CURRENT APPROACHES TO DISEASES IN PEDIATRIC SURGERY

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PREFACE

Health has always been at the center of science and technology since the earliest days of humankind, and it has also guided science and technology. In this respect, health has been the most studied scientific field by scientists from the past to the present. Specialization plays an important role in the development of science.

Pediatric surgery is a subspecialty of surgery that includes the surgery of fetuses, infants, children, adolescents, and young adults. Subspecialties within pediatric surgery include: neonatal surgery, minimally invasive surgery (Laparoscopy and thoracoscopy), and fetal surgery.

In all medical fields, including Pediatric Surgery, existing knowledge is both being updated and increasing. To be a knowledgeable and high-quality physician and to perform their professional duties in the best possible way, every physician must closely follow these advancements. As in all areas of health, in this field as well, it is extremely important for all scientists and staff working in Pediatric Surgery, who are dedicated to this profession, to have both fundamental and up-to-date knowledge through a multidisciplinary approach for their professional development.

As in all areas of health, in this field as well, it is extremely important for all scientists and employees working in Pediatric Surgery, who are dedicated to this profession, to have both fundamental and up-to-date information through a multidisciplinary approach in their development. For this purpose, with the book " Current approaches to diseases in pediatric surgery" we aimed to compile scientific studies from different disciplines of health sciences in our country and to convey fundamental and up-to-date information to our valued readers. We sincerely thank the academics who contributed and supported the book " Current approaches to diseases in pediatric surgery'' with their scientific studies. Editor.

İÇİNDEKİLER

BÖLÜM 1

UPPER GASTROINTESTINAL BLEEDING IN CHILDREN

AZİZ SERHAT BAYKARA[1]

Introduction

Gastrointestinal (GI) bleeding refers to blood loss originating from any segment of the digestive tract. Anatomically, GI bleeding is classified according to the ligament of Treitz: bleeding arising proximal to the ligament of Treitz is defined as upper gastrointestinal bleeding (UGIB), whereas bleeding originating distal to the ligament is classified as lower gastrointestinal bleeding (Polat et al., 2020).

UGIB in children differs from that in adults due to the often subtle clinical presentation, difficulties in accurately assessing the extent of blood loss, and the technical challenges associated with endoscopic interventions. Furthermore, the limited physiological reserve of pediatric patients increases the risk of rapid clinical deterioration. Therefore, early diagnosis, recognition of age-specific

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etiologies, and the timely implementation of appropriate therapeutic strategies are of paramount importance.

In this review, the etiology, clinical manifestations, diagnostic approaches, and treatment modalities of pediatric UGIB are discussed in light of the current literature.

Epidemiology

Although the true incidence of UGIB in the pediatric population is not precisely known, hospital-based studies have reported that it accounts for only a small proportion of all GI bleeding cases (Nasher et al., 2017). Despite being less common than in adults, pediatric UGIB remains an important cause of morbidity. Its incidence varies according to age group and underlying risk factors.

Although pediatric UGIB is a relatively uncommon reason for presentation to the emergency department, severe cases are associated with a high rate of hospitalization and intensive care unit admission due to the potential for rapid clinical deterioration (Tambucci et al., 2022). While mortality rates are generally lower than those observed in adults, they increase significantly in the presence of variceal bleeding and concomitant severe systemic diseases.

Etiology

The etiologies of UGIB in children exhibit considerable heterogeneity due to age-related physiological differences, underlying systemic diseases, and environmental factors (Table 1). The etiologic spectrum differs significantly from that observed in adults and encompasses a wide range of conditions from the neonatal period through adolescence. Therefore, etiological evaluation should be performed with careful consideration of age-specific clinical characteristics.

In the neonatal period, UGIB is most commonly associated with vitamin K deficiency, stress-related mucosal disease, and coagulopathies encountered in critically ill patients. During infancy, gastroesophageal reflux disease (GERD), esophagitis, and infectious gastritis represent the predominant causes. In older children, peptic ulcer disease (PUD) and Helicobacter pylori infection are among the major etiological factors. In adolescents, etiologies more closely resemble those observed in adults, with variceal bleeding, nonsteroidal anti-inflammatory drugs (NSAIDs) use, and Mallory–Weiss tears occurring more frequently (Polat et al., 2020).

Table 1. Age-specific etiological distribution and clinical characteristics of UGIB in children

Age Groups	Major Etiologic Causes	Clinical Characteristics
Neonate (0–28 days)	Coagulopathies, stress gastritis, asphyxia	Severe systemic illness
Infant (1–24 months)	GERD, esophagitis, gastritis, infections	Hematemesis, vomiting
Early childhood (2–5 years)	Drug-induced gastritis, infectious gastritis, foreign body ingestion	Clinical manifestations are variable; most cases are mild to moderate in severity
School-age children (6–12 years)	PUD, Helicobacter pylori infection, erosive lesions	Epigastric pain may accompany bleeding episodes
Adolescents (13–18 years)	PUD, variceal bleeding, Mallory–Weiss tear, NSAIDs use	Adult-type etiologies become increasingly predominant
All age groups	Portal hypertension, stress ulcers, hematologic disorders	Associated with severe clinical course and higher risk of adverse outcomes

Clinical Findings

UGIB in the pediatric population is less common than in adults; however, it represents an important emergency condition associated with significant morbidity and mortality. The clinical

presentation may vary widely, ranging from mild symptoms to hemorrhagic shock, depending on the location, severity, duration, and underlying cause of the bleeding.

The most common manifestations of UGIB are hematemesis and melena. Hematemesis suggests active or recent UGIB, whereas coffee-ground emesis indicates the presence of digested blood that has been exposed to gastric acid. Melena is typically observed in bleeding originating proximal to the ligament of Treitz. Although hematochezia is generally suggestive of lower gastrointestinal bleeding, it may also occur in cases of massive UGIB in children. Occult GI bleeding is defined by the detection of microscopic blood in the stool. Hemobilia refers to bleeding into the biliary tract and is usually associated with traumatic or iatrogenic causes.

Clinical findings of GI bleeding in children may be more subtle compared with adults. In particular, during infancy, nonspecific symptoms such as irritability, pallor, lethargy, fatigue, and feeding difficulties may be observed (Thomson et al., 2017). In older children, epigastric pain, nausea, and vomiting may accompany the clinical picture. In cases of severe bleeding, hypovolemia and hemodynamic instability may develop. Tachycardia, hypotension, prolonged capillary refill time, cold extremities, and altered mental status are signs of hemorrhagic shock. However, due to strong compensatory mechanisms in children, hypotension is a late finding, and clinical stability should not be assessed based on blood pressure alone (ATLS Subcommittee et al., 2013).

In portal hypertension–related variceal bleeding, massive hematemesis is common, whereas peptic ulcer bleeding may present in a more insidious manner. In stress-related mucosal disease, particularly in intensive care unit patients, occult or minimal bleeding is often predominant.

Diagnostic Approach

The diagnostic process in pediatric UGIB follows a multistep approach that begins with clinical suspicion, prioritizes hemodynamic stabilization, and is completed with endoscopic confirmation. The fundamental principle in this process is that diagnostic interventions must never delay life-saving resuscitation measures.

The first and most critical step in UGIB is the establishment of hemodynamic stability. Diagnostic and interventional procedures should be carried out simultaneously with stabilization. After achieving hemodynamic stability, a detailed history and physical examination form the cornerstone of the diagnostic evaluation. In the history, the type, duration, volume, and recurrence of bleeding should be carefully assessed. Findings such as hematemesis, coffee-ground emesis, and melena suggest an upper GI source of bleeding. A detailed medication history is particularly important. NSAIDs, corticosteroids, anticoagulants, and chemotherapeutic agents may increase the risk of mucosal injury and bleeding. In addition, chronic liver disease, portal hypertension, hematological disorders, a history of peptic ulcer disease, and previous episodes of GI bleeding should be investigated.

In physical examination, vital signs should be carefully evaluated. Tachycardia, hypotension, orthostatic changes, and cold extremities may suggest hypovolemia. Findings such as pallor, petechiae, and ecchymosis can be indicative of underlying hematological disorders. Hepatosplenomegaly, ascites, and abdominal venous collaterals are important physical examination findings that suggest variceal bleeding secondary to portal hypertension (Rosen et al., 2018).

Laboratory evaluation is important both for assessing the severity of bleeding and for identifying the underlying etiology. In

the complete blood count, hemoglobin, hematocrit, and platelet levels are assessed; however, it should be kept in mind that hemoglobin levels may initially remain normal in acute bleeding. Coagulation tests are particularly important in cases where liver disease or coagulopathy is suspected. In biochemical analyses, serum blood urea nitrogen (BUN), creatinine, and electrolyte levels are evaluated. Elevated BUN levels are frequently observed in upper gastrointestinal bleeding. Liver function tests are useful in the evaluation of portal hypertension and chronic liver diseases.

Risk stratification in pediatric patients with UGIB is critical for clinical management planning. Patients are generally classified into low-, moderate-, and high-risk groups. Patients with stable vital signs and minimal bleeding findings are considered low risk, while those with mild tachycardia and a limited decrease in hemoglobin are classified in the moderate-risk group. Hemodynamic instability, active massive bleeding, severe anemia, or ongoing hematemesis are considered high-risk indicators (Thomson et al., 2017).

