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Interpreting Clinical Investigations in ICU





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CHAPTER

4 Endocrinological Abnormalities

Gurudas Sadanand Pundpal, Rahul Anil Pandit

CASE SCENARIO 1: ADRENAL INSUFFICIENCY

A 23-year-old female admitted with 4 months history of persistent vomiting, initially thought because of gastritis, 6–8 episodes a day, and weight loss. She was lethargic and drowsy. In the emergency room (ER), the patient's blood pressure was 80 systolic and pulse rate was 114 beats/min. The patient was looking dehydrated with cold extremities. Fluid resuscitation with normal saline was started and she was shifted to critical care unit. Her hemoglobin was 12 g/dL, white blood cell (WBC) count 12,500/L, sodium 128 mEq/L, potassium 4.9 mEq/L, and C-reactive protein (CRP) 12. Arterial blood gas (ABG) analysis shows pH 7.42, partial pressure of carbon dioxide (PCO_2) 38, bicarbonate (HCO_3) 23, partial pressure of oxygen (PaO_2) 88, and lactate 2.4 with normal liver and renal function tests. Serum random cortisol level was requested in view of persistent hypotension despite adequate fluid resuscitation, which came back 9 μg/dL.

1. **What is the diagnosis?**
   Adrenal insufficiency—primary (Addison disease) or secondary adrenal insufficiency (pituitary issues).

2. **What is the clinical presentation of patient with adrenal insufficiency?**
   Most striking presentation of all the patients presenting to intensive care unit is adrenal crisis with shock refractory to fluid resuscitation and vasopressors and rapid improvement only following use of steroids. Secondary adrenal insufficiency has a similar presentation as that of primary adrenal insufficiency, except hyperpigmentation and absence of mineralocorticoid deficiency features, such as hyperkalemia.[1]

3. **What are the laboratory investigations that help us to diagnose adrenal insufficiency?**
   The laboratory findings include hyponatremia, hyperkalemia, and hypoglycemia. Hyponatremia is due to cortisol and aldosterone deficiency. Aldosterone deficiency causes sodium wasting, and cortisol deficiency results in increased antidiuretic hormone. This causes increased water absorption. Hypovolemia also triggers antidiuretic hormone (ADH) secretion. Hyperkalemia is secondary to low aldosterone levels, which causes natriuresis and potassium retention. Hyperkalemia does not occur in secondary disease; this helps to distinguish from primary adrenal insufficiency. Hypoglycemia is multifactorial, including decreased oral intake and lack of glucocorticoids, which are essential for gluconeogenesis. Hypoglycemia is multifactorial, including decreased oral

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intake and lack of glucocorticoids, which are essential for gluconeogenesis. Hypercalcemia may be present, which reflects extracellular fluid loss.

- *Cortisol level:*
  - *>18 μg/dL:* Normal
  - *<3 μg/dL:* Adrenal insufficiency
  - *3–19 μg/dL:* Equivocal and further evaluation is suggested.
- *Adrenocorticotropic hormone (ACTH) level and corticotropin stimulation test:*
  - *Primary adrenal insufficiency:* Elevated ACTH
  - *Central adrenal insufficiency:* Abnormally normal or low ACTH

  With ACTH stimulation:
  - *Normal response:* Adequate response (peak cortisol level >18 μg/dL)
  - *Adrenal insufficiency:* Less or no response

4. How to manage a patient with adrenal insufficiency?

Addison crisis is a severe endocrine emergency; immediate recognition and treatment are required. Beware that if not recognized and treated, the adrenal crisis can be fatal.

- Fluid resuscitation to restore the intravascular volume with intravenous (IV) normal saline.
- Dextrose to correct hypoglycemia
- Correction of the hormone deficiency, both glucocorticoid and mineralocorticoid

The immediate hormonal treatment is the administration of hydrocortisone. Hypoglycemia should be treated promptly.

