Disorder of the

Anti-Diuretic Hormone

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Overview of Antidiuretic Hormone (ADH)

Structure	Function
Hypothalamus	Produces ADH
Posterior Pituitary	Stores and releases ADH

When ADH is released:

- Kidney **reabsorbs water**
- Urine becomes **concentrated**
- Blood volume **increases**

- Serum osmolality **decreases**

Diabetes Insipidus (DI)

- Diabetes Insipidus is a disorder characterized by **deficiency of ADH or inability of the kidneys to respond to ADH**, resulting in **excessive urination and extreme thirst**.

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Diabetes Insipidus (DI)

Type	Cause

Central DI	Decreased ADH production from hypothalamus/pituitary
Nephrogenic DI	Kidneys do not respond to ADH
Dipsogenic DI	Excess water intake
Gestational DI	Placental enzymes destroy ADH

Diabetes Insipidus (DI)

- **Pathophysiology**

- Hypothalamus/Pituitary Damage
  ↓
  ↓ Decreased ADH secretion
  ↓
  Kidneys unable to reabsorb water
  ↓

Large amounts of dilute urine

↓

Fluid loss

↓

Dehydration

↓

Hypernatremia

↓

Increased serum osmolality

Diabetes Insipidus (DI)

- **Causes**

- **Central DI**
- Head trauma
- Brain surgery
- Pituitary tumors
- Meningitis

- Encephalitis
- Idiopathic
- **Nephrogenic DI**
- Chronic kidney disease
- Lithium therapy
- Hypercalcemia
- Hypokalemia

Diabetes Insipidus (DI)

- Clinical Manifestations

Symptom	Explanation
Polyuria	Urine output up to **4–20 L/day**
Polydipsia	Excessive thirst

Dilute urine	Low specific gravity
Nocturia	Frequent urination at night
Dehydration	Water loss
Hypotension	Fluid volume deficit
Tachycardia	Compensatory response
Hypernatremia	Sodium concentration increases

Diabetes Insipidus

Diabetes Insipidus (DI)

- Diagnostic Tests

Test	Finding

Urine Specific Gravity	<1.005
Urine Osmolality	Low
Serum Osmolality	High
Serum Sodium	High
Water Deprivation Test	Confirms DI
MRI of Brain	Identify pituitary damage

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Diabetes Insipidus (DI)

- Water Deprivation Test

- Procedure:

1. Withhold fluids

2.Monitor weight and urine concentration

3.Administer **Desmopressin**

Diabetes Insipidus (DI)

- Medical Management of DI

- **Monitor Fluid Balance**
- Strict **intake and output**
- Monitor urine volume
- **Monitor Vital Signs**
- Hypotension
- Tachycardia
- **Monitor Electrolytes**
- Sodium levels

- Serum osmolality

- **Prevent Dehydration**
- Encourage fluid intake
- IV fluids if severe
- **Medication Administration**
- Desmopressin as ordered
- **Safety Measures**
- Assess mental status
- Monitor neurological changes

Diabetes Insipidus (DI)

- Nursing Diagnosis

Diagnosis	Reason
Fluid volume deficit	Excessive urination
Risk for electrolyte imbalance	Hypernatremia
Impaired comfort	Excessive thirst

Risk for decreased cardiac output	Severe dehydration

Diabetes Insipidus (DI)

- **Patient Education**

- Take medication regularly
- Maintain adequate hydration
- Monitor urine output
- Recognize symptoms of dehydration
- Follow-up laboratory monitoring

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- SIADH is a disorder characterized by **excessive secretion of ADH**, causing **water retention, dilution of blood sodium, and hyponatremia**.

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Excess ADH secretion
  ↓
  Kidneys reabsorb excessive water
  ↓
  Water retention
  ↓
  Dilution of sodium in blood

↓

Hyponatremia

↓

Decreased serum osmolality

↓

Cellular swelling

↓

Neurological symptoms

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Causes of SIADH

- **Neurological Causes**
- Brain tumors
- Head trauma
- Stroke
- Meningitis

- **Pulmonary Causes**
- Pneumonia
- Tuberculosis
- Lung cancer

- **Drug-Induced**
- Antidepressants
- Carbamazepine
- Chemotherapy drugs

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Clinical Manifestations

System	Symptoms
Neurologic	Headache, confusion

Severe hyponatremia	Seizures
Gastrointestinal	Nausea, vomiting
Muscular	Weakness
Fluid status	No edema

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Diagnostic Findings

Test	Finding
Serum Sodium	Low (<135 mEq/L)

Serum Osmolality	Low
Urine Osmolality	High
Urine Specific Gravity	High
Urine Sodium	Elevated

