NURSING CARE OF CLIENTS EXPERIENCING STRESSORS OF ENDOCRINE FUNCTION

NR 40
FLORENCE MULLARKEY

ENDOCRINE FUNCTION: ANATOMY AND PHYSIOLOGY

- IDENTIFY AND LOCATE THE MAJOR ENDOCRINE ORGANS
  - HYPOTHALAMUS
  - PITUITARY:
    - ANTERIOR/ POSTERIOR
  - THYROID
  - PARATHYROID
  - ADRENAL

KEY QUESTIONS

- WHAT DO THESE ORGANS HAVE IN COMMON?

HORMONES

- CHEMICAL MESSENGERS

- RELEASE IS STIMULATED BY
  - OTHER HORMONES
  - IONS/NUTRIENTS
  - NERVE FIBERS

HORMONES

- ACT ON TARGET CELLS
  The receptor site on the target cell responds only to specific hormones....

- TRIGGER A CELLULAR RESPONSE

FEEDBACK SYSTEM

- HORMONE CONCENTRATION IS REGULATED BY NEGATIVE FEEDBACK
- A HORMONAL SETPOINT IS PREDETERMINED.
- RISE ABOVE SETPOINT DECREASES PRODUCTION
- DROP BELOW SETPOINT INCREASES PRODUCTION

HYPOTHALAMUS
RELEASING HORMONES THAT TARGET THE ANTERIOR PITUITARY

8  □ HYPOTHALAMUS

INHIBITING HORMONES PRODUCED BY THE HYPOTHALAMUS

9  □ The master gland
   …The pituitary gland

10  □ ANTERIOR PITUITARY HORMONES
    • TSH (THYROID STIMULATING HORMONE)
    • ACTH (ADRENOCORTICOTROPIC HORMONE)
    • FSH (FOLLICLE STIMULATING HORMONE)
    • PRL (PROLACTIN)
    • GH (GROWTH HORMONE)
    • MSH (MELANOCYTE-STIMULATING HORMONE)

11  □ POSTERIOR PITUITARY HORMONES
    These hormones are synthesized in the hypothalamus and are stored in the posterior pituitary gland.
    • VASOPRESSIN (ANTIDIURETIC HORMONE/ADH)
    • OXYTOCIN

12  □ NURSING CARE OF CLIENTS WITH THYROID/PARATHYROID DYSFUNCTION

13  □ THE THYROID GLAND DOES THE WORK OF RELEASING THYROID HORMONES
    – It is directly influenced by TSH produced by the anterior pituitary gland.
    – It is indirectly influenced by TRH released by the hypothalamus.

14  □ THYROID
    • THYROXINE (T4)
      – SECRETED IN RESPONSE TO TSH
    • TRIIODO-THYRONINE (T3)
      – CONVERTED FROM THYROXINE AT TARGET TISSUE

15  □ HYPOTHALAMUS/PITUITARY AXIS
    • THESE STRUCTURES RELEASE HORMONES THAT INHIBIT/STIMULATE THYROID FUNCTION
– **HYPOTHALAMUS:**
  • **TRH, SOMATOSTATIN**
– **ANTERIOR PITUITARY:**
  • **TSH**

16 **THYROID**

1  ● **CALCITONIN**

   ● (the serum calcium decreases)
   ● **DECREASES BONE RESORPTION**
     – increases calcium uptake in bone
   ● **DECREASES KIDNEY RESORPTION**
     – kidneys eliminate calcium

17 **THYROID STRESSORS OF HYPER-FUNCTION**

18 **TYPES OF HYPERTHYROIDISM AND ETIOLOGY**

   ● **GRAVES DISEASE AND IMMUNE MEDIATED THYROIDITIS**
     – AUTOIMMUNE
   ● **TOXIC MULTINODULAR GOITER**
     – THYROID NODULES INCREASE RELEASE
   ● **PITUITARY HYPERTHYROIDISM**
     – PITUITARY ADENOMA RESULTING IN EXCESSIVE TSH STIMULATION
   ● **T3 THYROTOXICOSIS**
     – INCREASE IN SECRETION OF T3, CAUSE UNKNOWN

19 **Figure 64-3**

   Goiter

20 **Figure 64-1**

   Ophthalmopathy—proptosis

21 **TYPES OF HYPERTHYROIDISM AND ETIOLOGY**

   ● **EXOGENOUS HYPERTHYROIDISM**
     – TOOK TOO MUCH THYROID MEDICATION
   ● **NEOPLASM**
     – THYROID OR OTHER BODY SITE

   **THYROID STORM IS A TRUE MEDICAL EMERGENCY**

22 **PATHOPHYSIOLOGY**

   ● **DEPENDING ON ETIOLOGY**
   ● **RESULT IS TOO MUCH CIRCULATING HORMONE THAT LEADS TO;**
– EXCESSIVE PROTEIN BREAKDOWN
– LOSS OF FAT STORES
– IMPAIRED GLUCOSE METABOLISM

23  GRAVES DISEASE
● INCIDENCE AND ETIOLOGY
  – MOST COMMON CAUSE OF HYPERTHYROIDISM
  – ASSOCIATED WITH AUTOIMMUNE ABNORMALITY
  – FAMILIAL
  – WOMEN>MEN
  – INCREASED INCIDENCE OF PRESENTATION IN AGE UNDER 40