Endoscopy is the gold standard method for both the diagnosis and treatment of UGIB. It allows direct visualization of the bleeding source, identification of the underlying cause, and therapeutic interventions in the same session. Peptic ulcers, esophageal varices, erosive gastritis, Mallory–Weiss tears, and vascular lesions are the main pathologies that can be detected by endoscopy. Endoscopic treatment methods include band ligation, sclerotherapy, thermal coagulation, hemoclip application, and injection therapies (Rosen et al., 2018). Early endoscopic evaluation in hemodynamically stabilized patients is important for improving prognosis.

In selected cases where endoscopy is insufficient or further evaluation is required, imaging modalities may be used. Abdominal ultrasonography (US) and Doppler US are particularly useful for assessing portal hypertension, splenomegaly, and portal venous flow. Computed tomography (CT) angiography can be used to identify the

active bleeding site, while scintigraphic methods are generally preferred in cases of occult or intermittent bleeding (Kim et al., 2014).

Differential Diagnosis

In pediatric UGIB, the differential diagnosis aims to identify the true source of bleeding and to exclude conditions that may present with a similar clinical picture. Since certain non-gastrointestinal conditions in children can mimic UGIB, it is essential to distinguish true gastrointestinal hemorrhage from pseudo-hematemesis. This differentiation is achieved through careful history taking, detailed physical examination, and, when necessary, laboratory investigations. The differential diagnostic process is crucial for preventing unnecessary invasive procedures and ensuring accurate etiological classification.

Gastrointestinal causes are generally characterized by mucosal disruption or bleeding arising from vascular structures. This group includes gastric or duodenal ulcers secondary to PUD, erosive gastritis and duodenitis, esophagitis related to reflux or medication use, esophageal varices due to portal hypertension, Mallory–Weiss syndrome, and gastric or esophageal polyps.

Portal hypertension–related causes arise from vascular changes secondary to increased portal venous pressure and represent a significant risk for variceal bleeding (Cifuentes et al., 2021). This group includes esophageal and gastric varices, portal hypertensive gastropathy, and secondary thrombocytopenia due to splenomegaly as the main contributing factors.

Systemic and hematological diseases may also be associated with UGIB or may mimic it clinically by increasing mucosal bleeding (Kliegman et al., 2023). These causes include coagulopathies such as hemophilia and von Willebrand disease,

thrombocytopenias related to immune thrombocytopenic purpura and leukemia, as well as liver failure, sepsis, and disseminated intravascular coagulation (DIC).

Drug and toxin-related conditions may also lead to UGIB by causing mucosal damage or impairing coagulation mechanisms (Thomson et al., 2017). In particular, the use of NSAIDs, corticosteroids, anticoagulant therapies, and caustic substance ingestion leading to esophageal or gastric burns are among the important etiological factors.

In addition, some non-gastrointestinal conditions may mimic true UGIB, resulting in "pseudo-hematemesis." Differentiating these conditions is of great importance in order to avoid unnecessary invasive procedures. In neonates, swallowed maternal blood, vomiting of swallowed blood following epistaxis, oropharyngeal bleeding, and discoloration due to foods and beverages such as beetroot, carrots, turnips, or red food dyes are among the causes of pseudo-hematemesis (Kliegman et al., 2023). Furthermore, the use of iron- and bismuth-containing medications may also cause color changes in vomitus or stool that may be mistaken for bleeding.

Management of Pediatric UGIB

In pediatric UGIB, the treatment approach will be a multidisciplinary and stepwise process that will include controlling the bleeding, ensuring hemodynamic stability, treating the underlying etiology, and preventing complications. Management will generally start with emergency stabilization and will continue with medical, endoscopic, and, rarely, surgical interventions.

In all pediatric patients with suspected UGIB, the initial approach will be based on basic life support principles and ABC (Airway–Breathing–Circulation) assessment. In the first stage, airway patency will be evaluated, and airway protection will be

ensured, particularly in patients with active hematemesis, altered consciousness, or aspiration risk. Respiratory status will be assessed, and oxygen support will be provided when necessary, with ventilatory support planned if required. In circulatory assessment, heart rate, blood pressure, capillary refill time, and peripheral perfusion will be carefully evaluated. In patients with hemodynamic instability, a wide-bore intravenous access will be established and isotonic fluid resuscitation will be initiated, and in appropriate cases, erythrocyte suspension and blood product replacement will be administered. Especially in pediatric patients, since compensatory mechanisms will be strong, hypotension will appear as a late finding; therefore, tachycardia and impaired peripheral perfusion will be considered early warning signs (Rosen et al., 2018).

Medical therapy is a fundamental step in achieving hemostasis and preventing recurrence in UGIB, and it constitutes an integral part of the treatment process. In this context, proton pump inhibitors (PPIs) provide an effective treatment option, particularly in acid-related lesions such as peptic ulcer disease and erosive gastritis, by suppressing gastric acid secretion. PPIs such as omeprazole and pantoprazole reduce the acidic environment, thereby supporting mucosal healing and contributing to bleeding control.

In variceal bleeding, octreotide induces splanchnic vasoconstriction, reducing portal venous pressure and thereby assisting hemostasis. Through this mechanism of action, it is particularly used as an important therapeutic agent in esophageal and gastric variceal bleeding.

In patients with portal hypertension and variceal bleeding, antibiotic prophylaxis also plays a critical role in preventing bacterial infections and spontaneous bacterial peritonitis. It has been reported that antibiotic therapy used for this purpose is effective in reducing morbidity (Thomson et al., 2017).

Endoscopic interventions constitute the cornerstone of standard treatment approaches due to their high success rates and dual diagnostic and therapeutic roles. Among endoscopic treatment modalities, band ligation is applied as the first-line therapy in esophageal variceal bleeding, achieving hemostasis through mechanical strangulation of varices (Cifuentes et al., 2021). Sclerotherapy, on the other hand, aims to achieve obliteration by injecting a sclerosant agent into the varix and is preferred as an alternative treatment option in cases where band ligation cannot be performed. Cauterization and thermal hemostasis techniques are used particularly in ulcerative, erosive, or actively bleeding lesions to achieve hemostasis through vascular coagulation.

Surgical approaches vary depending on the underlying pathology and may include portal decompressive procedures or resection techniques (Thomson et al., 2017). In current practice, surgical treatment is rarely required in pediatric UGIB and is generally reserved for cases unresponsive to medical and endoscopic therapies. Indications for surgical treatment include massive bleeding refractory to medical and endoscopic management, recurrent and uncontrollable variceal bleeding, and the presence of vascular or anatomical abnormalities.

Specific Clinical Conditions and Management

Swallowed Maternal Blood

In neonates presenting with UGIB, swallowed maternal blood is one of the most common benign causes. This condition typically occurs as a result of ingestion of blood originating from cracked maternal nipples during delivery or breastfeeding and may present with hematemesis or melena. In most cases, it is not associated with any serious underlying pathology and follows a self-limiting, benign course.

Differentiating swallowed maternal blood from neonatal GI bleeding is important to avoid unnecessary invasive procedures. The Apt–Downey test, used for this purpose, is based on the difference in alkali resistance between fetal hemoglobin (HbF) and adult hemoglobin (HbA) (Apt & Downey, 1955). HbF is more resistant to an alkaline environment, whereas HbA is denatured under alkaline conditions. In this test, the specimen (vomitus or stool) is processed, and 1% NaOH is added to the supernatant; the interpretation is based on the resulting color change. Persistence of a pink-red color indicates fetal hemoglobin (neonatal blood), whereas a transition to yellow-brown color suggests maternal blood. In conclusion, the Apt–Downey test is a rapid and practical method for distinguishing swallowed maternal blood in neonatal UGIB and helps prevent unnecessary further investigations.

Coagulopathy-Related Upper Gastrointestinal Bleedings

A significant proportion of UGIB observed in early life is related to coagulopathies. In neonates, the hemostatic system is physiologically immature compared to adults, with marked differences particularly in coagulation factors and components of the anticoagulant system (Monagle et al., 2010). Vitamin K–dependent factors (II, VII, IX, and X) are present at low levels, and natural anticoagulants such as protein C and protein S are also reduced. Although platelet counts are generally within normal ranges, incomplete functional maturation may impair the hemostatic response.

Vitamin K deficiency bleeding is one of the most important and preventable causes of UGIB in the neonatal period. Due to the sterile intestinal environment, endogenous synthesis does not occur, and the low vitamin K content in breast milk increases the risk, particularly in exclusively breastfed infants. When prophylaxis is not administered, the risk of bleeding increases significantly, and the

clinical presentation may range from mild findings such as hematemesis and melena to umbilical bleeding and, in severe cases, life-threatening intracranial hemorrhage. The primary preventive approach is the intramuscular administration of vitamin K immediately after birth. A single intramuscular dose of 1 mg is recommended for term neonates, while a dose of 0.5 mg is recommended for preterm infants weighing less than 1500 grams (Kliegman et al., 2023).

Disseminated Intravascular Coagulation–Associated Upper Gastrointestinal Bleeding

Disseminated intravascular coagulation (DIC) is a life-threatening and complex hemostatic disorder characterized by systemic activation of coagulation secondary to an underlying primary disease (Levi & Scully, 2018). In the pediatric population, the main etiological factors include sepsis, perinatal asphyxia, shock, and necrotizing enterocolitis. These conditions trigger excessive activation of the coagulation system, leading to widespread thrombin generation. The underlying pathophysiology involves systemic microthrombus formation and subsequent consumption of coagulation factors and platelets, resulting in a consumptive coagulopathy. Consequently, the balance of hemostasis is disrupted, and both thrombotic and bleeding tendencies may occur simultaneously.