Maintenance Phase

Life-long treatment with hormonal replacement is required. Maintenance therapy aims to provide a replacement to maintain a physiologic glucocorticoid and mineralocorticoid level. The usual doses are as follows:

- *Glucocorticoid:* Hydrocortisone 5–25 mg/day (can be divided into two or three doses) and prednisone 3–5 mg/day[2,3]
- *Mineralocorticoid:* Fludrocortisone 0.05–0.2 mg daily[2]

Treatment Considerations

In patients with Addison's disease, glucocorticoid secretion does not increase during stress. Therefore, in the presence of fever, infection, or other illnesses, the hydrocortisone dose should be increased to compensate for a possible stress response.[4]

CASE SCENARIO 2: CONN'S SYNDROME

A 37-year-old female, a known case of systemic hypertension, came to the ER with complaints of palpitation, headache, and shortness of breath since a week. In the ER, the patient was conscious and alert. Her pulse rate was 110 beats/min and blood pressure 180/110 mm Hg. Her systemic examination of respiratory, cardiovascular, and nervous system was unremarkable. Electrocardiography (ECG) was suggestive of left ventricular strain pattern. Her serum sodium was 136 mEq/L, potassium 2.9 mEq/L, and the rest of renal and

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liver functions were within normal range. The patient had uncontrolled hypertension despite of starting on three different antihypertensive medications—amlodipine, metoprolol, and telmisartan. She was on oral potassium supplement in view of low potassium levels. In view of drug-resistant hypertension and persistent hypokalemia, plasma aldosterone and renin activity were measured. Plasma aldosterone concentration (PAC) was 90 ng/dL and plasma renin activity (PRA) was 0.20 ng/mL/h with PAC/PRA ratio was 450. The elevated PAC/PAR ratio is suggestive of hyperaldosteronism, a contrast-enhanced computed tomography (CT) scan and magnetic resonance imaging (MRI) scan of abdomen done, suggestive of unilateral adenoma. Tablet spironolactone was started subsequently, which resulted into control of blood pressure and correction of serum potassium level.

1. **What is the diagnosis?**
   Conn's syndrome,

2. **What is Conn's syndrome?**
   Primary hyperaldosteronism (Conn's syndrome) is an endocrine disorder characterized by excessive aldosterone production from adrenal gland, resulting into reduced renin activity and uncontrolled hypertension.[5]

3. **What is the pathophysiology of Conn's syndrome?**
   Hyperaldosteronism results in hypokalemia, plasma volume expansion, hypertension, metabolic alkalosis, and reduced renin levels.

4. **How to diagnose Conn's syndrome?**
   - Hypokalemia in a hypertensive patient is the most common clue for primary hyperaldosteronism. However, normal serum potassium may be present in up to 38% of patients, especially in patients with adrenal hyperplasia or familial aldosteronism.
   - *ABG:* Hypokalemia, hypernatremia, and metabolic alkalosis are due to the actions of aldosterone on the distal convoluted tubule.
   - Urinary potassium excretion is elevated (>30 mmol/day).
   - *Aldosterone:renin ratio:* A ratio of 40 or more (20–40 ng/dL/h) or >135 (68–135 pmol/mU) has a sensitivity of 73–93% and a specificity of 71–84%, indicating the need for further confirmatory studies with salt loading (failure to lower plasma aldosterone level <10 ng/dL), fludrocortisone suppression test, or captopril suppression test. If screening test is suggestive of primary hyperaldosteronism, then confirmatory diagnostic tests are performed, including 24-hour urinary aldosterone excretion test, ambulatory salt-loading test, or fludrocortisone suppression test.

5. **How to treat Conn's syndrome?**
   The primary goals of treatment of primary aldosteronism are normalization of blood pressure, potassium level, and aldosterone activity.[6,7]

   Surgical treatment: Unilateral adrenalectomy in patients with a unilateral adenoma (Conn's syndrome) cures hypertension.