24  GRAVES DISEASE
● CLINICAL MANIFESTATIONS
  – HEAT INTOLERANCE
  – INSOMNIA
    • FATIGUE MAY BE PRESENT
  – VISUAL CHANGES(EXOPHTHALMOS)
  – WEIGHT LOSS
    • INCREASED APPETITE
    • INCREASED FREQUENCY OF BOWEL MOVEMENTS
  – CHANGES IN MENSTRUAL CYCLE
  – PALPITATIONS, CP IN OLDER CLIENTS

25  Figure 64-2
Exophthalmos

26  GRAVES DISEASE
● OBJECTIVE ASSESSMENT
  – VITAL SIGNS
  – NEURO
  – HEENT
  – NECK
  – RESPIRATORY
  – CV
  – GI
  – MS
  – REPRODUCTIVE
  – INTEGUMENT

27  TOXIC MULTINODULAR GOITER
● INCIDENCE AND ETIOLOGY
  • WOMEN AGE 60-70
  • INDEPENDENTLY FUNCTIONING THYROID NODULES THAT MAY BE EITHER BENIGN OR MALIGNANT
● CLINICAL MANIFESTATIONS
  • SLOWER TO DEVELOP
  • REPORTS GOITER PRESENT NUMBER OF YEARS
● OBJECTIVE ASSESSMENT
  • SMALL PALPABLE DISCRETE NODULES
28  THYROID CRISIS
   ● INCIDENCE AND ETIOLOGY
     • UNTREATED/OR YET TO BE DIAGNOSED GRAVES DISEASE/HYPERTHYROIDISM
     • PRIMARY STRESSOR
       • INFECTION, TRAUMA
       • INADVERTANT SURGICAL MANIPULATION OF THE THYROID GLAND
   ● CLINICAL MANIFESTATIONS
     • PROFOUND SYMPTOMS OF HYPERTHYROIDISM
   ● TRUE MEDICAL EMERGENCY

29  THYROID CRISIS
   ● OBJECTIVE ASSESSMENT
     – ALTERED MENTAL STATUS
       • AGITATION PROGRESSING TO SWIZURES
     – HYPERTHERMIA (102-106) F
     – ELEVATED SBP
     – TACHYCARDIA
     – RESPIRATORY DISTRESS
     – ABDOMINAL PAIN, NV/DIARRHEA
   ● TRUE MEDICAL EMERGENCY

30  ASSESSMENT OF THYROID FUNCTION
   DIAGNOSTIC TESTS: HYPERFUNCTION

31  ASSESSMENT OF THYROID FUNCTION
   ● RAI UPTAKE TEST
     – RADIOACTIVE IODINE TEST
       • THYROID SCAN
       • ORAL/IV RAD IODINE
       • SCAN 24 HOURS AFTER ADMINISTRATION
       • INCREASED UPTAKE SEEN IN GRAVES
   ● THYROID SUPPRESSION TEST
     – BASELINE THYROID TESTS PERFORMED
     – PT TAKES TH FOR 7-10 DAYS
     – REPEAT ASSAYS SHOULD SHOW SUPPRESSION

32  MANAGEMENT OF HYPERTHYROIDISM
   Discuss NIC and NOC for each:
   ● Risk for decreased cardiac output
   ● Risk for hyperthermia
   ● PC: altered nutrition < body requirements
   ● Activity intolerance
   ● Sensory perceptual alteration-vision
   ● Body image disturbance

33  OVERVIEW OF MEDICAL MANAGEMENT
   ● REDUCE TH SYNTHESIS
     – PTU/TAPAZOLE
● ELIMINATE SYMPTOMS
  – BETA BLOCKADE TX
● REDUCE THYROID SIZE/ VASCULARITY
  – RADIOACTIVE IODINE
  – SATURATED IODINE
    • reduces TH synthesis also
  – SURGICAL RESECTION

34 MANAGEMENT OF HYPERTHYROIDISM

1 TAPAZOLE
  PTU
    – INHIBIT TH PRODUCTION

BETA BLOCKERS
  – PRO-PRANOLOL TO TREAT SYMPTOMS

2 NSG CONSIDERATIONS:
  – WATCH FOR S/S OF HYPOTHYROIDISM
  – ADMIN SAME TIME EACH DAY
  – BLEEDING PRECAUTIONS
  – MAY REQUIRE LIFELONG ADMINISTRATION

NSG CONSIDERATIONS:
  – DOES NOT CORRECT DISEASE
  – HOLD FOR DECREASE HR/SBP

35 MANAGEMENT OF HYPERTHYROIDISM

1 RADIOACTIVE IODINE THERAPY
  – DESTROYS THYROID TISSUE
  – EUTHYROID IN 3-6 MO

2 NSG CONSIDERATIONS
  – ALLERGIES TO IODINE
  – ASSESS FOR POSS OF PREGNANCY
  – MAY DEVELOP HYPOTHYROIDISM

36 MANAGEMENT OF HYPERTHYROIDISM

1 IODINE SOURCES
  – INHIBIT TH SYNTHESIS
  – REDUCE VASCULARITY PRIOR TO SURGERY

2 NSG CONSIDERATIONS:
  • ALLERGIES TO IODINE
  • BLEEDING PRECAUTIONS
37  MANAGEMENT OF HYPERTHYROIDISM

- **SURGICAL OPTIONS**
  - **SUBTOTAL THYROIDECTOMY**
    - Used when the TH overproduction is a result of excess thyroid tissue
    - Vascularity is reduced prior to surgery through saturated iodine sources