Clinically, DIC presents with diffuse bleeding involving multiple organ systems. UGIB frequently accompanies the clinical picture, and hemorrhages may also be observed in the skin, mucous membranes, and at sites of invasive procedures. In severe cases, the condition may progress rapidly and lead to life-threatening hemorrhagic complications. Therefore, DIC is one of the rare but most severe causes of upper gastrointestinal bleeding in childhood,

and early diagnosis together with prompt treatment of the underlying etiology is critical for prognosis.

Liver Disease–Associated Upper Gastrointestinal Bleeding in Children

The liver is responsible for the synthesis of a large proportion of coagulation factors; therefore, diseases affecting this organ may lead to disruption of hemostatic balance and an increased tendency to bleeding. In the pediatric population, the main causes of liver-related coagulopathy include neonatal hepatitis and biliary atresia. These conditions impair the synthetic capacity of the liver, leading particularly to a reduction in vitamin K–dependent coagulation factors (II, VII, IX, and X) (Kliegman et al., 2023). The primary consequence of coagulopathy secondary to hepatic dysfunction is a deficiency of coagulation factors and an associated increased bleeding tendency.

Clinically, patients may present with UGIB, along with additional mucocutaneous or systemic bleeding manifestations. In cases of coagulopathy secondary to liver disease, early diagnosis and treatment of the underlying hepatobiliary pathology play a critical role in preventing bleeding complications.

Congenital Coagulation Factor Deficiency–Associated Upper Gastrointestinal Bleeding

Congenital coagulation factor deficiencies are characterized by the inherited absence of specific coagulation factors involved in the intrinsic pathway of coagulation. The most common causes of congenital coagulopathies are Hemophilia A (factor VIII deficiency) and Hemophilia B (factor IX deficiency) (Castaman & Matino, 2019). In these disorders, impairment of the coagulation cascade leads to an increased tendency to bleeding, with prominent

hemorrhagic episodes particularly following trauma or invasive procedures.

Clinically, bleeding manifestations are more commonly observed as post-traumatic intramuscular bleeding, hemarthrosis, and soft tissue hemorrhages. However, in patients with severe factor deficiencies, spontaneous mucosal bleeding and upper gastrointestinal bleeding may also occur.

Thrombocytopenia and Platelet Function Disorders–Associated Upper Gastrointestinal Bleeding

Thrombocytopenia and platelet function disorders are among the important hematological causes of UGIB in childhood. These conditions increase bleeding tendency due to either a quantitative reduction in platelets or qualitative impairment of platelet function, both of which are essential components of primary hemostasis. In the pediatric population, the main causes of thrombocytopenia include neonatal alloimmune thrombocytopenia, sepsis, and prematurity (Roberts & Eikelboom, 2009). These conditions lead to impaired hemostatic mechanisms through decreased platelet production, increased peripheral destruction, or functional immaturity.

Clinically, mucosal bleeding is the most common manifestation in these patients. However, GI bleeding may also occur, presenting as UGIB. The severity of bleeding is directly related to the underlying cause and the degree of thrombocytopenia.

Stress Gastritis and Gastric Erosions

Stress gastritis and gastric erosions are among the important but relatively less common causes of UGIB in the pediatric population. They are particularly observed in infants and children who are critically ill and managed in intensive care units. Major risk factors include perinatal asphyxia, sepsis, respiratory distress

syndrome, and prematurity. Patients requiring mechanical ventilation, as well as those presenting with shock and hypotension, are at increased risk of gastric mucosal ischemia (Kliegman et al., 2023).

The pathophysiology is based on the disruption of the balance between mucosal defense mechanisms and aggressive factors. Reduced gastric perfusion due to hypoxia and hypotension leads to mucosal ischemia, which, combined with increased acid secretion and impairment of the mucus–bicarbonate barrier, decreases mucosal resistance. As a result, superficial erosions and bleeding may occur. Clinically, hematemesis and melena are the most common findings. The presence of blood in nasogastric aspirate is an important clinical sign. In some cases, occult bleeding may occur, presenting only as a decline in hemoglobin levels without overt hemorrhage.

Management primarily focuses on correcting the underlying condition. Supportive treatment includes hemodynamic stabilization and blood transfusion when necessary. Acid suppression therapy with H2 receptor blockers and PPIs may be used. Endoscopy is generally reserved for selected cases that do not respond to conservative management.

Trauma-related Upper Gastrointestinal Bleeding

Trauma-related UGIB is among the important clinical conditions encountered particularly in pediatric patients in intensive care and emergency department settings. These types of hemorrhages generally develop as a result of iatrogenic or mechanical disruption of mucosal integrity. Mucosal injury that may occur in the oropharyngeal and esophageal regions during procedures such as nasogastric tube placement or endotracheal intubation can lead to upper gastrointestinal bleeding. These conditions most commonly present as superficial mucosal erosions;

however, in the presence of accompanying coagulopathy, hypoxia, or stress states, the bleeding may become more pronounced.

Esophagitis/Reflux-Related Upper Gastrointestinal Bleeding

Esophagitis and GERD are among the important causes of pediatric UGIB. Prematurity, prolonged nasogastric tube use, mechanical ventilation, sepsis, hypoxia/ischemia, and stress-related increases in acid secretion are the main risk factors. These conditions may disrupt the integrity of the esophageal mucosal barrier, leading to erosive injury.

Clinically, patients may present with irritability, feeding intolerance, and mild, insidious bleeding. Hemorrhage can manifest as hematemesis, coffee-ground–like vomitus, melena, or occult blood positivity (Rosen et al., 2018). For diagnosis, blood detected in nasogastric aspirate and positive fecal occult blood testing are helpful; definitive diagnosis is established by endoscopy in selected cases. In most cases, a conservative approach is sufficient in treatment. Nutritional regulation, head elevation, and positional measures are recommended; pharmacologically, H2 receptor antagonists and, when necessary, proton pump inhibitors (PPIs) may be used (Lightdale & Gremse, 2013). In severe cases, fluid resuscitation, blood transfusion, and rarely endoscopic intervention may be required.

Vascular Anomalies

UGIB secondary to vascular anomalies, although rare in childhood, represents an important etiological group that may lead to significant morbidity and mortality. These lesions typically develop due to congenital or acquired vascular structural abnormalities and may present clinically as acute or chronic bleeding episodes.

Angiodysplasia is a vascular malformation characterized by dilation and fragility of small submucosal vessels (Rosen et al., 2018). Clinically, it may present with hematemesis or melena. Diagnosis is most commonly established via endoscopic evaluation, which allows direct visualization of the bleeding source. The first-line treatment is endoscopic coagulation techniques, with argon plasma coagulation being frequently preferred. In rare cases where endoscopic therapy fails, surgical intervention may be required.

Dieulafoy's lesion is a rare but life-threatening cause of UGIB, resulting from erosion of an abnormally large-caliber artery running in the submucosa beneath an otherwise normal gastric mucosa (Baxter & Aly, 2010). It is most commonly located along the lesser curvature of the stomach, although it may also be found in the esophagus, duodenum, and other gastrointestinal segments. Clinically, it may present with sudden-onset, massive, and recurrent hematemesis. Diagnosis is generally made by endoscopic examination. First-line treatment consists of endoscopic hemostatic methods, including clip application, band ligation, and epinephrine injection. In refractory cases, interventional radiology or surgical treatment may be required.

Hereditary hemorrhagic telangiectasia, also known as Osler–Weber–Rendu syndrome, is an autosomal dominant systemic disorder characterized by mucocutaneous and visceral vascular malformations (Shovlin, 2010). The underlying pathology consists of telangiectasias and arteriovenous malformations (AVMs) at the level of small vessels. Clinically, the most common manifestation is recurrent epistaxis, while GI bleeding typically occurs in later life and may lead to chronic iron deficiency anemia. In addition, pulmonary, cerebral, and hepatic involvement may result in hypoxemia, neurological complications, and high-output heart failure. Diagnosis is based on clinical findings and family history, and is confirmed when necessary by genetic testing and imaging

studies. Treatment includes endoscopic interventions, iron supplementation, and blood transfusion when indicated.

AVMs are defined as abnormal vascular connections between arteries and veins without an intervening capillary bed. They may present with a wide spectrum of clinical manifestations, ranging from chronic occult bleeding to acute massive hemorrhage (Shovlin, 2010). Endoscopy is the first-line diagnostic modality, while in advanced cases angiography plays both a diagnostic and therapeutic role. Treatment options include endovascular embolization and surgical resection in selected cases.

Gastric antral vascular ectasia (GAVE) is a rare condition characterized by mucosal and submucosal vascular dilatation, predominantly located in the gastric antrum, and is also referred to as “watermelon stomach” due to its characteristic endoscopic appearance (Hsu et al., 2018). It is very rarely seen in the pediatric population and typically presents with iron deficiency anemia secondary to chronic and insidious blood loss. Diagnosis is established endoscopically, with the typical appearance of radially arranged erythematous vascular stripes in the antrum. The most commonly used treatment is endoscopic argon plasma coagulation. In refractory cases, alternative endoscopic or surgical approaches may be required.

Hemangiomas, particularly infantile hemangiomas, are benign vascular tumors of vascular origin and represent the most common soft tissue tumors of childhood (Kliegman et al., 2023). Although they are most frequently cutaneous, visceral involvement may also occur. In cases of gastrointestinal involvement, patients may present with iron deficiency anemia due to occult blood loss or, rarely, with hematemesis and melena. Endoscopic evaluation plays an important role in diagnosis. The first-line treatment is

propranolol, while corticosteroids may be used as an alternative. In selected and refractory cases, surgical treatment may be required.

