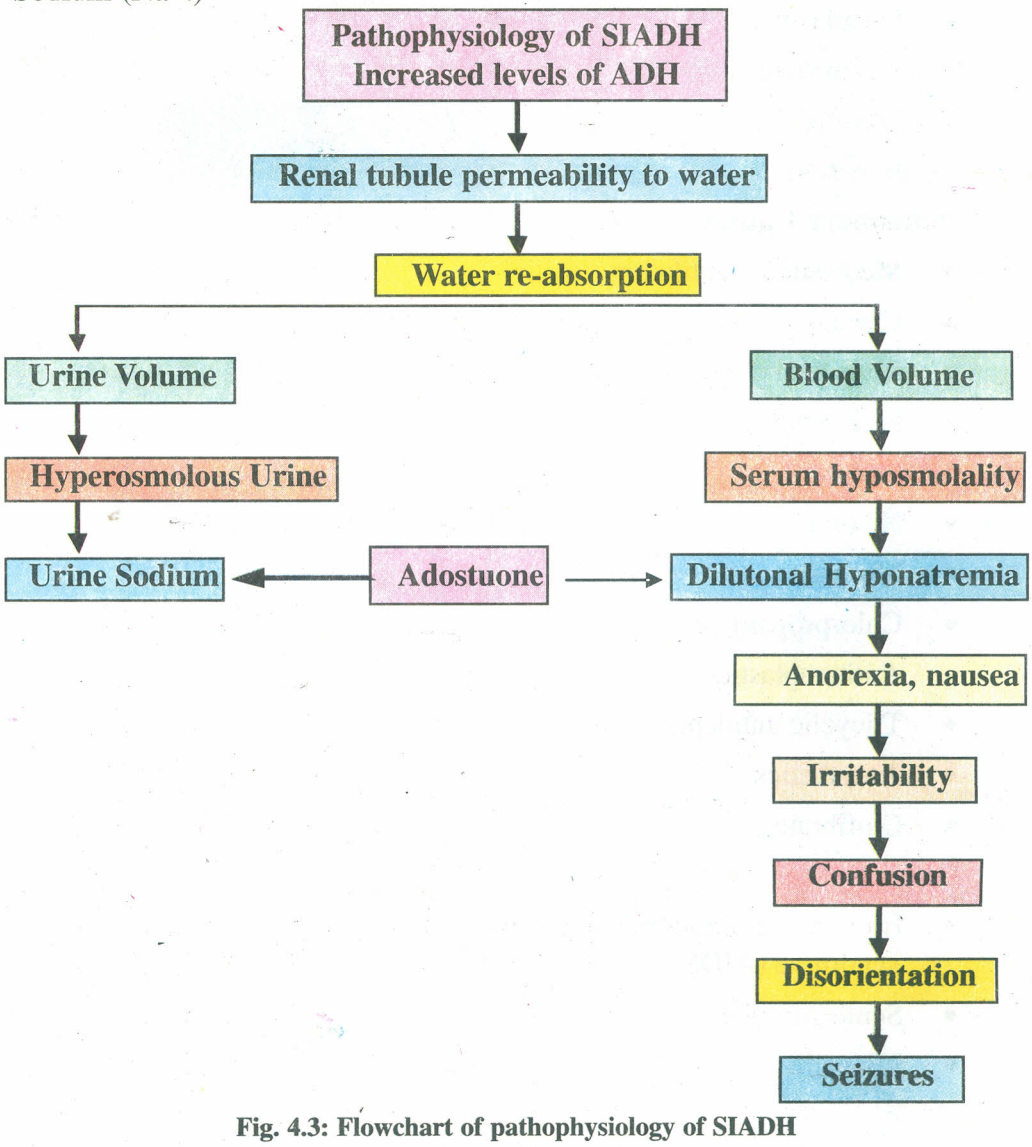

Fig. 4.3: Flowchart of pathophysiology of SIADH

4.5.3 Assessment and Investigations

You will find that SIADH is characterized by water retention and eventually water intoxication secondary to sustained ADH effect. The signs and symptoms produced by SIADH are predominantly neurological and gastrointestinal. Signs and symptoms you need to look for are as follows:

- Personality changes
- Headache
- Decreased mentation
- Lethargy
- Abdominal cramps
- Nausea, Vomiting
- Diarrhea
- Anorexia
- Decreased tendon reflexes
- Disorientation, confusion
- Seizures
- Coma

Physical evidence of hyponatremia includes dyspnea, jugular venous distension, restlessness, hypothermia, weight gain, edema, reduced and concentrated urine, and disorientation. You as a nurse will be the first to identify these early, subtle signs.

Investigations

The diagnostic evaluation of a patient with SIADH includes following:

Urine specific gravity	-	High (> 1.025)
Total Urine Output	-	Decreased (< 30 ml/hr)
BUN	-	Decreased
Creatinine	-	Decreased
Hb	-	Decreased
Hematocrit	-	Decreased
Plasma ADH	-	Increased

4.5.4 Management of Patient with SIADH

There are three goals in the management of SIADH: (1) treating the underlying disease, (2) alleviating excessive water retention, and (3) providing the comprehensive care needed when the patient has a depressed level of consciousness. No drug completely inhibits the release of ADH from the pituitary gland or a tumor. When the cause of the SIADH is unknown, the treatment consists of fluid restriction.

Nursing interventions : You have to work collaboratively to achieve the above goals.

- Restrict fluid intake. It slows renal blood flow and glomerular filtration, enhancing proximal tubular reabsorption of salt and water; increases

aldosterone secretion; and enhances distal tubule sodium reabsorption. Water intake should not exceed urinary output until the serum sodium concentration normalizes.

- Administer 3% hypertonic saline and furosemide as advised to correct hyponatremia in an emergency situation. Hypertonic saline should be infused at 0.1 mg/kg/minute to prevent rapid volume overload and pulmonary edema. Usually, less than 1,000 mL is given at one time. Rapid correction of hyponatremia may lead to brain dehydration, cerebral bleeding, demyelination, neurological injury, or death.
- Demeclocycline, an antibiotic, phenytoin, lithium and fludrocortisone may be administered as advised as it interferes with ADH effect in kidney tubules.
- Monitor fluid and electrolyte balance by intake and output hourly chart, serum sodium level and signs of fluid overload.
- Evaluate Neurological status.
- Look for disorientation and decreasing consciousness.
- Watch for complications of SIADH which may be neurological deterioration leading to seizures, coma, and death.
- Provide good oral care and you may offer substitutions for fluids (e.g., Toothettes, lemon-glycerin swabs) to help clients overcome thirst since fluids you have to restrict.

Evaluation

Prognosis depends upon cause and sodium level and serum osmolarity. It is poor for patients with bronchogenic carcinoma. Seizures and coma contribute to chronic brain dysfunction. Patient should be free from signs of hyponatremia and complications of fluid retention.

Check Your Progress 3

i) Addison's disease is also called as .....

ii) Clinical manifestation of Adrenal crisis include .....,
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iii) Syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a condition characterized by ..... of Antidiuretic hormone secretion.

iv) The key features of SIADH

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4.6 LET US SUM UP

In this unit we have discussed about thyrotoxicosis, myxedema coma, adrenal crisis ands syndrome of appropriate anti diuretic hormone secretions Thyroid, Adrenal and pituitary disorders are not common but are extremely complex and diverse. Most of these conditions are acute and affect many body systems, whereas others become chronic and lead to a wide variety of other problems. The conditions require immediate identification and prompt management under expert supervision. Patients require admission in ICU and strict hemodynamic monitoring.

4.7 GLOSSARY

Hyperthyroidism : Excessive secretion of thyroid hormone.

Hypothyroidism : Deficiency of thyroid hormone.

Paralytic ileus : Lack of peristaltic activity.