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Medical Management of SIADH

Treatment	Purpose

Fluid restriction	First-line treatment
Hypertonic saline (3%)	Severe hyponatremia
Loop diuretics	Promote water excretion
Vasopressin receptor antagonists (Vaptans)	Block ADH effect
Demeclocycline	Reduce ADH response

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Nursing Management of SIADH

- **Fluid Restriction**
- Usually **800–1000 mL/day**
- **Monitor Electrolytes**
- Sodium levels
- Serum osmolality
- **Neurological Assessment**
- Monitor mental status
- Assess for seizures
- **Monitor Intake and Output**
- **Daily Weight**
- Detect fluid retention
- **Safety Measures**
- Seizure precautions

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Nursing Diagnosis

Diagnosis	Reason
Excess fluid volume	Water retention
Risk for decreased cerebral perfusion	Hyponatremia
Risk for injury	Seizures
Confusion	Cerebral edema

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Patient Education

- Follow fluid restriction
- Monitor weight daily

- Recognize symptoms of hyponatremia
- Avoid medications that trigger SIADH

Comparison: Diabetes Insipidus vs SIADH

Feature	Diabetes Insipidus	SIADH
ADH Level	Low	High
Urine Output	High	Low
Urine Concentration	Dilute	Concentrated
Serum Sodium	High	Low

Serum Osmolality	High	Low
Urine Osmolality	Low	High
Hydration Status	Dehydration	Fluid overload
Treatment	ADH replacement	Fluid restriction

Memory Tip

- **DI = Dry Inside**
  - Dehydration
  - Dilute urine
  - Decreased ADH
- **SIADH = Soaked Inside**
  - Water retention
  - Hyponatremia
  - Excess ADH

Comparative Chart

- Possible Complications

Diabetes Insipidus	SIADH
Severe dehydration	Severe hyponatremia
Hypovolemic shock	Seizures
Hypernatremia	Cerebral edema
Kidney injury	Coma

Disorder of the

PANCREAS

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DIABETES MELLITUS

- **Diabetes Mellitus (DM)** is a **chronic metabolic disorder characterized by hyperglycemia (high blood glucose)**resulting from:

- Defects in **insulin secretion**
- **Insulin resistance**
- Or both
- Insulin is a hormone produced by the **beta cells of the pancreas** that regulates blood glucose levels.

Pathophysiology

Normal Glucose Metabolism

Role of Insulin

Insulin allows glucose to **enter the cells** where it is used for **energy production**.

Process

Glucose from food
↓
Absorbed into bloodstream
↓
Pancreas releases insulin
↓
Glucose enters body cells
↓
Energy production

When insulin is absent or ineffective → **glucose accumulates in the blood → hyperglycemia**

Pathophysiology

- Insulin deficiency or resistance
  ↓
  Glucose cannot enter cells
  ↓
  Hyperglycemia develops
  ↓
  Cells lack energy
  ↓
  Body breaks down fat and protein

↓

Ketone production

↓

Metabolic disturbances

Type	Description
Type 1 DM	Autoimmune destruction of pancreatic beta cells
Type 2 DM	Insulin resistance with relative insulin deficiency
Gestational Diabetes	Diabetes occurring during pregnancy
Secondary Diabetes	Due to disease or medications

Type 1 Diabetes Mellitus

- **Type 1 Diabetes Mellitus**

- **Characteristics**
- Usually occurs in **children or young adults**
- **Absolute insulin deficiency**
- Requires **lifelong insulin therapy**
- **Causes**
- Autoimmune destruction of beta cells
- Genetic predisposition
- Environmental triggers

Type 1 Diabetes Mellitus

- **Type 1 Diabetes Mellitus**

- **Characteristics**
- Usually occurs in **children or young adults**
- **Absolute insulin deficiency**
- Requires **lifelong insulin therapy**
- **Causes**
- Autoimmune destruction of beta cells
- Genetic predisposition

- Environmental triggers

Type 2 Diabetes Mellitus

- **Type 2 Diabetes Mellitus**

- **Characteristics**
- Most common type
- Usually occurs in **adults**
- Associated with **obesity and sedentary lifestyle**
- **Pathophysiology**
- Insulin resistance
  ↓
  Pancreas produces more insulin
  ↓
  Beta cell exhaustion
  ↓
  Relative insulin deficiency

Risk Factors for Type 2 Diabetes

- Obesity

- Family history
- Sedentary lifestyle
- Age over 45
- Hypertension
- Dyslipidemia
- History of gestational diabetes

Clinical Manifestations

Clinical Manifestations

Symptom	Explanation
Polyuria	Excessive urination

Polydipsia	Excessive thirst
Polyphagia	Increased hunger

Diagnostic Criteria

Test	Diagnostic Value
Fasting Blood Glucose	≥126 mg/dL
Random Blood Glucose	≥200 mg/dL
HbA1c	≥6.5%