Mallory–Weiss Syndrome

Mallory–Weiss syndrome is one of the important causes of UGIB and is characterized by mucosal lacerations at the gastroesophageal junction, typically following severe vomiting (Lee & Ahn, 2019). In children, the most common causes include severe vomiting due to gastroenteritis and feeding intolerance. In addition, increased intra-abdominal pressure due to severe coughing episodes, crying, and straining may also lead to disruption of mucosal integrity. Rarely, trauma may also be an etiological factor. In infants with GERD, reflux-related straining may also contribute. Definitive diagnosis is made by upper GI endoscopy. The endoscopic finding of linear mucosal tears at the gastroesophageal junction is characteristic.

Most Mallory–Weiss tears heal spontaneously, and conservative management is usually sufficient. Treatment initially includes temporary cessation of oral intake, intravenous fluid support, and PPIs to reduce gastric acid secretion. Rarely, in cases of ongoing or severe bleeding, endoscopic therapy may be required. For this purpose, epinephrine injection or endoscopic clip application may be performed.

Hemobilia

Hemobilia is a rare but important cause of UGIB that occurs due to abnormal communication between the hepatobiliary vascular system and the biliary tree. The most common causes are liver trauma and hepatobiliary interventional procedures; it may develop following liver biopsy, percutaneous interventions, or surgical procedures. In addition, biliary tract anomalies, hepatic tumors, and

vascular malformations may also play a role in its etiology (Green et al., 2001).

Clinically, hemobilia is classically described by Quincke's triad—hematemesis or melena, right upper quadrant abdominal pain, and jaundice—although these findings are not always present simultaneously in all patients. Laboratory findings may reveal decreased hemoglobin levels, elevated direct bilirubin, and varying degrees of liver enzyme abnormalities.

Imaging modalities are essential in diagnosis. US and Doppler US may demonstrate blood or clots within the biliary tree as well as vascular abnormalities, while CT is useful for identifying active bleeding sites, intrahepatic hematomas, and pseudoaneurysms. Magnetic resonance cholangiopancreatography (MRCP) can provide detailed evaluation of biliary obstruction. The gold standard for both diagnosis and treatment is angiography, which also allows for simultaneous embolization.

Complications of Pediatric Upper Gastrointestinal Bleeding

Early hemodynamic stabilization, accurate etiological assessment, and appropriate endoscopic treatment can significantly reduce mortality and morbidity in UGIB. Complications primarily arise from the severity of blood loss, delays in diagnosis and treatment, and the presence of underlying systemic diseases. The most serious acute complication is hemorrhagic shock, which is characterized by tachycardia, hypotension, impaired peripheral perfusion, and altered mental status. Delays in appropriate fluid resuscitation and blood product support may lead to multiorgan failure (Thomson et al., 2017).

As a result of recurrent acute or chronic low-volume bleeding, iron deficiency anemia may develop. Particularly in non-variceal chronic bleeding, pallor, fatigue, exercise intolerance, and

growth retardation may be observed. Conditions such as esophageal varices, peptic ulcer disease, and Helicobacter pylori–associated gastritis are associated with an increased risk of recurrent bleeding. Persistent portal hypertension and inadequate treatment further predispose to rebleeding.

Prognosis

The most important determinant of prognosis is the etiology of the bleeding. In cases of bleeding due to causes such as peptic ulcer disease, erosive gastritis, and drug-induced mucosal injury, recovery rates are high with appropriate medical and endoscopic treatment, and the risk of recurrence is lower (Thomson et al., 2017). In contrast, variceal bleeding secondary to portal hypertension follows a more complex clinical course due to underlying chronic liver disease, and the prognosis is generally less favorable. Early endoscopic evaluation significantly improves outcomes by enabling both identification of the bleeding source and simultaneous therapeutic intervention.

Conclusions

UGIB in children, although less common compared to adults, is a life-threatening medical emergency due to its potential for rapid clinical deterioration. The etiology varies according to age; however, infections, peptic ulcer disease, variceal bleeding, and vascular lesions are among the leading causes. Successful clinical management relies on early diagnosis, effective hemodynamic stabilization, and prompt initiation of etiology-specific treatment. This approach plays a critical role in reducing both morbidity and mortality.

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BÖLÜM 2

Approach to empyema in the pediatric age group:

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1. Introduction: Empyema, known as the accumulation of purulent pus in any body cavity but commonly used as an indicator of pleural infection, has been a serious problem for centuries.Although pneumonia is the most frequent cause of empyema, the increasing number and variety of surgical interventions due to advances in thoracic surgery have also increased the incidence of postoperative empyema.Treatment can range from repeated needle aspirations or chest tube insertion in the appropriate location along with parenteral antibiotic therapy to thoracotomy (1).

2.History: Hippocrates practiced open drainage for the treatment of empyema in 500 BC (2). Hawit made the next advance in the treatment of empyema in 1876 by describing the closed tube drainage method, which involves placing a tube into the empyema cavity.Hawit was also the first person to use a closed underwater drainage system. Open drainage was accepted as the only treatment method for empyema following pneumonia, which was common during World War I, but it was unsuccessful. As a result, the empyema commission, headed by Dr. Evarts Graham, made the following conclusions, which form the basis of empyema treatment today and have continued from history to the present (3):

1. Pleural fluid should be drained, but open pneumothorax should be avoided in the acute pneumonic phase.
2. Patients should be protected from empyema by rapid sterilization and narrowing of the infected cavity.
3. Special attention should be paid to the patient's nutrition.

3.Pleural Effusions: Before the development of antibiotics in the 1930s, empyema was seen in 10% of patients with pneumonia. With the effective treatment of pneumonia with antibiotics, the incidence of parapneumonic empyema has also decreased significantly. However, despite the widespread use of broad-spectrum antibiotics, bacterial pneumonias and parapneumonic effusions and empyemas still pose significant problems in Turkey and worldwide, especially in children. Parapneumonic empyema in children is a serious complication seen in approximately 0.6% of patients with bacterial pneumonia. Despite effective antibiotics and intensive care, the mortality rate is significantly higher in children with empyema (6-12%). The most common cause of pleural effusion in children is pneumococcal pneumonia. Bacteria in the pleural fluid form infection products that prevent the full expansion of the lung. Thoracic empyema may be missed if not suspected early, and the diagnosis may be masked by another underlying disease (4). Most pleural effusions due to pneumonia spontaneously resorb with treatment of the primary disease without any treatment for the effusion, while 10% require surgical intervention. Delay in appropriate treatment of the effusion significantly increases morbidity (5).

Pleural effusions are divided into three types (6):

1. Dry (plastic) pleurisy: Develops during acute bacterial lung infections or acute upper respiratory tract infections. It can also be seen in tuberculosis, collagen tissue diseases, and lung abscesses. The disease is usually in the visceral pleura. A very small amount of yellow, serous fluid may be present.

2. Serofibrinous pleurisy: Can be seen in all lung infections, especially tuberculosis. It can also develop in mediastinal and abdominal infections. Less frequently, it occurs as a complication of connective tissue diseases. It is usually unilateral. Pleurisy developing in non-tuberculosis infections heals quickly unless the fluid takes on a purulent character. Effusion seen in collagen tissue diseases and tuberculosis, however, lasts longer and adhesions may occur between the pleural layers over time.

3. Empyema (purulent pleurisy): There are four stages in the development of empyema, which occurs as a result of bacterial invasion of the pleural space, that cannot be clearly distinguished from each other (7):

3a. Pleuritis sicca stage: The inflammatory process in the lung parenchyma reacts in the pleura and extends to the visceral pleura. This causes characteristic chest pain resulting from pleural friction and the sensitive innervation of the parietal pleura. In a significant proportion of pneumonic patients, pleuritic chest pain occurs without the development of pleural effusion, and in most patients, pleural involvement remains limited to this stage (3). Exudative stage: Characterized by the rapid filling of the pleural space with sterile pleural fluid. This fluid is thought to be due to increased permeability secondary to pneumonia. The pleural fluid is usually clear, sterile, and small in quantity. It contains a small number of polymorphonuclear leukocytes (PNL), LDH <1000 IU/L, pH and glucose are normal, and the fluid is clear and sterile. In this stage, appropriate antibiotic treatment alone is sufficient.

3b.Fibropurulent stage: In patients who have not been treated with appropriate antibiotics or who have not received adequate dose and duration of treatment, bacteria from the pneumonic lesion pass into the pleural fluid. The pleural space fills with a large amount of thick, viscous, inflammatory fluid containing numerous PNLs, bacteria, and cellular debris. Fibrin membranes form, leading to loculations due to the accumulation of fibrin debris on both pleural layers. These loculations prevent the spread of empyema but, on the other hand, make drainage difficult with thoracentesis and tube thoracostomy, ultimately reducing the lung's expansion capacity. With the increase in metabolic and cytological activity in the effusion, the pH and glucose of the pleural fluid progressively decrease, and the LDH level increases significantly. Although radiography alone is not sufficient for diagnosing empyema, loculated fluid should be suspected if a fluid layer is not seen on a lateral decubitus radiograph.

3c.Organization phase: In this phase, fibroblasts and capillaries invade the visceral and parietal pleura to form a non-elastic membrane (pleural shell). These non-elastic membranes surround the lung, making re-expansion difficult and reducing its function.

