Gastroenterology

1-Acute Gastroenteritis (GE)

It is an infection of the small intestine, which present with a combination of **diarrhea** and vomiting [1] , but sometimes present without vomiting [2].

Etiology

1-**Rota virus** is the most common pathogen in children **under 2 years** [3], other causes include:

A-Acute **bacterial infections** (shigellae, Salmonellae, E coli and Vibirio cholera which secrete enterotoxins) [3].

B-**Parasites** like E. histolytica, and Giardia lambilia [2].

Clinical Features

1-**Rotaviruse cause watery diarrhea** . **Respiratory illness** occur in about half of patients followed by vomiting and diarrhea [1, 2].

2-Acute **bacterial** infection cause invasion of GIT, so there is **fever**, and small volume **bloody stool** [3].

Complication of Gastroenteritis

Dehydration, **metabolic disturbances** and even **death** [4].

Treatment

1-Uncomplicated viral GE requires no specific treatment except attention to fluid and electrolyte replacement [3] Most of these episodes are self-limited [4].

2-There is **no role for antiemetic or antidiarrheal** in GE [1].

3-**Antibiotics are rarely indicated** except for specific infections such as invasive salmonellosis, cholera , amebiasis or giardiasis [1, 3].

4-The **key management of GE is rehydration** with correction of fluid and electrolyte imbalance [1].

A-Unless the child has persistent vomiting,oral fluid is the best means for rehydration, smaller more frequent sips may be better tolerated and should be encouraged [1].

B-**Mild Dehydration**: ORS are used [1].

C-**Moderate dehydration:** Oral rehydration is still indicated if tolerated.

D-**I.V fluid** should be reserved for those **with vomiting or severe**

dehydration [1].

5-**Zinc supplementation** (10–20 mg for 10–14 days) has been recommended by the WHO for the treatment and prevention of diarrheal disease in children in developing countries [4].

6-**Continuation of oral feeding**, despite diarrheal episodes, decreases the duration of illness; and improves nutritional status [4].

2-Viral Hepatitis

Etiology

1-There are six primary hepatitis viruses: Hepatitis A virus (HAV), Hepatitis B virus (HBV), Hepatitis C virus (HCV), Hepatitis D virus (HDV), Hepatitis E virus (HEV) and Hepatitis G virus (HGV) [1].

2-They differ in their transmission, severity, likelihood of persistence, and subsequent risk of hepatocellular carcinoma [1].

	HAV	HBV	HCV	HDV	HEV	HGV
Transmission	Fecal-oral	Transfusion, sexual, perinatal	Parenteral, transfusion, perinatal	Similar to HBV	Fecal-oral	Parenteral transfusion

Note - The most important risk factor for acquisition of **HBV in children is perinatal exposure to infected mother**. In most cases, transmission **occurred at the time of delivery**; virus contained in amniotic fluid or in maternal blood may be the source. However less commonly intrauterine infection occurred [2].

3-**HBV and HCV cause chronic infection**, which may lead to **cirrhosis** and is a significant risk factor for **hepatocellular carcinoma** [1].

Clinical Manifestations

1-**Asymptomatic or mild**, nonspecific illness without icterus (jaundice) **is common** with HAV, HBV, and HCV, **especially in young children**[1].

2-The **preicteric phase**, which lasts approximately 1 week, is characterized by headache, anorexia, malaise, abdominal discomfort, nausea, and vomiting and usually precedes the onset of clinically detectable disease [1].

3-**Jaundice** and tender **hepatomegaly** are the most common physical

findings and are characteristic of the **icteric phase**. Hepatic enzymes may increase 15- to 20-fold [1].

4-Resolution of the hyperbilirubinemia and normalization of the transaminases may take 6 to 8 weeks [1].

Complications

1-Most cases of acute viral hepatitis resolve without specific therapy, with less than 0.1% of cases progressing to **fulminant hepatic necrosis** which is associated with a high mortality rate [1].