38  NURSING CARE-SURGERY

- Subtotal thyroidectomy—leave functioning remnant to produce TH and prevent hypothyroidism
- If malignancy, total thyroidectomy—life-long replacement necessary
- Have client in balanced metabolic/euthyroid state
- Antithyroids, beta blockers, anti anxiety meds
- Iodine preparations—decrease vascularity

39  NURSING CARE-SURGERY

- Prevent strain on suture line, by elevating HOB, avoid abrupt turning and changing positions, support the head with hands
- Prevent, detect, relieve complications
  - Hemorrhage—bulging dressing, dampness at back of neck or on pillow, resp difficulty
  - Respiratory distress—r/t hemorrhage, edema, tetany and laryngeal spasm r/t parathyroid gland removal, tetany seen 1-7 days after surgery Tracheostomy tray at bedside
  - Laryngeal nerve damage—test voice, hoarseness may be present temporarily

40  NURSING CARE-SURGERY

- Temporary hypoparathyroidism can occur. Drop in serum calcium 24-48 hours post-op
  - Positive Chvostek’s sign think chopsticks
  - Positive Trousseau’s sign think tourniquet
  
  *Have calcium gluconate or calcium chloride on hand after surgery*

41  CARE OF POST-OP SBTL THYROIDECTOMY

42  THYROID CRISIS(STORM)

- Sometimes fatal, acute episode of thyroid overactivity, may be precipitated by acute stressors eg., infections, trauma, CV disease, characterized by:
  - High fever—often cardinal symptom (102-106)
  - Severe HTN, tachycardia, CHF
  - Delerium
  - Dehydration
  - Extreme irritability
  - Adrenocortical malfunction
  - Extreme hypermetabolism
43 MANAGEMENT OF THYROID CRISIS
- Hypothermia, ice packs, no ASA—activates thyroid hormone
- Beta blocking agents
- Digoxin and diuretics
- Quiet, calm environment
- IV fluids
- Large doses of antithyroid meds
- Adrenal corticosteroids

44 THYROID DISORDERS OF HYPO-FUNCTION

45 THYROID DISORDERS

HYPO-FUNCTION

PRIMARY
- CONGENITAL DEFECT OR SURGICAL REMOVAL
- AUTOIMMUNE THYROIDITIS
- IODINE DEFICIENCY

SECONDARY
- INSUFFICIENT STIMULATION
- PERIPHERAL RESISTANCE TO TH
- PITUITARY ADENOMA

46 HYPOTHYROIDISM

- INCIDENCE AND ETIOLOGY
  - WOMEN>MEN
  - INCREASED INCIDENCE OF PRESENTATION IN AGE BETWEEN 30-60 YEARS
  - LACK OF USE OF IODIZED SALT
  - USE OF ANTITHYROID DRUGS/SURGICAL REMOVAL
  - FAMILIAL LINK IN HASHIMOTO’S THYROIDITIS
  - AUTOIMMUNE CAUSE IN HASHIMOTO’S

47 HYPOTHYROIDISM

- CLINICAL MANIFESTATIONS
  - CONFUSION/SYNCOPE
    - LETHARGY, MEMORY IMPAIRMENT
  - COLD INTOLERANCE
  - MUSCLES ACHES/STIFFNESS
  - FATIGUE
  - WEIGHT GAIN
    - DECREASED APPETITE
    - CONSTIPATION
    - FLUID RETENTION
  - ANOVULATION/IMPOTENCE

48 HYPOTHYROIDISM

- OBJECTIVE ASSESSMENT
  - VITAL SIGNS
  - NEURO
  - HEENT
– NECK
– RESPIRATORY
– CV
– GI
– MS
– REPRODUCTIVE
– INTEGUMENT

Figure 64-4
Myxedema

ASSESSMENT OF THYROID FUNCTION
DIAGNOSTIC TEST: HYPOFUNCTION

MANAGEMENT OF HYPOTHYROIDISM
Discuss NIC and NOC
● PC: DECREASED CARDIAC OUTPUT
● Risk for ineffective respiratory function
● Risk for hypothermia
● Constipation
● Altered thought processes
● PC: fluid and electrolyte imbalance
● Body image disturbance

OVERVIEW OF MEDICAL MANAGEMENT
● THYROID REPLACEMENT THERAPY
  – INCREASE LEVELS OF TH
  – INCREASE METABOLIC RATE
● RESECTION OF GOITER
  – IN CASES OF RESPIRATORY DIFFICULTY OR DYSPHAGIA

MANAGEMENT OF HYPOTHYROIDISM

1 T4 REPLACEMENT
  – LEVOTHYROXINE
    • SYNTHROID

T3 REPLACEMENT
  – CYTOMEL
  – THYROLAR
  – EUTHROID

2 NSG CONSIDERATIONS:
– WATCH FOR S/S OF HYPERTHYROIDISM
– ADMIN 1 HR BEFORE OR 2 HRS AFTER MEALS
– BLEEDING PRECAUTIONS
– MONITOR FOR DIG TOXICITY
– REQUIRE LIFELONG ADMINISTRATION
– AVOID TOO MUCH TURNIPS, CABBAGE, CARROTS SPINACH, PEACHES