Fluid viscosity is increased, pH is 7.0, glucose is below 40 mg/dL, leukocyte count is greater than 15,000/mm3, and LDH level is greater than 1,000 IU/L. This phase begins 7-10 days after the accumulation of effusion and is completed in 4-6 weeks. If left untreated, it can lead to a high-risk chronic empyema condition such as permanent defects in lung function, bronchopleural fistula, lung abscess, or empyema necessitate (drainage of infected fluid through a weak area of the chest wall). The pleural membrane may be visible on a postero-anterior (PA) chest X-ray, but it is often more clearly seen on computed tomography (CT).Thoracentesis performed at this stage may be dry or purulent fluid may not be obtained due to inhibition caused by leukocyte movement in fibrin membranes (8).

4.Etiology: Empyema is usually a secondary disease, and microorganisms reach the pleura either directly through proximity or metastatically via hematogenous or lymphatic routes. Empyema most frequently develops after pneumonia in both adults and children. In children, the causes of empyema, in order of frequency, are pneumonia, trauma, surgical procedures, collagen tissue diseases, metastatic carcinoma, and cardiac diseases.

5.Epidemiology: The bacteriology of infected pleural fluid in children differs from that in adults. Although the causative agent varies according to the child's age, the most common responsible microorganisms are H. influenza (often type B), Strep. pneumoniae, and Staph. aureus. In patients younger than six months, Staph. aureus has been detected in more than 50% of cases. Although H. influenza is the predominant pathogen from 7 to 24 months of age, it is detected less frequently between the ages of 2 and 15 years.

The most frequently isolated pathogens in later ages are Staf. aureus and Strep. pneumonia. Anaerobic organisms are rarely isolated. Anaerobes, other gram (-) bacteria, multibacterial isolates and atypical microorganisms are most frequently detected between the ages of 5 and 15 years (9).

Parapneumonic effusion and empyema are frequently encountered in spring and winter months. They are observed more often in boys than in girls. Although most pediatric patients have not had any previous complaints, patients with underlying diseases such as cerebral palsy, hypogammaglobulinemia, chronic granulomatous diseases, Down syndrome, congenital heart disease, prematurity, cystic fibrosis, tuberculosis, and esophageal stricture have also been identified (4).

5.Clinical: The clinical course of parapneumonic effusion or empyema varies depending on

the causative microorganisms, the level of pus in the pleural space, previous antibiotic treatment, and the child's general condition. The most common clinical signs in patients with empyema in whom aerobic microorganisms are detected are fever that does not subside, other common complaints include weakness, mild non-productive cough, chest pain, mild shortness of breath, and weight loss (10). Severe respiratory distress occurs, especially in infants. The patient takes on a toxic appearance. Empyema should be considered if pneumonia takes a septic course or if existing symptoms are exacerbated. Antibiotic treatment may make the clinical picture less pronounced and may make differentiating between pneumonia and empyema more difficult. The most common symptoms are shortness of breath (82%), fever (81%), cough (70%), and chest pain (67%). All of these symptoms are also present in pneumonia. The appearance or progression of these symptoms in a patient with febrile respiratory disease should suggest the possibility of empyema. Occasionally, patients present with signs of sepsis, including severe respiratory distress and hypotension (4). Not all patients with aerobic empyema are acutely ill. Therefore, the absence of fever or chest pain should not rule out the diagnosis of empyema. Anaerobic empyema is rare in children. Unlike aerobic empyema, these patients have a subacute clinical picture. These children often have poor oral hygiene and an accumulation of anaerobic bacteria in their oropharynx. If empyema has been present for 6 weeks or longer, it is in the chronic phase. In chronic empyema, the movement of the affected hemithorax may be restricted. In chronic empyema resulting from delayed diagnosis or inappropriate treatment, patients are usually weak and may have anemia and clubbing of the fingers.

On physical examination, fever, tachypnea, decreased breath sounds, dullness in the affected areas, contractions in the affected hemithorax over time, and restricted chest movement are detected in the acute phase. Chest wall abscesses may develop due to erosion of the chest wall by empyema. Scoliosis may develop in severe empyema and chronic cases. In childhood, bronchopulmonary fistulas and pyopneumothorax are frequently seen, more often after staphylococcal empyema. Purulent pericarditis, lung abscess, osteomyelitis, meningitis, and arthritis may also occur. Ultrasonography (USG) can be used to differentiate pleural effusion from pleural thickening. USG-guided thoracentesis has been shown to be accurate and useful, especially in young children with early pleural effusions (11). Magnetic resonance imaging (MRI), especially T-sagittal sections, shows the chest wall layers in detail. It can be useful in determining the cause and content of undiagnosed fluid accumulations in the pleural space, but it does not eliminate the need for thoracentesis or other invasive diagnostic procedures. However, thoracentesis is important for differentiating between parapneumonic effusion and empyema. 30-50 cc of pleural fluid obtained with 18-19 Gauge needles is examined using macroscopic, microscopic, biochemical, and microbiological methods. If clinically suspected, tuberculosis, fungal culture, and staining should be performed. Purulent fluid is an important diagnostic method for empyema. Cloudy fluid should be centrifuged to determine whether the cause is leukocytes or lipids. After centrifugation, clear fluid suggests leukocytes, while cloudy fluid suggests lipids. If the fluid in the pleural space is clear during the first thoracentesis, it should be examined for exudate or transudate. However, it has been concluded that the important features in differentiating transudate and exudate in adults are not the same as in children (12). Gram staining, aerobic and anaerobic culture antibiogram are performed on the fluid. The pH of the cloudy fluid should be studied. In cases where obvious pus is obtained, there is no need to examine the pH.

6.Treatment: The main goal in the treatment of childhood parapneumonic empyema is to drain the infected pleural space, correct the parenchymal infection with appropriate antibiotic treatment, ensure re-expansion of the compressed lung, and prevent complications that may develop in the acute and chronic phases. Due to the increase in childhood parapneumonic empyema, the variety of treatment approaches (repeated thoracentesis, tube thoracostomy, fibrinolytic therapy, video-assisted thoracoscopy (VATS) or decortication with open thoracotomy) is also increasing. However, the most appropriate treatment method has not yet been definitively determined within this variety.

The treatment principles in complicated parapneumonic effusion and empyema are generally as follows (13):

1. Preservation of life
2. Drainage of effusion or empyema
3.Elimination of pleural space for re-expansion of the compressed lung
4. Increasing the patient's natural resistance with appropriate antibiotics
5. Ensuring chest wall and diaphragm mobility
6. Nutritional support
7. Ensuring normal lung function
8. Prevention of complications and chronicity
9. Shortening hospital stay

All methods used in the treatment of childhood parapneumonic empyema, such as repeated thoracentesis, image-guided catheters, closed tube thoracostomy, open drainage, fibrinolytic therapy, thoracoscopy or decortication with thoracotomy, have been reported with success rates between 10% and 90% (7). The reason for the varying success rates is mostly attributed to the stage of empyema. In the clinic, the stage of empyema can generally be estimated by the duration of symptoms (4). However, the three stages of pleural empyema may not develop sequentially and at the same time in every patient.

There may also be empyemas that reach the organization stage in as little as one week (5).

The macroscopic appearance of pleural fluid, glucose, LDH, pH levels, and Gram staining studies play an effective role in performing tube thoracostomy.Since fluid accumulation in the pleural region can lodge after two days and obstruct tube drainage, these laboratory studies should be completed as soon as possible (14). In thoracentesis, a dark-consistency and colored fluid indicates the possibility of empyema, and in this case, it suggests the necessity of performing tube thoracostomy on the patient. Intermittent thoracentesis is unnecessary in children with complicated empyema. The pH and glucose levels of the fluid accumulated in the pleural space are important for tube thoracostomy in parapneumonic effusion. However, in cases of fluid accumulation in the pleural space due to systemic acidosis, rheumatoid disease, malignancy, or tuberculosis, tube thoracostomy is not necessary despite low pH and glucose levels.Although the treatment of parapneumonic empyema in children is generally similar to that in adults, treatment practices in children are still controversial. While many studies indicate that surgical procedures are rarely necessary (10,15-17), some authors have stated the benefits of decortication in advanced cases (1,8).

The specific treatment depends on the degree of disease at diagnosis, whether there is severe lung compression, and the patient's general condition. However, in any case, tube thoracostomy should be the first treatment option for childhood parapneumonic empyema, as it has been found to be successful in 80-90% of cases with tube thoracostomy alone (15,18). Antibiotic selection should always be broad-spectrum, based on the culture result and/or the patient's age and predisposition.

7**a.Conservative surgical interventions**

7a.1. Tube thoracostomy: In complicated pleural effusions and empyemas, drainage of the pleural space is as important as antibiotic treatment. The chest tube should be placed in the area where the pleural effusion is located. The inadequacy of tube thoracostomy is often due to improperly placed chest tubes (19). Applying negative pressure to the chest tube will help expand the underlying lung. The size of the chest tube to be used should be as large as possible according to the density of the fluid. The success of closed underwater drainage in empyema is understood by the clinicaland radiological improvement of the patient within 24 hours. Otherwise, drainage is insufficient and/or inappropriate antibiotics are being used. USG and CT evaluation should be repeated, the position of the chest tube should be changed according to the loculations, or a second tube should be inserted. In children, tube thoracostomy is curative in most patients.