2-**HAV and HEV cause acute infection only**. HBV, HCV, and HDV may persist as **chronic infection** with chronic inflammation, fibrosis, and **cirrhosis and the associated risk of hepatocellular carcinoma** [1].

Diagnosis

The diagnosis of viral hepatitis is confirmed by **serologic testing** [1].

Treatment

1-The treatment of acute hepatitis [1] (**except HCV** [3]) is largely supportive and involves rest, hydration, and adequate nutrition. Hospitalization is indicated for severe cases [1].

2-**Chronic HBV** infection may be treated with **interferon alfa-2b or lamivudine**, and HCV may be treated with interferon alfa usually in combination with Ribavirin [1].

Respiratory Disorders

1-Cystic Fibrosis

Background

1-Cystic fibrosis (CF) is an autosomal recessive multisystem disorder caused by mutations in the *cystic fibrosis transmembrane regulator* **(CFTR)** gene [1].

2- CFTR is important for the proper movement of salt and water across epithelial cell membranes especially in the airways, liver, and pancreas [2]. The term *cystic fibrosis* arises from the fibrotic scar tissue that replaces the destroyed pancreas [3].

Pathophysiology

A-Pulmonary System

1-In CF, there are reduced chloride secretion with excessive sodium resorption which lead to dehydration of the airway lining [4] leading to airway obstruction. This, in turn, leads to colonization with bacteria especially *Staphylococcus aureus* and *Pseudomonas aeruginosa* [2].

2-Chronic lung disease is a hallmark of CF, leading to death in 90% of patients [5]. CF patients will usually experience **chronic respiratory**

infections [6].

B-Gastrointestinal Involvement

1- Approximately 10% of patients with CF are born with intestinal obstruction caused by inspissated meconium (**meconium ileus**). In older patients, intestinal obstruction may result from thick inspissated mucus in the intestinal lumen [2].

C-Hepatic Involvement

1-In patients with CF, there is reduction in water and sodium movement into the bile. **The resulting decrease in the volume and flow of bile leads to stasis and obstruction of the biliary tree**. With chronic obstruction, this leads to **biliary cirrhosis** [3].

D-Pancreatic Involvement

1-The obstruction of the pancreatic ducts result in the inability to excrete pancreatic enzymes into the intestine. This leads to malabsorption of proteins, sugars (to a lesser extent), and **especially fat**. Fat malabsorption manifests clinically as **steatorrhea** (large foul-smelling stools), **deficiencies of fat-soluble vitamins** (A, D, E, and K), and **failure to thrive** [2].

E-Sweat Gland

In the sweat duct, CFTR reabsorbs chloride from sweat. Dysfunctional CFTR results in a **nearly fivefold elevation in sweat chloride concentrations**. This is the principal laboratory criterion for diagnosis of CF (**sweat chloride test**) [7].

Diagnosis

CF is most commonly diagnosed on the basis of typical **signs and symptoms** and an abnormal sweat chloride concentration (>60 mEq/L) (**sweat chloride test**) [3, 7].

Treatment

The treatment of CF is multifactorial, but it is primarily directed toward the gastrointestinal and pulmonary complications [2].

A-Gastrointestinal System

1-**Pancreatic enzyme replacement** (lipase, protease, and amylase) is the mainstay of gastrointestinal therapy [5].

2-**Fat-soluble vitamins (A, D, E, and K) supplementation** is usually required in pancreatic insufficiency [5].

3-The use **of ursodeoxycholic acid (UDCA)** may improves bile flow, prevent obstruction and slow progression of liver disease [5, 7].

B-Treatment of Cystic Fibrosis Airway Disease

Treatment of CF airway disease involves the use of medications and techniques to mobilize pulmonary secretions, and antibiotics to manage infection [3].

1-Mucociliary Clearance

A-Physical Therapy: Airway clearance can be performed using various techniques. These techniques are recommended **on a daily basis to help mobilize secretions** [8].