Oral Glucose Tolerance Test	≥200 mg/dL

Glycosylated Hemoglobin (HbA1c)

HbA1c Level	Interpretation
<5.7%	Normal
5.7–6.4%	Prediabetes
≥6.5%	Diabetes

Acute Complications of Diabetes

Diabetic Ketoacidosis (DKA)

- Insulin deficiency

↓

Fat breakdown

↓

Ketone production

↓

Metabolic acidosis

Diabetic Ketoacidosis (DKA)

- Signs and Symptoms of DKA
  - Hyperglycemia
  - Ketonuria
  - Kussmaul Breathing
  - Fruity Breath Odor
  - Abdominal Pain
  - Nausea and Vomiting
  - Dehydration

Diabetic Ketoacidosis (DKA)

- Management of DKA

Treatment	Purpose
IV fluids	Correct dehydration

Insulin infusion	Reduce blood glucose
Electrolyte replacement	Correct potassium imbalance
Frequent monitoring	Prevent complications

Hyperosmolar Hyperglycemic State (HHS)

- Severe hyperglycemia

- Severe dehydration
- No significant ketosis

Hyperosmolar Hyperglycemic State (HHS)

- Symptoms of HHS
  - Extreme Hyperglycemia
  - Severe Dehydration
  - Confusion
  - Coma

DKA vs HHS Comparison Table

Feature	Diabetic Ketoacidosis (DKA)	Hyperosmolar Hyperglycemic State (HHS)
Common in	Type 1 Diabetes	Type 2 Diabetes
Cause	Severe insulin deficiency	Relative insulin deficiency
Onset	Rapid (hours–days)	Gradual (days–weeks)
Blood Glucose	250–600 mg/dL	Often >600 mg/dL
Ketones	Present	Minimal or absent
Acidosis	Present	Absent
Serum Osmolality	Mildly increased	Very high
Dehydration	Moderate	Severe
Respirations	Kussmaul breathing	Normal
Breath odor	Fruity odor	None
Mental status	Alert to drowsy	Confusion to coma

Mortality rate	Lower	Higher

Chronic Complications of Diabetes

Pathophysiology

- **Microvascular Damage**
- Damage to **small blood vessels**

Organ	Complication

Eye	Diabetic Retinopathy
Kidney	Diabetic Nephropathy
Nerves	Diabetic Neuropathy

Microvascular Complications

- **Diabetic Retinopathy**

- Damage to retinal blood vessels.
- Symptoms:
- Blurred vision
- Vision loss
- Blindness

Microvascular Complications

- **Diabetic Nephropathy**

- Damage to kidney filtration system.
- Signs:
- Proteinuria
- Edema
- Renal failure

Microvascular Complications

- **Diabetic Neuropathy**

- Damage to peripheral nerves.
- Symptoms:
- Numbness
- Tingling
- Burning sensation
- Loss of protective sensation

Pathophysiology

- **Macrovascular Damage**

- Damage to **large blood vessels**

↓

- Accelerated **atherosclerosis**

↓

- Complications:
- Coronary artery disease
- Stroke
- Peripheral artery disease

Diabetic Foot

- Due to:
  - Neuropathy
  - Poor circulation
  - Infection

- Signs:
- Foot ulcers
- Gangrene
- Amputation risk

Diabetic Foot Care

Recommendation
Inspect feet daily
Wash and dry feet carefully
Wear comfortable shoes

Avoid walking barefoot

Trim nails carefully

DIABETES MELLITUS

Medical Management of Diabetes

- Management focuses on:

1. Blood glucose control
2. Preventing complications

3.Lifestyle modification

Insulin Therapy

Type	Onset	Peak	Duration
Rapid-acting	15 min	1 hr	3–5 hrs
Short-acting	30 min	2–4 hrs	6–8 hrs
Intermediate	1–2 hrs	6–12 hrs	18 hrs
Long-acting	1–2 hrs	None	24 hrs

DIABETES MELLITUS - INSULIN THERAPY

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DIABETES MELLITUS - INSULIN THERAPY

DIABETES MELLITUS - INSULIN THERAPY COMPLICATIONS

Feature	**Dawn Phenomenon**	**Somogyi Phenomenon**
Basic Cause	Early morning hormone surge	Rebound from nighttime hypoglycemia
What happens at night?	Blood sugar stays normal	Blood sugar drops (hypoglycemia)
Early morning glucose	High	High