54

MYXEDEMA COMA
● Result of long standing unrecognized or inadequately treated hypothyroidism
● Result of trauma, infection, CNS depressants, radioactive iodine use to treat hyperthyroidism
● Sometimes precipitated by thyroidectomy

55

Manifestations
● Severe hypometabolic state
● All s/s of hypothyroidism extremely exaggerated
  – Profound hypothermia
  – Hypoventilation
  – Hypotension, bradycardia
  – CNS depression, lethargy, coma
  – Resp acidosis, hyponatremia, hypoglycemia, elevated SGOT, CPK, LDH
  – Deep, coarse voice, paranoia, etc, etc, etc.

56

Treatment
● Maintain airway, ventilation
● Fluid and electrolyte replacement
● Thyroid hormone replacement, IV slowly, also glucose and corticosteroids
● Acid-base balance
● Pressor agents
● Rewarm slowly

57

NURSING CARE OF PATIENTS WITH DISORDERS OF THE PARATHYROID GLANDS

58

PARATHYROID

1. PTH
   – AT KIDNEYS
     • DECREASES CALCIUM EXCRETION
     • INCREASES PHOSPHATE EXCRETION

2. PTH
   – AT BONES
     • INCREASES BONE RESORPTION
     • RESULTS IN LOSS OF BONE MASS
     • INCREASES CALCIUM RELEASED FROM BONES

59

HYPERPARATHYROIDISM

1. PRIMARY
   • PTH PRODUCTION UNRESPONSIVE TO FEEDBACK MECHANISMS
   • SECONDARY
• UNABLE TO ACHIEVE NORMAL CA LEVELS DUE TO FAILURE OF KIDNEYS

★ TERTIARY
• HYPERPLASIA AND LOSS OF SENSITIVITY TO CALCIUM LEVELS

2

– ADENOMA

– CHRONIC HYPOCALCEMIA

– CHRONIC RENAL FAILURE

60 □ HYPERPARATHYROIDISM

★★ INCIDENCE AND ETIOLOGY
– WOMEN>MEN
– OLDER ADULTS
– 80% RESULT OF CHIEF CELL ADENOMA
– RARE DISORDER THAT IS USUALLY DISCOVERED WITH THE DISCOVERY OF RELATED DISEASE
• RENAL/URINARY
• OSTEOPOROSIS

61 □ Hyperparathyroidism

★★ CLINICAL MANIFESTATIONS
• Stones – renal calculi, polyuria, polydipsia, uremia
• Bones – osteitis, osteoporosis, bone cysts, osteomalacia, rickets
• Abdominal groans – constipation, indigestion, PUD
• Psychic moans – lethargy, fatigue, depression, psychoses
• Other – proximal muscle weakness, HTN, arrhythmias keratitis, conjunctivitis

62 □ HYPERPARATHYROIDISM

★★ DIAGNOSIS
– 6 MONTH HISTORY OF SYMPTOMS
– EXCLUSION OF OTHER CAUSES
– RADIOGRAPHIC STUDIES
– BONE SCAN
– LAB TESTS
  – CA > 10.5
  – PHOS: LOW LEVELS
  – CHLORIDE LEVELS ELEVATED
  – PARATHORMONE LEVELS ELEVATED

63 □ MANAGEMENT OF HYPERPARATHYROIDISM

1

– PC- decreased cardiac output

– RISK FOR ALTERED URINARY ELIMINATION

– Risk for injury

– PAIN
- Assess for signs of fluid overload
- Monitor I/O, daily weight
- Administer infusion therapy
- Administer furosemide
- Monitor electrolytes
- Strain urine for calculi

- Prevent pathologic fractures
- Administer analgesics

64 HYPERPARATHYROIDISM

- Treatment
  - Decrease calcium levels
    - Large amounts of saline infusion
    - Diuretic therapy
      - Avoid thiazide diuretics
    - Promote calcium retention
    - Calcitonin SQ or nasal spray
      - Decrease bone resorption (keeps calcium in bones)
    - Phosphate administration
    - Low calcium and Vit D diet

65 HYPERPARATHYROIDISM

- Surgery
  - Removal of parathyroid glands affected by hyperplasia or adenoma—one half of one gland remaining is sufficient to supply PTH, minimally invasive technic
  - Care same as for thyroidectomy
  - Autotransplantation of healthy parathyroid gland tissue into brachioradialis muscle of forearm—takes time to come to full effect, in meantime, client must supplement diet with calcium and vitamin D

66 HYPOPARATHYROIDISM

- Disorder of decreased circulating parathyroid hormone
- Results from decreased amounts or decreased sensitivity resulting in hypocalcemia and hyperphosphatemia

67 HYPOPARATHYROIDISM

- Incidence and etiology
  - S/P subtotal thyroidectomy
    - Damage/accidental removal
  - Can result from hypomagnesia
    - Etiology unknown
    - PTH corrected with magnesium correction
      - Suspect in alcoholism
      - Malnutrition/malabsorption