4.8 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

i) Thyroid

ii) Increased

ii) Tachycardia, Palpitation, Dysrythmia

iv) Decreased

v) Myxedema coma

Check Your Progress 2

i) Primary adrenal insufficiency

ii) Nausea/vomiting, diarrhea, orthostatic hypotension, fatigue

iii) Excess

iv) Decreased.

Check Your Progress 3

i) Adrenal insufficiency Adrinal crisis

ii)
- Aldosterone deficiency
  - Hyperkalemia
  - Hyponatremia
  - Hypovolemia
  - Elevated (BUN)
- Cortisol deficiency
  - Hypoglycemia
  - Decreased gastric motility
  - Decreased vascular tone

- o Hypercalcemia
- Generalized signs and symptoms
  - o Anorexia
  - o Nausea and vomiting
  - o Abdominal cramping
  - o Diarrhea
  - o Tachycardia
  - o Orthostatic hypotension
  - o Headache, lethargy
  - o Fatigue, weakness
  - o Hyperkalemic electrocardiographic changes
  - o Hyperpigmentation

iii) Access of antiduretic hormon production

iv) a) Water relention
b) Hyponatremia
c) Hyposmolabity.

NOTES

SOHS-IGNOU/P.O. 2T/March, 2012

BNS-032 Nursing Management in Critical Care Conditions

Block 1 Care of Critically ill Patients with Problems of Gastrointestinal System

Unit 1 Acute Gastrointestinal bleeding
Unit 2 Acute intestinal obstruction, Intra abdominal Compartment Syndrome, Perforative Peritonitis.
Unit 3 Hepatic disorders – Fulminent hepatic failure and hepatic encephalopathy
Unit 4 Acute Pancreatitis

Block 2 Care of Critically Ill Patients with Problems of Respiratory System

Unit 1 Acute Respiratory Distress Syndrome
Unit 2 Chest trauma (Hemothorax, Pneumothorax Chylothorax, Pyothorax, Hydrothorax)
Unit 3 Pulmonary Edema, Pulmonary Embolism, Atelectasis
Unit 4 Acute exacerbation of Chronic Obstructive Pulmonary Diseases And Status Asthmatics
Unit 5 Interstitial Lung Disease, Pneumonia, Pleural Effusion

Block 3 Care of Critically Ill Patients with Problems of Cardiothoracic System

Unit 1 Acute Coronary Syndrome
Unit 2 Hypertensive Crisis
Unit 3 Cardiac dysrrhythemia and cardiac arrest
Unit 4 Conductive Disturbances and Heart Block (Pacemaker)
Unit 5 Heart Failure, Aneurysm (Aortic LV)
Unit 6 Open heart surgery, cardiac Tampanade and Heart Transplantation

Block 4 Care of Critically Ill Patients with Problems Of Nervous System

Unit 1 Altered Sensorium And Increased Intracranial Pressure (↑ ICP)
Unit 2 Cerebro-vascular Accident (Stroke)
Unit 3 Traumatic Brain injury, Spinal Cord Injury and Statin Epileptics[
Unit 4 Myasthenic Crisis Multiple Scterosis And Guillain Barre Syndrome

Block 5 Care of critically ill patients with problem of renal and endocrine system

Unit 1 Care of Patients With Renal Transplantation
Unit 2 Acute Tubular Necrosis And Bladder Trauma
Unit 3 Diabetic Ketoacidosis
Unit 4 Thyroid crisis, Myxedema coma, Adrenal crisis and Syndrome of inappropriate hypersecretion of anti diuretic hormone (SIADH))

Block 6 Care Of Critically Ill Patients With Various Emergency Conditions

Unit 1 Burn
Unit 2 Poly Trauma (Multiple Organ Failure /organ transplantation/ triage/coding)
Unit3 Shock, Septicemia, Multiple Organ Dysfunction Syndrome, Disseminated Intravascular Coagulation, Status Epilepticus
Unit 4 Drug Over Dose And Poisoning, Anaphylaxis
Unit 5 Drowning

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