7**a.2. Intrapleural fibrinolytic therapy:** This is based on the intrapleural use of fibrinolytic agents to achieve enzymatic lysis and debridement of pleural adhesions in loculated pleural effusions. In adults, 250,000 IU streptokinase or 100,000 IU urokinase is used. One of these agents is diluted with 100 cc of saline and administered into the intrapleural space via a chest tube, and the tube is kept closed for 1-4 hours. This procedure can be repeated several times. Its use in children was first reported in 1993. Since this date, reports on the use of both

streptokinase and urokinase have yielded very different results regarding the recommended dose, timing, and treatment outcomes for children (1,2,20). Although fibrinolytic therapy appears less invasive, it can cause traumatic and intolerable side effects, especially in young children. Due to the systemic absorption of streptokinase, systemic side effects such as major hemorrhage, fever, and pleural pain may occur. Urokinase is more advantageous because it is non-antigenic and non-pyrogenic. However, it should not be forgotten that both agents can cause anaphylaxis and acute hypoxemic respiratory failure.They can cause a measurable change in systemic coagulation parameters. The high failure rate of intrapleural fibrinolytic therapy and the lack of comparative studies in children make its indication in children controversial.

7**a.3. Thoracoscopy:** In cases of complicated parapneumonic effusion and empyema where the response to tube drainage, antibiotics, and possibly intrapleural fibrinolytic therapy is insufficient, it is the next treatment step for separation and debridement of adhesions. Although considered a minimally invasive method, it is a procedure that requires general anesthesia.Intrapleural fibrinolytic therapy is very successful when applied in the early stages of the disease (21,22). However, a single chest tube with a sufficiently large diameter and placed in a suitable location may be sufficient for the loculations to merge and drain easily in multiloculated empyemas (15).

7b.Aggressive surgical interventions

7b.1. Open drainage: This is a method rarely applied in patients who are too high-risk to tolerate thoracoscopy or thoracotomy. The pleural space is directly exposed to atmospheric pressure.The classic open drainage technique is absolutely unsuitable in children due to the skeletal deformities that may occur in the late stages. However, in children in whom pneumothorax is not expected to develop after the end of closed underwater drainage, a thin polyethylene catheter can be inserted in place of the old chest tube to ensure drainage of the empyema cavity, and the part of the catheter outside the thorax can be followed under a pet (15).

7**b.2.Decortication/pleurectomy:** This is a surgical intervention that can be preferred in patients in whom conservative treatment such as closed underwater drainage tube thoracostomy, intrapleural fibrinolytic agents, and thoracoscopy is insufficient. All fibrous tissues are peeled away from the visceral pleura, and inflammation is removed from the pleural space. This is done to allow the underlying trapped lung to expand. However, it should be remembered that pleural thickenings may resorb spontaneously over time (23). Resorption is faster in children than in adults. In addition, empyema can be successfully treated with other conservative methods in children without the need for decortication (15). On the other hand, it has been reported that early thoracotomy provides early recovery and that long-term follow-ups are excellent (8,24).

7**b.3. Thoracoplasty:** This is a treatment method that is not used unless absolutely necessary in the treatment of parapneumonic empyema in adults. It is not recommended to apply it in children due to long-term complications.

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BÖLÜM 3

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PRENATAL ETIOLOGY AND MANAGEMENT OF FETAL HYDRONEPHROSIS

INTRODUCTION

Fetal hydronephrosis (dilation of the renal pelvis dilatation or expansion of marine without renal), is a common finding on antenatal ultrasound. In most cases, renal pelvic dilation is a transient physiological condition. In some other cases, fetal hydronephrosis, congenital anomalies of kidney and urinary tract (CAKUT) or vesicoureteral reflux (vur) is the first presentation of. These conditions may be associated with the development of impaired kidney and/or urinary tract infection, the patient and/or may lead to an increased risk of kidney damage.

Fetal hydronephrosis definition, etiology, and management is reviewed here. Fetal hydronephrosis and postnatal urological evaluation of fetal hydronephrosis that may arise as particular cases are discussed separately:

DESCRIPTION AND RATINGS

Overview of Fetal hydronephrosis have been developed to diagnose the severity and rate of various systems [1].

●Renal pelvic diameter (RPD), also anterior-posterior renal pelvic diameter (APD) is known as

●Society for fetal urology (SFU) grading system

●Urinary tract dilatation (UTD) classification system

These systems focused on the components of the fetal kidney is slightly different. Prenatal screening with ultrasound for RPD, in other parts of the urinary tract (Kali, the ureters, and bladder) for risk stratification is supported by the observations of the dilation parameter on the primary startup;

Renal parenchyma, bladder, and urethra of view; and the assessment of oligohydramnios [1].

The severity of hydronephrosis, urinary tract obstruction is associated with the possibility of important [2]. The most important risk pregnancy before 28 weeks RPD 7 mm or ≥10 mm by 28 weeks after conception, is associated with permanent bilateral urinary tract dilatation at any time. These values, kidney and urinary tract (CAKUT) provides a reasonable threshold to detect fetuses at risk for congenital anomalies most cases transient and benign hydronephrosis us kacindirir possible due to unnecessary tests [3].

Renal pelvic diameter — Fetal hydronephrosis is the most generally accepted method to identify and rank the transverse plane or APD RPD, known as the maximum anteroposterior diameter of the renal pelvis and fetal ultrasound measurement [4]. RPD, is a measure of the expansion of the collection system, and increased echogenicity, parenchymal thinning or kaliektazi as hydronephrosis and does not reflect changes size. Measurement of the fetal spine at 6 o'clock or 12 o'clock position and the serial ultrasounds should be done in the same position that was used for. The gestational age of the fetus depends on interpretation of RPD (Table 1) [1]:

Table 1	renal pelvis diameter (RPD)	other ultrasound findings are
normal risk	28. the week before: <4 mm after 28 Weeks: <7 mm	kidneys, bladder, or have any abnormalities in the urinary tract.
Low Risk	28. the week before: ≥4 <7 mm after 28 Weeks: ≥7 to <10 mm	or isolated plant Calix Normal dilatation (SFU 2. degrees)
of the increased risk of	28. the week before: ≥7 mm after 28 Weeks: ≥10 mm	and/or any of these: the expansion of Peripheral Calyx (SFU degree ≥3) the thickness of the renal parenchyma or view abnormal Ureteral dilatation of bilateral hydronephrosis and Abnormal bladder (thickened, megacystis, ureterocele) or dilated posterior urethra amniotic fluid decrease

In this table, 28 of pregnancy. prenatal ultrasound findings made the week before or after, based on the classification of the risk of having clinically significant renal or kidney disease are summarized. Risk category, based on the most alarming findings in ultrasound. For example, the measurement of RPD low risk category, even though *the presence of abnormal renal parenchyma* is classified as increased risk.

●Before 28 weeks of pregnancy:

Normal – RPD <4 mm

Mild dilatation

– RPD ≥4 <7 mm

Moderate to severe dilatation

RPD ≥7 mm

•Moderate to severe dilation

RPD ≥7 mm

●After 28 weeks of pregnancy

•Normal – RPD <7 mm

•Moderate dilatation – RPD ≥7 to <10 mm

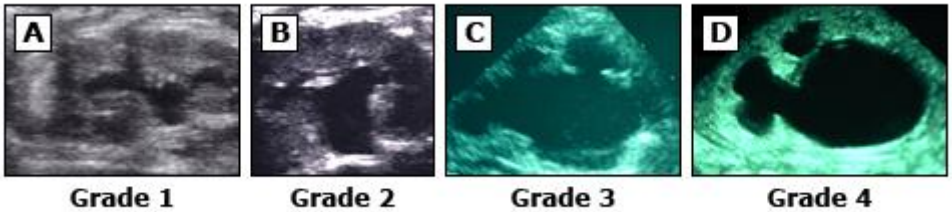
•Moderate to severe dilatation RPD ≥10 mm

This classification, in the second trimester RPD> 10 mm that is associated with an increased risk for CAKUT is based on several studies [2,4-6]. RPD in the third trimester> 15 mm are at the greatest risk for fetuses with cakut [2,4-7]. Pyelektazi known as mild renal pelvic dilatation in the second trimester, an RPD ≥4 to 7 mm is defined as [4-6,8]. Mild renal pelvic dilatation of the kidney will be fine and the majority of cases will have an impact on the development of Neonatal Clinical, although not permanent, there are reports of cases requiring intervention postpartum [2,9-11]. Therefore, it is recommended that an ultrasound follow such babies after 32 weeks [1].

Gestational age, maternal hydronephrosis, and bladder distention can affect the degree of maternal hydration RPD [12-15].

For Fetal Urology hydronephrosis in the community rating system

Picture 1



Degrees 0 – No expansion (not shown)

1. Degree – Only in the renal pelvis can be viewed.

2. Degree of the renal pelvis and ureter (but not all) can be viewed.

3. Degrees – almost all problems become visible.

4. Degree – 3. degree is similar, but compared with the normal renal parenchymal thinning, there are.

Fetal Urology Association — SFU class for the grading of hydronephrosis on postnatal ultrasound is the most commonly used system, however, can be applied to prenatal ultrasound. Degree, and the degree of pelvic dilatation of the region, and on the presence and severity of parenchymal atrophy seen in the number of marine (**Figure 1**) [16] based on the findings of kidney focuses on. The rating system, ureters, and bladder does not consider the status of:

●Grade 0 – normal renal pelvis is dilated, where examination.

●Garde I – just mild dilatation of the renal pelvis (**Figure 2**).