68 HYPOPARATHYROIDISM

- Clinical manifestations
– NEUROLOGIC
  • MENTAL STATUS/AFFECT
    – CONFUSION, ENCEPHALOPATHY, DEPRESSION, PSYCHOSIS
  • TETANY, CONVULSIONS
  • SPASM
    – LARYNGOSPASM
    – CARPOPEDAL SPASM
    – MUSCLE ACHE

69 HYPOPARATHYROIDISM
  ● OBJECTIVE ASSESSMENT
  – POSITIVE CHVOSTEK’S
    (THINK CHOPSTICKS)
    • FACIAL CONTRACTION AFTER TAPPING THE FACIAL NERVE

70 HYPOPARATHYROIDISM
  ● OBJECTIVE ASSESSMENT
  – POSITIVE TROUSSEAU’S
    (THINK TOURNIQUET)
    • CARPAL SPASM DISTAL TO TOURNIQUET APPLIED FOR THREE MINUTES

71 LAB DIAGNOSTICS
  ● DECREASED SERUM CALCIUM
  ● DECREASED PTH HORMONE
  ● POSSIBLE DECREASED MAGNESIUM
  ● INCREASED PHOSPHATE LEVELS

72 MANAGEMENT OF HYPOPARATHYROIDISM
  1 – RISK FOR INJURY
    – RISK FOR DECREASED CARDIAC OUTPUT
    – KNOWLEDGE DEFICIT

  2 – MONITOR FOR TETANY/SEIZURE ACTIVITY
    – ADMINISTER CALCIUM REPLACEMENT AS ORDERED
    – MONITOR SERUM CALCIUM LEVELS
    – MON ECG FOR DYSRHYTHMIA
    – ASSESS FOR DIG TOXICITY
DIETARY INSTRUCTION

HYPOPARATHYROIDISM

**TREATMENT**
- **NORMALIZE CALCIUM/PHOSPHATE**
  - CALCIUM GLUCONATE IV IF TETANY IS PRESENT
  - ORAL CALCIUM 1-7 GM /DAY
  - PHOSPHATE BINDERS IF SECONDARY TO RENAL FAILURE
  - CORRECT HYPMAGNESEMIA WITH MAG SULFATE IV OR IM
  - ROCALTROL, IF SECONDARY TO VITAMIN D DEFICIENCY

NURSING CARE OF THE PATIENTS WITH ADRENAL GLAND DISORDERS

ADRENAL GLANDS

2 PARTS:
- **CORTEX**
- **MEDULLA**

ADRENAL CORTEX

*ADRENAL CORTEX*
- **RELEASES GLUCOCORTICOIDs**
  - CORTISOL
  - CORTISONE
- **RELEASES MINERALOCORTICoids**
  - ALDOSTERONE
- **RELEASES ANDROGENS**

ADRENAL CORTEX

1. **INCREASE BLOOD GLUCOSE**
   - MOBILIZE FATTY ACIDS
   - MUSCLES USE FATTY ACIDS INSTEAD OF GLUCOSE
2. **DECREASE IMMUNE RESPONSE**
   - INHIBITS INFLAMMATORY RESPONSE
   - LESS EFFECTIVE IMMUNE RESPONSE

– DECREASE IN CIRCULATING VOLUME/BP...
– RESULTS IN RENIN RELEASE...
– LEADS TO FORMATION OF ANGIOTENSIN...
– CAUSES ALDOSTERONE RELEASE...
79  WHAT’S THE PRIMARY DIFFERENCE BETWEEN ADH AND ALDOSTERONE?
- ADH causes the distal renal tubules/collecting ducts to reabsorb water. No direct effect on electrolytes.
- Aldosterone retains sodium in loop of Henle and distal tubules/collecting ducts; water follows.

80  ADRENAL MEDULLA

1  ● ADRENALINE
   - EPINEPHRINE
     • INCREASE BLOOD GLUCOSE
     • STIMULATES ACTH RELEASE
     • RATE/FORCE CARDIAC CONTRACTION

2  ● NORADRENALINE
   - NOREPIHOREPHRINE
     • VASOCONSTRICTION THROUGHOUT THE BODY
     • INCREASES RATE/FORCE CARDIAC CONTRACTION

81  IF ADRENALIN STIMULATES ACTH RELEASE....

WHAT EFFECT WILL THAT HAVE ON THE ADRENAL CORTEX?
- Glucocorticoid Release

82  ADRENAL GLAND DISORDERS

● HYPERCORTICAL FUNCTION (From The Adrenal Cortex)
   - CUSHING DISEASE
   - CUSHING SYNDROME
   - HYPERALDOSTERONISM

● HYPOCORTICAL FUNCTION
   - ADDISON’S DISEASE

83  CUSHING’S

● INCIDENCE AND ETIOLOGY
   - WOMEN >MEN
   - AGE 30-50 YEARS
   - LONG TERM STEROID THERAPY
     • TRANSPLANT RECIPIENTS
     • IATROGENIC CUSHING’S SYNDROME

84  CUSHING’S

● PRIMARY (SYNDROME)
   - ADRENAL TUMOR INCREASES CORTISOL PRODUCTION

● SECONDARY
   - PITUITARY OR HYPOTHALAMUS DISORDER INCREASES ACTH PRODUCTION (DISEASE)
ECTOPIC CANCER CELLS PRODUCE ACTH (SYNDROME)