B-Mucolytic Therapy: Sputum viscosity is increased by the large quantities of extracellular DNA that result from chronic airway inflammation and degradation of neutrophils [9]. **Inhaled recombinant human deoxyribonuclease** (rhDNase, dornase alpha) cleaves extracellular DNA in sputum [9].

C-Airway Hydration Therapies : Inhalation of hypertonic saline rehydrates the airways through osmotic flow of water [3].

D-Bronchodilators: β-Agonists keep airways open and facilitate airway clearance [8].

2-Antibiotics

1-Antibiotics are used to treat lung infection. *Typical regimens for severe infections include an antipseudomonal β-lactam plus an aminoglycoside for added synergy and delay of resistance development* [5].

2-**Fluoroquinolone** use is common among CF patients infected with ***P. aeruginosa*,** even in children [5].

3-**Chronic maintenance antibiotic therapy may be used in patients with *Pseudomonas* colonization** in an attempt to prevent bacterial overgrowth [5]. **Inhaled tobramycin** has been studied the most extensively [5].

Pharmacokinetic Considerations

CF patients have **larger volumes of distribution of many antibiotics** and also have an enhanced total body clearance [5]. As a result of these pharmacokinetic changes, **higher doses of antibiotics** (e.g. aminoglycosides, and β-lactam antibiotics) are needed [5].

Lung transplantation

Lung transplantation is currently the only definitive treatment for advanced cystic fibrosis [9].

Prognosis

The longevity of patients with cystic fibrosis is increasing, and the median survival age is over 35 years. Death occurs mostly from pulmonary complications [9].

Endocrinology

1-Diabetic ketoacidosis (DKA)

1-**Definition**: Arterial pH <7.30, bicarbonate <15 meq/L, glucose >250 mg/dL, and Urinary ketones [1, 2].

2-DKA is a major **medical emergency** and remains a serious cause of morbidity, principally in people **with type 1 diabetes** [3] (**More common in type 1 DM** but can occur in type 2 DM) [4].

3-A significant number of **newly diagnosed diabetic children** present with DKA. In children with known diabetes, DKA occurs in patient who omit insulin doses or who do not successfully manage an intercurrent illness [3, 5].

Risk Factors

1-**Omission of insulin** is the most common precipitant of DKA [6].

2-**Infections**, acute medical illnesses, and stress of recent surgical procedures can contribute to the development of DKA [6].

Pathophysiology

1-The **hyperglycaemia** causes a profound **osmotic diuresis** leading to **dehydration**, hyperosmolarity, and **electrolyte loss**, particularly of sodium and potassium [3, 6].

2-Owing to increased lipolysis and decreased lipogenesis, free fatty acids are converted to ketone bodies and lead to **metabolic acidosis** [6, 7].

3- **Electrolyte abnormalities** occur through a loss of electrolytes in the urine [7]. In addition, The resulting metabolic acidosis causes efflux of potassium from cells, results in intracellular potassium depletion [3, 6].

Clinical Presentation

1-Patients with DKA present initially with **polyuria**, **polydipsia**, **nausea**, and **vomiting**. **Abdominal pain** occurs frequently [7].

2-Respiratory compensation for acidosis results in **tachypnea with deep (Küssmaul) respirations**. The **fruity odor of acetone** frequently can be

detected on the patient's breath [7].
3-An altered mental status can occur, ranging from disorientation to coma [7].

Management

1-DKA is a **medical emergency** which should be treated in hospital [3]. The principal components of treatment are :

- The administration of **short-acting** (soluble) insulin [3].
- **Fluid** replacement [3].
- **Potassium** replacement [3].
- The administration of **antibiotics** if infection is present [3].

Note: for more detailed about for the management of ketoacidosis , there is **a Diabetic Ketoacidosis Treatment Protocol** [5].

Complications

The most concerning complication of DKA is **cerebral oedema** (Treatment by Mannitol 1 g/kg IV)