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CASE SCENARIO 3: HYPEROSMOLAR HYPERGLYCEMIC STATE

A 56-year-old female, a known case of type II diabetes mellitus, presented with complaints of fever and burning sensation while passing urine. The patient had pain in abdomen, multiple episodes of vomiting, and breathing difficulty. The patient was a diagnosed case of type II diabetes mellitus and had history of poor glycemic control. On clinical examination, the patient was febrile. She was clinically dehydrated with a dry tongue and reduced skin turgor. There was tachycardia of 130 beats/min with blood pressure of 90/60 mm Hg and respiratory rate was 24 cycles/min. The chest was clear on auscultation.

1. **What is the diagnosis?**
   Hyperosmolar hyperglycemic state (HHS).

2. **What are the characteristic features of HHS?**
   - Hypovolemia—severely dehydrated and unwell
   - Marked hyperglycemia (>30 mmol/L) without significant ketonuria (<3 mmol/L) or acidosis (pH > 7.3 and bicarbonate > 15 mmol/L)
   - Osmolality is usually 320 mOsm/kg or more.

		Reference values
pH	7.35	7.35–7.45
PaO_2 (mm Hg)	103	83–108
$PaCO_2$ (mm Hg)	38	32–48
HCO_3^- (mmol/L)	23	22–28
Glucose (mg/dL)	786	65–95
Lactate (mmol/L)	2.2	0.4–0.8
Sodium (mmol/L)	150	136–145
Potassium (mmol/L)	4.5	3.4–4.5
Chloride (mmol/L)	106	98–107
Creatinine (mg/dL)	2.5	0.45–1.09

3. **What are the most common precipitants of HHS?**
   Infection, inadequate or missed insulin doses, pancreatitis, myocardial infarction, cerebrovascular accident, and drugs (e.g., corticosteroids, thiazides, and sympathomimetics) are the most common precipitants of HHS.

4. **Why are ketone bodies absent in HHS?**
   Relative insulin deficiency results in hyperglycemia and subsequent osmotic diuresis. However, insulin levels are adequate to prevent lipolysis and hepatic ketogenesis.[8]

5. **What are the signs of severe HHS?**
   - Osmolality >350 mOsmol/kg
   - Sodium above 160 mmol/L
   - Venous/arterial pH below 7.1

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- Hypokalemia (<3.5 mmol/L) or hyperkalemia (>6 mmol/L) on admission
- Glasgow Coma Scale (GCS) < 12
- Oxygen saturation below 92% on air (assuming normal baseline respiratory function)
- Systolic blood pressure below 90 mm Hg
- Pulse over 100 or below 60 beats/min
- Urine output <0.5 mL/kg/h
- Serum creatinine above 200 μmol/L
- Hypothermia
- Macrovascular event, such as myocardial infarction or stroke.

6. What are the principles of management of HHS?

The key principles of management of HHS are:

- Replace fluid and electrolyte losses
- Slowly restore osmolality and glucose to normal levels
- Prevent secondary complications.

7. How to do fluid management?

- Expect a 200 mL/kg total water deficit.
- *Commence fluid resuscitation:*
  - 15–20 mL/kg in the first hour
  - 4–14 mL/kg in the second hour (of 0.45% NaCl)
  - 4–14 mL/kg again in the third hour (use 0.9% NaCl if the sodium is low)
  - When glucose is under 15 mmol/L, start 5% dextrose 100–250 mL/h

Fluid resuscitation will bring down sugar. Insulin therapy may not be required and may even be dangerous.

In HHS, the serum glucose level will often fall with initial fluid resuscitation, and patients are normally highly insulin sensitive. Therefore, there is a risk of a precipitous initial fall in blood glucose and serum osmolality. Consequently, the presence of relative hypoinsulinemia should be identified by testing for significant ketonemia (blood 3-beta-hydroxybutyrate > 1 mmol/L). Only if this is present should insulin be started? For all other patients, insulin is only introduced when the rate of fall of glucose from fluid resuscitation has reached a plateau. Where insulin is started, the dose is reduced in HHS to 0.05 units/kg as a fixed rate insulin infusion. A maximum decrease in blood glucose of 5 mmol/L/h should be targeted.