CUSHING’S

● CLINICAL MANIFESTATIONS
  – GENERAL SURVEY
    • BUFFALO HUMP/MOON FACIES
  – NEURO
    • EMOTIONAL LABILITY, FATIGUE
  – CV
    • HYPERTENSION
  – GI
    • PUD, TRUNCAL OBESITY

Figure 63-6
Appearance of a client with Cushing’s disease or syndrome

CUSHING’S

– MS
  • WASTING, WEAKNESS, OSTEOPOROSIS
– INTEGUMENT
  • THIN SKIN, POOR WOUND HEALING, INCREASED BODY HAIR
– RENAL/GU
  • CALCULI, GLYcosuria
    – POLYURIA/POLYDIPSIA
    – HYPOKALEMIA/HYPERNATREMIA
  – REPRODUCTIVE
    • OLIGOMENORRHEA, IMPOTENCE
    • DECREASED LIBIDO

CUSHING’S

● TESTING
● SAME LABS AS FOR ADDISONS, HOWEVER RESULTS WILL BE OPPOSITE
  – PLASMA CORTISOL LEVELS INCREASED
  – ACTH LEVELS
    • DECREASED IN PRIMARY
    • INCREASED IN SECONDARY
  – 24 HOUR URINE FOR CORTISOL
  – Serum Ca+, Na+, Gluc elevated
  – SERUM K+ CAN BE DECREASED

CUSHING’S

● TESTING
  – ACTH SUPPRESSION TEST
    • DEXAMETHASONE ADMINISTRATION
      – HIGH DOSE SUPPRESSES ACTH = PRIMARY CUSHING’S
      – ACTH NOT SUPPRESSED = SECONDARY CUSHING’S BECAUSE DEXAMETHASONE WORKS ON THE PITUITARY ONLY
MANAGEMENT OF CUSHING’S

- FLUID VOLUME EXCESS
  - RISK FOR INJURY R/O EXCESS CORTISOL
  - RISK FOR INFECTION

- ASSESS FOR S/S OF FLUID OVERLOAD; RESP/CV
  - MON I/O, DAILY WEIGHT, VS
  - FLUID RESTRICTION AS ORDERED
  - MONITOR ELECTROLYTES

- MAINTAIN SAFE ENVIRONMENT
- ENCOURAGE USE OF ASSISTIVE DEVICES/NON-SKID SLIPPERS

- MON S/S OF INFECTION
- MAINTAIN ASEPSIS
- MON CBC, TEMP, HR

CUSHING’S

- MANAGEMENT IS SPECIFIC FOR CAUSE AND INCLUDES:
  - RADIATION
     - IMPLANTS OR LOCAL IRRADIATION TO DESTROY THE PITUITARY GLAND. REQUIRES LIFELONG HORMONE REPLACEMENT
  - MEDICATION
     - INOPERABLE MALIGNANCIES
  - SURGERY
     - HYPOPHYSECTOMY
     - ADRENALECTOMY

Management-Pharmacology

- Medications that interfere with ACTH production or adrenal hormone synthesis
  - Mitotane(lysodren) suppresses adrenal cortex activity and decreases peripheral metabolism of corticosteroids
  - Amenoglutethimide(cytadren) and Trilostane(Modrastan) block synthesis of glucocorticoids and adrenal steroids
  - Cyproheptadine(periactin) interferes with ACTH production, therefore decreasing effect on adrenals

Surgery

- When Cushing’s syndrome is caused by adrenal tumor, adrenalectomy is done, one adrenal gland removed.
- If ectopic ACTH producing tumor involved, then bilateral adrenalectomy necessary
- Hypophysectomy—Surgical removal of pituitary, when it is cause of Cushing’s

Figure 63-3
Post-op Care
- Strict monitoring for symptoms of adrenal insufficiency (Addison’s)
- Hemorrhage, wound infection
- When transphenoidal hypophysectomy done, no nose blowing, coughing, sneezing, straining—risk for CSF leak
- Watch for sudden decrease in hypercortisol levels—depression, fatigue, etc. Leveling when maintenance dose reached, however pathologic changes remain

Post-op care cont’d
- Client and family instructed in need for additional replacement during times of stress
- Follow-up essential for adjustment of glucocorticoid regime
- If hypophysectomy done, all hormonal secretions dependent on the pituitary must be evaluated for brief or life-long replacement—glucocorticoids, thyroid hormone, gonadal steroids, ADH

ADRENAL CORTEX HYPOFUNCTION
- PRIMARY
  - DEFICIENCY ARISING FROM ADRENAL CORTEX
    - ADDISON’S DISEASE
    - BILATERAL ADRENALECTOMY
    - OTHER DISEASES
- SECONDARY
  - ACTH DEFICIENCY ARISING FROM PITUITARY
    - TUMORS, SURGERY

ADRENAL CORTEX HYPOFUNCTION

RESULTS IN DEFICIENCY OF:
- GLUCOCORTICOIDs
- MINERALOCORTICOIDs
- ANDROGENs

ADDISON’S DISEASE
- INCIDENCE AND ETIOLOGY
  - WOMEN UNDER AGE OF 60
  - ABRUPT WITHDRAWAL OF LONG TERM STEROID THERAPY
  - INCREASED RISK WITH TB, AIDS
  - SLOW ONSET