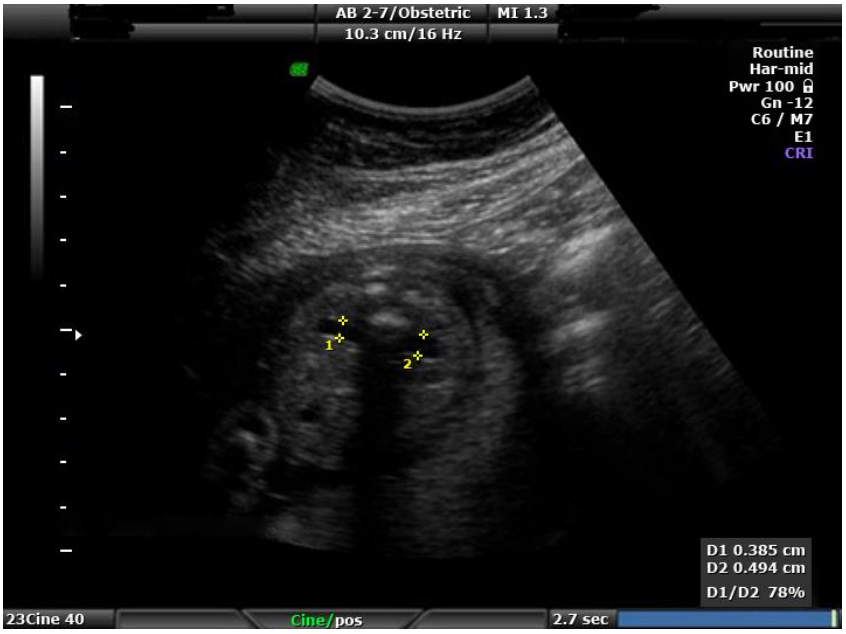


Figure 2: bilateral enlargement of the fetus in the urinary tract on both sides of 4 mm was detected.

●Grade II – moderate expansion of the renal pelvis and Calyx, including a few

.●Grade III – dilated renal pelvis and the renal parenchyma with normal visualization of all the disorders that are properly expansion.

●Kalye of Grade IV renal pelvis and III degree, plus a similar view, with thinning of the renal parenchyma (**Figure 3**).

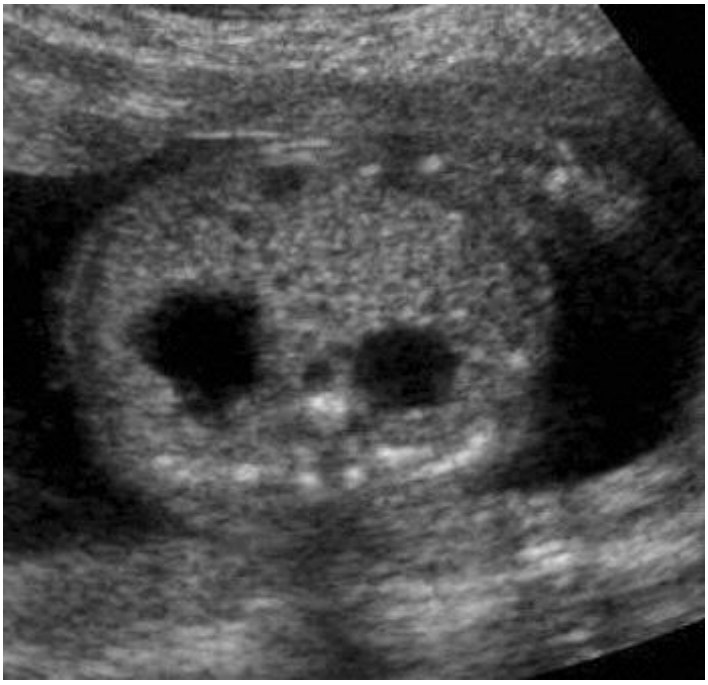


Figure 3: axial image of the abdomen of a fetus in the second trimester; spine, kidney and hydronephrosis 2 (nefroz) is observed.

Urinary tract dilation classification system: the classification UTD, gestational age, and birth after birth, whether before or classified according to the following six detection is based on ultrasound findings (**Figure 1**) [17]. Ultrasound findings the severity of the most severe, according to findings by rating, a numerical system (1 through 3, the most severe degree 3) is indicated by.

●Front-to-rear RPD

●Expansion Calyx

●Renal parenchymal thickness

●The appearance of the renal parenchyma

●Bladder abnormalities

●Ureteral abnormalities

The use of the system UTD, SFU modifiers requires more comprehensive compared to the system to determine the level of urinary tract obstruction. For example, the SFU patient's IV. degrees ureteropelvic junction (UPJ) SFU hydronephrosis or IV. junction type hydronephrosis

that is ureterovezikal degrees when specifying the severity and etiology of urologic abnormalities is not clearly documented. This, ureteral dilatation of the ureter and the bladder behind by defining the presence or absence of the diameter is provided. In contrast, UTD 3. degrees, numerous additional diagnosis and to convey the severity modifier requires.

Several observational studies are available that examine the benefits of Utd [18-20]. For example, in a study it conducted on isolated antenatal hidronefrozlu baby is about 500, the rate of kidney damage in early adulthood, was associated with postnatal ultrasonography in the Utd classification (UTD P1 for 0%, UTD P2 %P3 than for Utd and 14 56%) [20].

EPIDEMIOLOGY

Reported pregnancies, the incidence of Fetal hydronephrosis 0.6 to 4.5% ranged from. Differences in the reported data, disorder of the urinary system and ultrasound criteria that are used to describe different experts may be due to the level of his attention to [2,9,21-25]. Hydronephrosis, is approximately twice as common men than women. 20% of cases are bilateral and 40 [26].

Prevalence data examples include the following:

●17 studies, meta-analysis, 1678 104.572 woman's fetus (1.6 percent) fetal hydronephrosis was detected [2]. However, the criteria which are used for the diagnosis of hydronephrosis, differed between the included studies.

●Attended by a Belgian prospective study of randomly selected women 5643, renal pelvis diameter (RPD) ≥4 mm was defined as mild fetal hydronephrosis, pregnancies %4,5% were detected by ultrasonography in the second trimester [9].

ETIOLOGY

Hydronephrosis is transient: Transient hydronephrosis, and remitted clinically significant time in non-expansion of the renal pelvis in prenatal period (as defined above) refers. Fetal hydronephrosis is usually temporary and hidronefrozlu particularly in light of the fetus (before 28 weeks of pregnancy renal pelvis diameter [RPD] 4 <7 mm) patients (25% to 75% have been reported in [2,3,27,28]. Remaining stable or resolved without clinical impact of the proposed mechanisms as the fetus matures, the development in the early stages of the ureteropelvic junction (UPJ) or temporary a temporary narrowing of the fetal curves.

Congenital anomalies of kidney and urinary tract (CAKUT) — congenital anomalies of kidney and urinary tract (CAKUT), obstructive (e.g., UPJ obstruction, posterior urethral caps [PUV]) or non-obstructive (e.g. primary vesicoureteral reflux [SHOOT]), various upper and/or lower urinary tract abnormalities identifies. Any one of these disorders with fetal hydronephrosis may show itself.

Of these cases during pregnancy or immediately after birth is important because the determination of these potential risk factors for urinary tract infection and chronic kidney disease. However, varies depending on the cause of the clinical effect. Obstructive causes such as PUV, surgical intervention may limit independent of life or serious long-term consequences, while hit-and the majority of cases any long-term resolves on its own without any sequela.

1678 baby's Fetal hydronephrosis diagnosed in meta-analyses, postpartum assessment of fetal hydronephrosis has determined that an underlying cause of cakut in one third of patients [2].

●The most common types of cakut were identified :

•Ureteropelvic junction obstruction (**Figure 4**), in general, along with prenatal hydronephrosis was the most common diagnosis by increasing the frequency and severity of

light hidronefrozlu fetus in approximately 5% in fetuses with severe hidronefrozlu to 55% has changed.

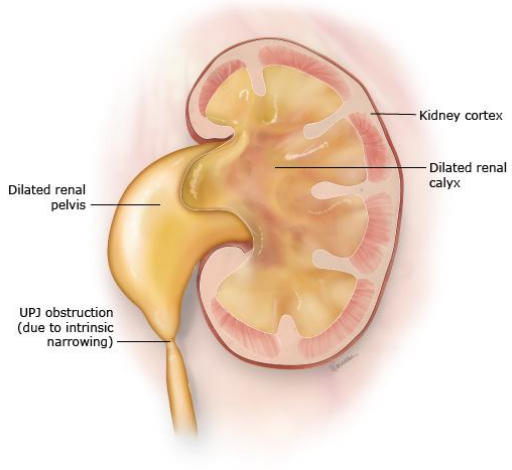


Figure 4: ureteropelvic junction obstruction because of the expansion of the renal pelvis and ureter in the kidney that is seen in illustration. In this case, the congestion is the cause of ureteropelvic junction obstruction is the most common stems from the inner contraction.

•SHOOT, light hidronefrozlu fetus in fetus with severe hidronefrozlu approximately 4 to 9% at rates ranging, the second most common diagnosis. The clinical significance of management and administration, the severity and primer (ureterovezikal of the junction resulting from the functional failure) or secondary (caused by abnormally high pressure bladder) depends on. Primary VUR is usually spontaneous remission in infancy.

●Less common causes include:

•Lower urinary tract obstruction, PUV (men) (**picture 5a**) or urethral stenosis/atresia included.