8. What is the target serum osmolarity?

Serum sodium will inevitably rise due to the movement of water back into the intracellular space. A sodium rise of 2.4 mmol/L occurs for each 5.5 mmol/L fall in glucose. A safe drop is 3–8 mOsm/kg/h. Excessively rapid correction places the patient at risk of neurological complications, such as central pontine myelinolysis and cerebral edema.

9. How do you calculate serum osmolality?

Calculated osmolality = 2Na + glucose + urea

Excessively rapid correction places the patient at risk of neurological complications, such as central pontine myelinolysis and cerebral edema.

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10. What are the complications of HHS?

The complications are as:

- Thromboembolism
- Cerebral complications—confusion, decreased consciousness, seizures, cerebral edema, and central pontine myelinolysis.

CASE SCENARIO 4: DIABETIC KETOACIDOSIS

A 56-year-old female, a known case of type II diabetes mellitus, presented with complaints of fever and burning sensation while passing urine. The patient had pain in abdomen, multiple episodes of vomiting, and breathing difficulty. The patient was diagnosed with a case of type II diabetes mellitus and had a history of poor glycemic control. On clinical examination, the patient was febrile. She was clinically dehydrated with a dry tongue and reduced skin turgor. There was tachycardia of 130 beats/min with blood pressure of 90/60 mm Hg and respiratory rate was 24 breaths/min. The chest was clear on auscultation.

The ABG analysis is as follows:

		Reference values
pH	7.06	7.35–7.45
PaO_2 (mm Hg)	80	83–108
$PaCO_2$ (mm Hg)	22	32–48
HCO_3^- (mmol/L)	9	22–28
Glucose (mg/dL)	450	65–95
Lactate (mmol/L)	1.2	0.4–0.8
Sodium (mmol/L)	128	136–145
Potassium (mmol/L)	3.2	3.4–4.5
Chloride (mmol/L)	96	98–107
Creatinine (mg/dL)	1.12	0.45–1.09

1. What is the diagnosis?

Diabetic ketoacidosis (DKA).

2. What are the key elements of pathophysiology?

The key elements of pathophysiology of DKA are:[9]

- *Insulin deficiency and hyperglycemia:* Insulin deficiency leads to increased secretion of glucagon, which accelerates hepatic glucose production. As a result of increased production and decreased metabolism, blood glucose level increases.
- *Ketosis and metabolic acidosis:* As glucose cannot be utilized as a source of energy, increased lipolysis provides free fatty acids as a source of energy. Glucagon converts free fatty acids into ketone bodies in the liver. Predominant ketone bodies are beta-hydroxybutyrate and acetoacetic acid. Increased production of ketone bodies leads to high anion gap metabolic acidosis.

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- *Fluid and electrolyte abnormalities:* Hyperglycemia-induced osmotic diuresis leads to loss of water and electrolytes. Severe hyperglycemia results into fluid loss of about 6–8 liters in patients with DKA. Osmotic diuresis also causes marked depletion of electrolytes, such as sodium, potassium, chloride, magnesium, and phosphate. Although, there is depletion of fluid and electrolytes in DKA, serum electrolytes may be within normal range due to shift of water and electrolytes from intracellular to extracellular compartment as a result of increased serum osmolality due to hyperglycemic state and metabolic acidosis.[10]

3. What is the clinical presentation of DKA?

There is severe dehydration, dry skin and tongue, loss of skin turgor, sunken eyeballs, and reduced urine output. Patients have tachycardia, deep and labored breathing, also called Kussmaul's breathing with fruity odor of acetone in the breath. Patients with DKA are often lethargic. If untreated at this stage, the patient may progress to coma and hypotension, leading to circulatory failure and death.

Indications for critical care admission are as follows:

- Blood ketones > 6 mmol/L
- Bicarbonate level < 5 mmol/L
- Venous/arterial pH < 7.0
- Hypokalemia on admission (under 3.5 mmol/L)
- GCS < 12
- Oxygen saturation below 92% on air (assuming normal baseline respiratory function)
- Systolic blood pressure below 90 mm Hg
- Pulse over 100 or below 60 beats/min
- Anion gap above 16 [Anion gap = $(Na + K) - (Cl + HCO_3)$].