ADDISON’S DISEASE
- CLINICAL MANIFESTATIONS
  - METABOLIC
    - HYponatremIA, HYPERkalemIA, HYPOglycemIA FROM ALDOSTERONE DEFICIENCY
  - NEURO
    - LETHARGy, CONFUSION, TREMORS FROM ELECTROlyTE IMBALANCES
ADDISON’S DISEASE

CLINICAL MANIFESTATIONS
- MS
  • WEAKNESS, MUSCLE PAIN, WASTING
- INTEGUMENT
  • HYPER or HYPOPIGMENTATION
- GI
  • ANOREXIA, NAUSEA, DIARRHEA, SALT CRAVING
- REPRODUCTIVE
  • MENSTRUAL CHANGES.
  • S/S NOT SEEN AS MUCH IN MALES BECAUSE TESTES SUPPLY SOME ANDROGENS

DIAGNOSTIC/LAB TESTS
- CORTISOL LEVELS DECREASED
- SER Na+, Gluc DECREASED
- BUN, SER K+ INCREASED
- ACTH STIMULATION TEST
  • CORTISOL RISES WITH PITUITARY
  • NO ELEVATION IN ADRENAL
- CT HEAD

MANAGEMENT
HORMONE REPLACEMENT:
Must Replace Both Cortical Hormones
1. GLUCOCORTICOIDS:
   HYDROCORTISONE

2. MINERALCORTICOIDS
   FLORINEF

NSG CONSIDERATIONS
- TAKE WITH FOOD
- MONITOR FOR BLOOD IN STOOL
- LIFELONG THERAPY
- MONITOR FOR STRESSORS
- MONITOR F&E BALANCE
- REPORT S/S OF CUSHING'S TO MD

MANAGEMENT OF ADDISON’S
- FLUID VOLUME DEFICIT
  - PC: ADDISON CRISIS
– RISK FOR INJURY

– ASSESS FOR S/S OF FLUID DEFICIT; CV, INTEGUMENT, MUCOUS MEMBRANES, URINE OUTPUT
– MON I/O, DAILY WEIGHT, VS
– FLUID REPLACEMENT AS ORDERED (APPROX 3000 CC'S)
– MONITOR ELECTROLYTES
– ASSESS FOR S/S SHOCK

– MON S/S OF ORTHOSTATIC CHANGES
– PROVIDE ASSISTANCE AS NECESSARY

105  MANAGEMENT CONTINUED
● Life-long replacement therapy
● Adequate reserve supply, cannot run out of meds
● Medic alert bracelet
● Kit with IM preparation, Dexamethasone with instructions for administering

106  ADDISONIAN CRISIS
● Critical adrenal insufficiency, may occur with stressors or abrupt withdrawal of glucocorticoids
● Sudden, profound weakness, severe abdominal, leg and back pain, hyperpyrexia, hypothermia, CV collapse

107  MANAGEMENT OF CRISIS
● Reverse shock
● Restore blood circulation
● Replenish body with needed steroids
● Fluid balance is usually restored in 4-6 hours.
● Watch for overdosage of glucocorticoids, may get Cushing’s s/s

108  ADRENAL MEDULLARRY HYPERFUNCTION
● PHEOCHROMOCYTOMA: catecholamine (epinephrine and norepinephrine) secreting tumor of the chromaffin cells of the adrenal medulla
● Causes 0.1% of HTN
● Etiology unknown
● Pregnancy and stress precipitate & amplify symptoms

109  MANIFESTATIONS
● Similar to diabetes mellitus
● Elevated blood sugar
● Glucosuria
● Elevated blood pressure, principal sign
● Pounding headache
● S/s hyperthyroidism
All the manifestations of SNS activity
DIAGNOSIS

- CAREFUL H&P
  - INTERMITTENT HTN
  - CHEST PAIN, N/V
  - HEAT INTOLERANCE, WEIGHT LOSS, TREMORS
- URINARY CATECHOLAMINES ELEVATED
- PLASMA CATECHOLAMINES ELEVATED
- POSITIVE PHENTOLAMINE (REGITINE) TEST
- CT, X-RAY VISUALIZES TUMOR

Management

- Surgery, treatment of choice, remove tumor only. Alert: during anesthesia induction and/or tumor manipulation excessive press or discharge may skyrocket blood pressure
- Alert: may see precipitous fall in BP after surgery
- Control of BP AAT

NURSING CARE OF THE CLIENT WITH POSTERIOR PITUITARY DISORDERS

POSTERIOR PITUITARY

1. 2 P's = 2 hormones

PITUITARY DISORDERS

- ANTERIOR PITUITARY
  - GROWTH HORMONE
    - ACROMEGALY
    - GIGANTISM
- POSTERIOR PITUITARY
  - SIADH
  - DIABETES INSIPIDUS

SIADH

- CAUSES
  - POST-OP FLUID VOLUME SHIFTS
  - ADVERSE EFFECT OF MEDICATIONS
    - HYPOGLYCEMICS
    - BARBITUATES
    - GENERAL ANESTHETICS
    - DIURETICS
SIADH/PATHOPHYSIOLOGY

- TOO MUCH CIRCULATING ADH
- TOO MUCH WATER CONSERVATION IN KIDNEYS
- PLASMA BECOMES DILUTED
- HYPONATREMIA RESULTS FROM DILUTION
- ALDOSTERONE IS SUPPRESSED
- MORE SODIUM IS LOST
- FLUID SHIFTS TO INTERSTITIUM

Clinical manifestations

- CNS: when Na falls below 120mEq/L
  - Lethargy, headache, agitation, disorientation, LOC, coma.
- GI: anorexia, N/V,
- F&E: Na low, plasma osmolality low,
  - no peripheral edema, water retention, not Na.
- X-rays, CT scans - visualize etiologic factors

SIGNS AND SYMPTOMS OF SIADH

- weight gain
- diminished urination
- nausea
- hyponatremia
- lethargy
- a decreased level of consciousness, convulsions, and coma.