Mechanism	Cortisol, growth hormone ↑ → insulin resistance	Low glucose → body releases glucagon & epinephrine → rebound hyperglycemia
Time	3 AM – 8 AM	Usually after midnight hypoglycemia
3 AM blood glucose	Normal or slightly high	LOW
Key Problem	Not enough insulin in early morning	Too much insulin at night

Key Diagnostic Clue

- 👉 Check **3 AM blood glucose**

Result	Interpretation
Normal or High	Dawn Phenomenon

Low	Somogyi Phenomenon

Clinical Signs

- **Dawn Phenomenon**

- High fasting blood sugar
- No night symptoms

- **Somogyi Phenomenon**
- Night sweats
- Nightmares
- Morning headache
- Fatigue upon waking

Nursing Management

Condition	Management
Dawn Phenomenon	Increase evening insulin or adjust timing
Somogyi Phenomenon	Reduce nighttime insulin or give bedtime snack

DIABETES MELLITUS - INSULIN THERAPY COMPLICATIONS

- LOCAL ALLERGIC REACTIONS
- INSULIN LIPODYSTROPHY

DIABETES MELLITUS - ORAL HYPOGLYCEMIC MEDICATIONS (OHA)

Oral Antidiabetic Drugs

Drug Class	Mechanism

Biguanides (Metformin)	Decreases glucose production
Sulfonylureas	Stimulate insulin release
DPP-4 inhibitors	Increase incretin hormones
SGLT2 inhibitors	Increase glucose excretion
Thiazolidinediones	Improve insulin sensitivity

Nutritional Management

- Goals:
- Maintain normal blood glucose
- Achieve healthy body weight

Recommendation

Balanced diet

Controlled carbohydrate intake

High fiber

Limit sugar and refined carbohydrates

Reduce saturated fats

Exercise Management

- Benefits:

- Improves insulin sensitivity
- Helps weight control
- Reduces cardiovascular risk
- Recommended:
- **150 minutes of moderate exercise weekly**
- Examples:
- Walking
- Cycling
- Swimming

DIABETES MELLITUS - HYPOGLYCEMIA ASSESSMENT

Mild Hypoglycemia	Moderate Hypoglycemia	Sever Hypoglycemia
Hunger	Confusion	Difficulty Arousing
Nervousness	Double Vision	Disoriented Behavior
Palpitations	Drowsiness	Loss of Consciousness
Sweating	Emotional Changes	Seizures
Tachycardia	Headache	
Tremors	Impaired Coordination	
	Inability to Concentrate	
	Irrational or Combative Behavior	
	Lightheadedness	

Numbness of the Lips
and tongue

Slurred Speech

70mg/dL (4.0mmol/L	**40mg/dL (2.2mmol/L)**	**20mg/dL (1.1mmol/L)**

DIABETES MELLITUS - ACUTE COMPLICATION

Simple Carbohydrates to treat Hypoglycemia

- Commercially prepared glucose tablets
- Hard Candies
- 4 tsp of sugar
- 4 sugar cubes
- 1tbsp of honey or syrup
- ½ cup of fruit juice or regular soft drinks
- 8oz of low fat milk
- 6 saltine crackers
- 3 Graham Cracker

- DO NOT ATTEMPT TO ADMINISTER ORAL FOOD OR FLUIDS TO THE CLIENT EXPERIENCING A SEVERE

HYPOGLYCEMIC REACTION WHO IS SEMICONSCIOUS OR UNCONCSIOUS AND IS UNABLE TO SWALLOW

DIABETES MELLITUS - HYPOGLYCEMIA – INTERVENTIONS (15/15 Rule)

Nursing Management of Diabetes

- **Assessment**

- Monitor:
- Blood glucose levels
- Vital signs

- Signs of complications
- Neurological status

Nursing Interventions

Intervention	Rationale
Monitor blood glucose	Detect hyper/hypoglycemia
Administer insulin	Control blood glucose
Provide patient education	Promote self-care
Monitor feet daily	Prevent ulcers

Encourage healthy lifestyle	Prevent complications

Nursing Diagnosis

Diagnosis

Imbalanced nutrition

Risk for unstable blood glucose

Risk for infection

Impaired skin integrity

Deficient knowledge

Patient Education

- Patients should learn:
  - Blood glucose monitoring
  - Proper insulin administration
  - Healthy diet
  - Exercise
  - Foot care
  - Medication adherence

Easy Memory Tips

- **Type 1 DM = "NO insulin"**
  - Needs insulin **forever**
  - Risk for **DKA**
- **Type 2 DM = "NOT working insulin"**

- Insulin is present but **ineffective**
- Risk for **HHS**

Management Overview

Area	Key Points
Diet	Control carbohydrates
Exercise	At least 30 mins/day
Medication	Insulin or oral drugs
Monitoring	Check blood glucose regularly
Education	Lifelong self-care