4. How do you diagnose DKA?

Laboratory investigation findings in DKA are as follows:

- *Blood glucose level:* Blood glucose concentration in DKA is typically in the range of 400–600 mg/dL. Some cases of DKA present with blood sugar level < 300 mg/dL, also called euglycemic DKA.
- *Renal function tests:* Blood urea nitrogen (BUN) and creatinine are typically elevated in patients with DKA.
- *Serum electrolytes:*
  - *Sodium:* Serum sodium level is variable ranging from 120 to 160 mEq/L in patients with DKA. Elevated sodium level reflects the degree of free water loss and dehydration. Shift of free water from intracellular to extracellular compartment because of osmotic gradient created by hyperglycemia result in abnormally low serum sodium concentration. There is a fall in measured sodium concentration by 1.6 mEq/L with each 100 mg/dL increase in blood sugar above 100 mg/dL. Corrected sodium levels should be calculated in all patients with DKA.
  - *Potassium:* At presentation, serum potassium levels are usually elevated in patients with DKA due to dehydration and shift of potassium from intracellular to extracellular compartment due to severe metabolic acidosis. Serum potassium

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levels should be monitored closely during treatment of DKA because after initiation of insulin therapy, there is intracellular shift of potassium, leading to precipitous fall in serum potassium concentration. Prompt correction of potassium level is necessary during treatment, otherwise cardiac arrhythmias may be precipitated due to hypokalemia.

- *Magnesium:* Similar to potassium, magnesium concentration is usually elevated in initial presentation, but it falls with initiation of treatment.
- *Phosphate:* During the course of correction, phosphate levels also should be monitored and corrected as phosphate is an important component of adenosine triphosphate (ATP) production.

- *Arterial blood gas and ketone bodies:* Arterial blood pH is always <7.3 in patients with DKA and usually <7.0 in cases of severe DKA. DKA is typically present with high anion gap metabolic acidosis. Rarely metabolic alkalosis may accompany anion gap acidosis due to severe vomiting in patients with DKA. Serum ketone bodies are always present in DKA. Commonly done test to measure ketone bodies is nitroprusside test, which measures only acetoacetate and acetone and does not measure beta-hydroxybutyrate.
- *Complete blood count:* Hemoglobin and hematocrit levels are usually elevated in initial stages of DKA due to plasma volume contraction. WBC count is always elevated even in the absence of infection.
- *Urine analysis:* Urine ketone should be checked as part of initial diagnosis algorithm. Urinary ketones should not be used to guide treatment as they will remain positive even after the DKA has resolved.

5. How do you manage a patient with DKA?

The principles of management of DKA are as follows:

- Fluid resuscitation
- Replacement of electrolytes
- Insulin therapy
- Treatment of precipitation factors

Patients with DKA should be admitted to the intensive care unit. Treatment of DKA is directed toward replacement of fluid, electrolytes, and insulin in that order.

- *Fluid resuscitation:* Newer studies advocate the use of the balanced salt solution to maintain the electrolyte hemostasis. Further, fluid replacement rate should be determined by patient's clinical response and urine output. About 75% of fluid deficit should be corrected in the first 24 hours. After initial fluid resuscitation, the replacement fluid may be changed to 1–2 liters of 0.45% saline in patients with hypernatremia. Once plasma glucose level falls to 180–200 mg/dL, 0.9% saline should be replaced by 5% or 10% dextrose for fluid replacement to prevent hypoglycemia. IV insulin therapy needs to be continued as ketogenesis continues despite a fall in plasma glucose level.
- *Replacement of electrolytes:*
  - *Potassium:* Although the initial serum potassium level is normal, there is significant total body potassium deficit in DKA. Once fluid and insulin replacement have started, serum potassium levels fall and frequently become subnormal during

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treatment of DKA. Prompt replacement of potassium with IV potassium chloride is necessary. Severe hypokalemia can precipitate life-threatening cardiac arrhythmia. Serum potassium levels and ECG should be monitored periodically during treatment. Rate of potassium replacement is adjusted according to serum potassium levels.