DIFFERENTIATING SIADH AND DI

<table>
<thead>
<tr>
<th>Test</th>
<th>DI</th>
<th>SIADH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Sodium</td>
<td>&gt; 145 mEq/L</td>
<td>&lt; 130</td>
</tr>
<tr>
<td>Serum osmolality</td>
<td>&gt; 295 mOsm/L</td>
<td>&lt; 275</td>
</tr>
<tr>
<td>Urine osmolality</td>
<td>&lt; 150 mOsm/L</td>
<td>&gt; 1200</td>
</tr>
<tr>
<td>Urine specific gravity</td>
<td>&lt; 1.005</td>
<td>&gt; 1.020</td>
</tr>
</tbody>
</table>

SIADH TREATMENT

- IV SODIUM REPLACEMENT. 3% NACL. NA SHOULD NOT RISE ABOVE 12 MEQ/L/DAY
- FLUID RESTRICTION
- DIURETICS-LASIX
- DECROMYCIN- USED IN DI
- FLUDROCORTISONE (FLORINEF)- NA RETENTION
DIABETES INSIPIDUS
INSUFFICIENT ADH

WHAT GOES IN COMES RIGHT OUT
INABILITY TO CONSERVE WATER AT KIDNEYS

DIABETES INSIPIDUS

● CAUSES
  – NEUROGENIC
    • LESIONS OF HYPOTHALAMUS OR PITUITARY GLAND
  – NEPHROGENIC
    • INADEQUATE RESPONSE TO ADH IN RENAL TUBULES
    • DISORDERS OR DRUGS
  – PSYCHOGENIC
    • FLUID INTAKE DILUTES ADH LEVEL

DIABETES INSIPIDUS

● RISK FACTORS
  ● Head injuries/infections: interfere with hypothalamic pituitary function
  ● Medications: alcohol, lithium carbonate, declomycin—interfere with synthesis or release of ADH or alter renal response.
  ● Renal disease
  ● Idiopathic

DIABETES INSIPIDUS

● NOT ENOUGH CIRCULATING ADH
● IMMEDIATE EXCRETION OF LARGE AMOUNTS OF DILUTE URINE
● THIRST IS TRIGGERED RESULTING IN POLYDIPSIA
● DEHYDRATION AND HYPERNATREMIA ENSUE

SIGNS AND SYMPTOMS OF DI

● Onset may be insidious or abrupt
● Occur at any age
● Polydipsia and polyuria.
  • Enormous quantities of fluid may be ingested, and large volumes (3 to 30 L/day) of very dilute urine (sp gr usually < 1.005 and osmolality < 200 mOsm/L) are excreted.
● Nocturia
● Dehydration/hypovolemia

DIFFERENTIATING SIADH AND DI

<table>
<thead>
<tr>
<th>Test</th>
<th>DI</th>
<th>SIADH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Sodium</td>
<td>&gt; 145 mEq/L</td>
<td>&lt; 130</td>
</tr>
</tbody>
</table>
THE WATER DEPRIVATION TEST
Should be performed only while the patient is under constant supervision.
- started in the morning
  - weighing the patient,
  - obtaining serum electrolyte/osmolality
  - measuring urinary osmolality.
- Voided urine is collected hourly
  - sp gr or osmolality (preferable) is measured.

Dehydration is continued until
- (1) orthostatic hypotension and postural tachycardia appear,
- (2) 5% or more of the initial body weight has been lost, or
- (3) the urinary concentration does not increase more than 0.001 sp gr or 30 mOsm/L in sequentially voided specimens.

At this point, serum electrolytes and osmolality are again determined, and 5 U of aqueous vasopressin is injected sc.
- Urine for sp gr or osmolality measurement is collected one final time 60 min postinjection, and the test is terminated.

TREATMENT OF DI
- Nonhormonal therapy:
  - (1) various diuretics
    - thiazides paradoxically reduce urine volume in partial and complete DI and NDI
  - (2) ADH-releasing drugs
    - Chlorpropamide, Carbamazepine, clofibrate
    - causes some release of ADH but also potentiates the action of ADH on the kidney

- Hormonal therapy:
  - Aqueous vasopressin sc or IM 5 to 10 U
    - to provide an antidiuretic response that usually lasts 6 h or less.
  - Synthetic vasopressin bid to qid nasal spray
  - DDAVP (desmopressin acetate, 1-deamino-8-D-arginine vasopressin) intranasally, sc, or IV.
    - has prolonged antidiuretic activity lasting for 12 to 24 h in most patients