- *Phosphate:* Serum phosphate concentration falls during treatment of DKA. Although, serum phosphate falls below the normal level, phosphate replacement is rarely needed in patients with severe hypophosphatemia (<1.0 mg/dL). Potassium phosphate is used for phosphate replacement whenever necessary. Severe hypophosphatemia can lead to muscle weakness, respiratory failure due to involvement of respiratory muscles, rhabdomyolysis, and depressed cardiac contractility and neurological disturbances.
- *Bicarbonate:* Even if patients with DKA present with metabolic acidosis, bicarbonate therapy is not needed for correction of acidosis. In fact, bicarbonate treatment may have deleterious effects, such as worsening hypokalemia, late metabolic alkalosis, and cerebral edema, especially in children.

- *Insulin therapy:* Insulin should be provided via a weight-based fixed-rate intravenous insulin infusion (FRIII). Insulin at 0.1 units/kg should be started as soon as fluid resuscitation is commenced.

  Blood glucose levels should be monitored every 1–2 hours initially. Insulin infusion is titrated to achieve fall in blood glucose level at a rate of 75–100 mg/dL. Target blood sugar level in the first 24 hours is between 150 and 200 mg/dL. Both hypoglycemia and development of hyperglycemia should be avoided. Even if blood sugar falls below 200 mg/dL, insulin infusion should be continued as ketogenesis may continue despite normalization of blood sugar level. Hypoglycemia is prevented by replacing 0.9% saline with 5% or 10% dextrose solution for fluid replacement.

  Arterial blood gas analysis for resolution of acidosis and normalization of anion gap guides about clearance of ketosis. Once the anion gap is normalized, the patient is conscious, alert, clinically stable, and is able to eat and drink. Insulin treatment with subcutaneous insulin is started. Insulin infusion should be continued till onset of action of subcutaneous insulin.
- *Treatment of precipitation factors:* Identification of precipitating factor and appropriate treatment of same is very important. Active search for presence of infection should be done in each case of DKA.

6. What are the complications associated with DKA?

Complications associated with DKA are:

- *Hypoglycemia:* To prevent hypoglycemia, it is advisable to keep blood glucose level close to 200 mg/dL in the first 24 hours. If blood glucose level falls below 200 mg/dL, replacement fluid is changed to 5% dextrose instead of normal saline.
- Cerebral edema
- Thrombotic complications
- Renal failure.

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CASE SCENARIO 5: HYPOGLYCEMIA

A 64-year-old male patient, a known case of type II diabetes mellitus, on oral hypoglycemic treatment, was brought by relatives with history of sudden-onset altered sensorium. Relatives denied history of headache, vomiting, head injury, or witnessed seizure. In the ER, the patient was drowsy, difficult to arouse. There was no neck stiffness and no focal neuro deficit. He was not obeying commands and was afebrile. His pulse rate was 112 beats/min with sinus tachycardia, blood pressure 90/50 mm Hg, and respiratory rate 22 breaths/min. His laboratory investigations were as follows: Hemoglobin 10 g/dL and WBC 12 × 10^9/L. Liver function test was essentially normal, creatinine was 2.3 mg/dL, and BUN 34 mg/dL. Blood sugar was 36 mg/dL. Immediately 50 mL of 50% dextrose was infused to patient. The patient became conscious and alert post dextrose infusion. On detailed history taking, history of fever and burning micturition was present since last 4 days. Relatives also gave history of reduced oral intake and decreases urine output since last 2 days.

		Reference values
pH	7.39	7.35–7.45
PaO_2 (mm Hg)	80	83–108
$PaCO_2$ (mm Hg)	22	32–48
HCO_3^- (mmol/L)	24	22–28
Glucose (mg/dL)	36	65–95
Lactate (mmol/L)	1.2	0.4–0.8
Sodium (mmol/L)	138	136–145
Potassium (mmol/L)	3.9	3.4–4.5
Chloride (mmol/L)	104	98–107
Creatinine (mg/dL)	2.3	0.45–1.09

1. **What is hypoglycemia?**
   Hypoglycemia is not defined by any specific blood sugar level. The Whipple's triad of hypoglycemia consists of:
   - Documentation of low blood glucose level
   - Presence of symptoms consistent with hypoglycemia
   - Resolution of hypoglycemic symptoms post glucose administration.

2. **What are the causes for development of hypoglycemia?**
   Hormonal imbalance principally involving insulin is most common cause of hypoglycemia.[11]
   - Insulin overdose (iatrogenic)
   - Insulin secreting tumors—insulinoma and insulin-secreting nonislet tumor
   - Hypoglycemia with use of oral hypoglycemic agents—some of the sulfonylureas have half-life >24 hours. Prolongation of duration of action due to hepatic or renal dysfunction can result in prolonged hypoglycemia.

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3. **Which drugs other than insulin can cause hypoglycemia?**
   - Ethanol causes hypoglycemia by suppressing hepatic gluconeogenesis.
   - Beta-blocker agents—noncardioselective agents such as propranolol and nadolol may precipitate hypoglycemia.
   - Antiarrhythmic agents—quinidine is known to cause hypoglycemia in susceptible individuals.
   - Other agents rarely causing hypoglycemia include salicylates, angiotensin-converting enzyme (ACE) inhibitors, and antibiotics such as pentamidine and gatifloxacin.

4. **How to manage a patient with hypoglycemia?**
   All comatose patients' blood sugar should be checked with finger-prick test and IV glucose should be administered promptly, if hypoglycemia is present. 50 mL of 50% dextrose solution is administered over the period of 2–5 minutes as an initial bolus in comatose patients. Rapid improvement in sensorium following glucose administration confirms hypoglycemia as a cause for altered sensorium. Oral glucose is preferred in alert and cooperative patients. After initial bolus dose of glucose, an infusion of 5% or 10% dextrose should be started to maintain blood glucose level around 100 mg/dL. If hypoglycemia is due to overdose of long-acting insulin, initial bolus dose of 50% dextrose should be followed by continuous infusion of 5–10% dextrose.[12,13]

5. **What blood investigations will you do in a patient with hypoglycemia?**
   In all patients with symptomatic hypoglycemia, blood glucose measurement should be done in clinical laboratory. Other laboratory tests to determine the cause of hypoglycemia include renal and liver function tests. In all the patients with hypoglycemic coma serum and plasma, samples are collected for measurement of C-peptide and proinsulin levels, if clinically indicated.

6. **How do you manage sulfonylurea-induced hypoglycemia?**
   If sulfonylureas are a cause for development of hypoglycemia, patient should be hospitalized as these agents have long duration of action. Activated charcoal may prevent absorption of the drug from the stomach, while alkalinization of urine may enhance excretion of the drug. Sulfonylurea-induced hypoglycemia typically takes 2–3 days to resolve.

7. **How is persistent hypoglycemia treated?**
   - Continuous IV dextrose supplementation
   - Glucocorticoids
   - Octreotide
   - Diazoxide.

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Interpreting Clinical Investigations in ICU

The duty to administer clinical care to a diverse population that is connected solely by the severity of their illness is what makes medicine special. Nowadays, we have access to hundreds of different diagnostic techniques. Choosing the right diagnostic test for a particular clinical circumstance is one of the challenges that professionals in the medical field regularly confront. It is best to utilize a test that is accessible, reliable, repeatable, and appropriate for the circumstances at hand whenever possible. This little book aims to provide a simplified approach for choosing and interpreting investigations. Since providing a strong clinical viewpoint was our main objective, all of the contributors are practising physicians. We would greatly appreciate any comments from readers, as there may have been tests that we missed, there may be typos in the text, and there are a number of other changes that we may, and will, make in subsequent editions.

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