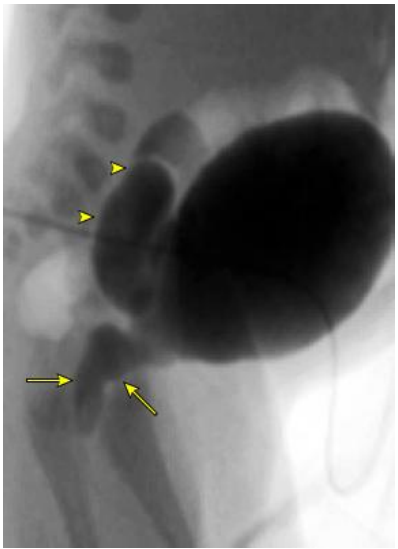


Image 5a: the urethra, enlarged Arrows, arrowheads demonstrate vesicoureteral reflux associated with dilated ureters with.

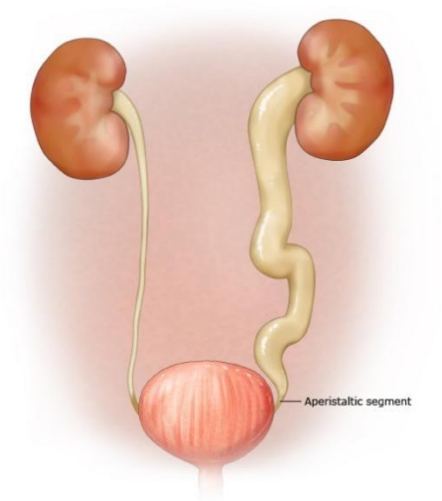


Figure 6: a schematic diagram of the primary megaureter, peristaltic ureterovezikal intersection non-segment shows the location of the occlusion. Megaureter, is defined as the ureter, which is larger than 7 mm in diameter (infants and in children under 12 years of age).

•Multi-cystic dysplastic kidney.

•Ureterocele (**Figure 5 A,B, and Figure 6**).

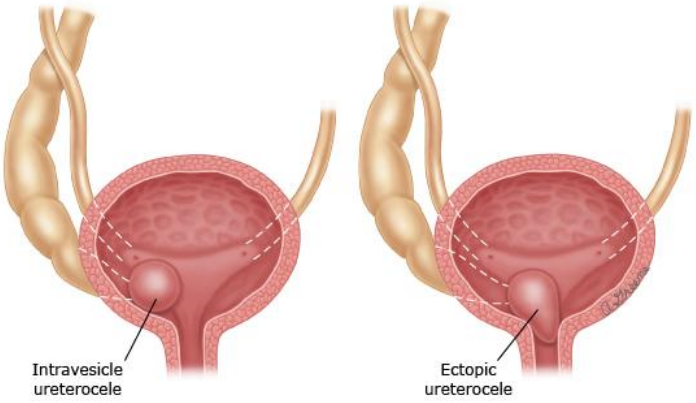
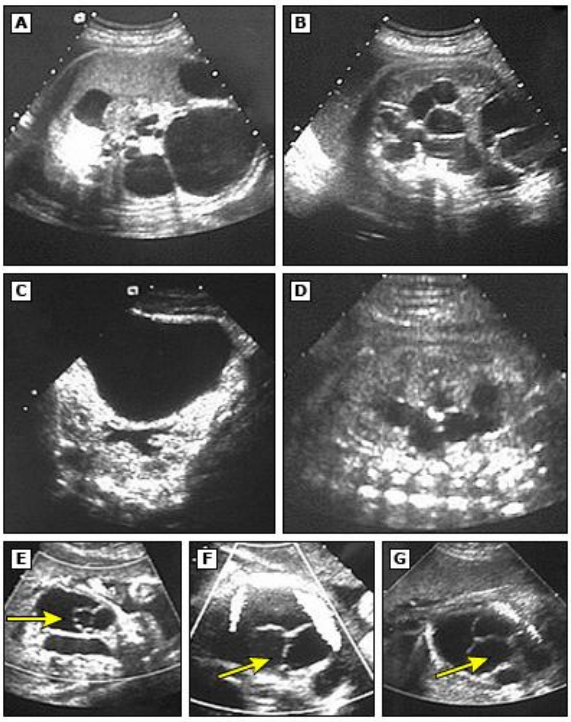


Figure 6: located within the bladder wall, ureterocele, ureteral cystic expansion of the segment. Intravesical ureterosel is contained within the bladder completely. If the urethra or bladder neck ureterosel partially ectopic location.



Picture 5b: 22 of gestation. held in the week of prenatal ultrasonography in ureterosel causing bilateral hydronephrosis was determined.

(A, b) Left hydronephrosis.

(C, D) right hydronephrosis.

(E, f, g) two-sided ureterocele in the bladder ultrasonography.

•Ectopic ureter.

•Prune Belly Syndrome syndrome.

•Bidirectional collection

•Cloacal and urogenital sinus anomalies.

Genetic and syndromic abnormalities associated

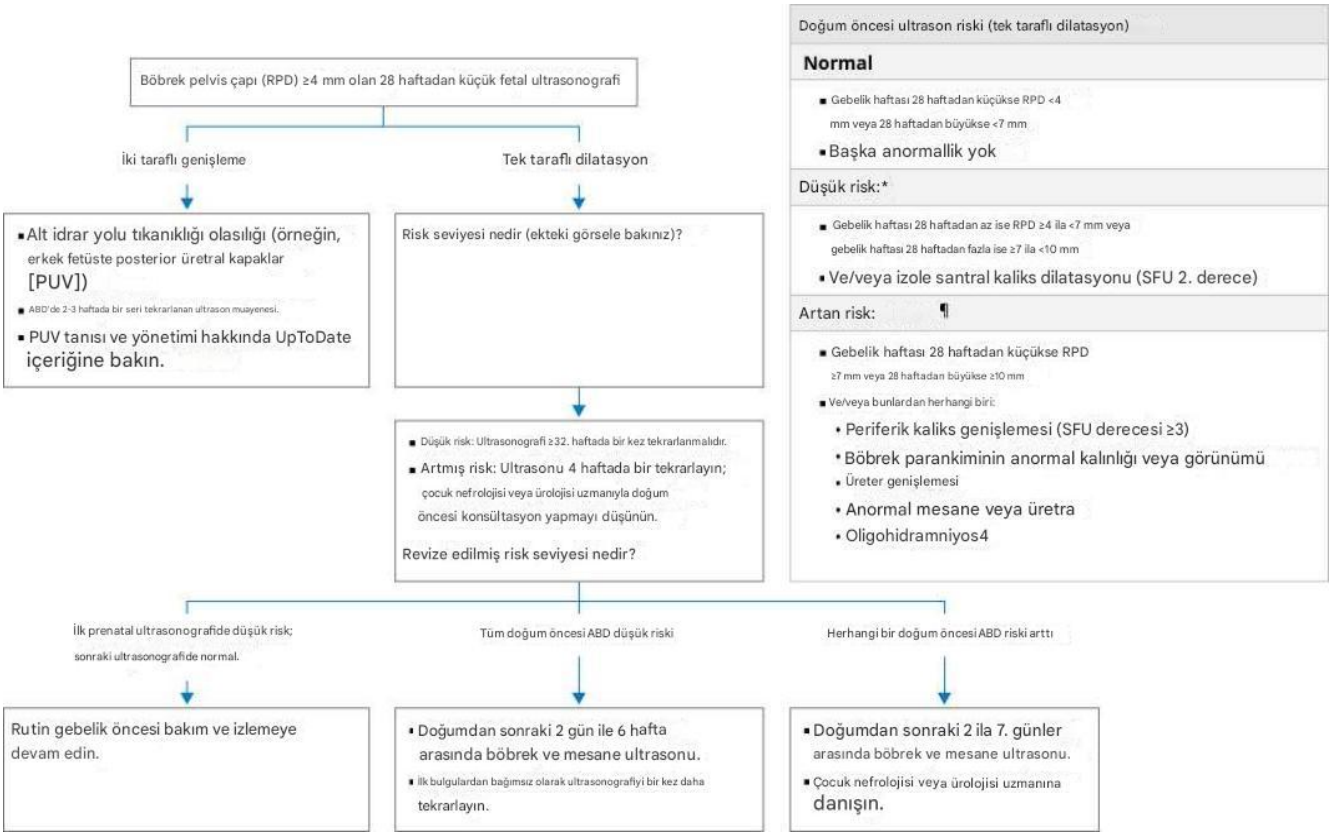
●**Down syndrome** – down's syndrome fetuses with mild hydronephrosis is often seen. Approximately 18 percent of fetuses with mild hydronephrosis seen in the second trimester down's syndrome, Down Syndrome, Non-in the fetus, this rate of 0% to 3% are among the [24,29,30]. Maternal serum cell-free deoxyribonucleic acid (DNA) testing made with prenatal screening of Down syndrome (trisomy 21), and many other valuable and pregnancy

to detect chromosomal abnormalities in the early period (approximately 10. week) can be made. If the screening result is positive, the patient is a diagnostic test such as chorionic villus sampling or amniocentesis for genetic counseling and definitive diagnosis may be offered.

●**Other anomalies** – other kidney/urinary tract non-fetal hydronephrosis is frequently associated with congenital anomalies [31]. Hydronephrosis, genetic and sporadic multiple malformation syndrome in more than 60 have been reported as part of a malformation syndrome [32,33]. Other fetal structural anomalies is detected if there is a risk of the fetus having a chromosomal abnormality [33]. In this case, a diagnostic test such as amniocentesis for fetal genetic diagnosis can provide a definitive mikrodizi.

PRENATAL ULTRASOUND KIDNEY AND UROLOGIC

Fetal hydronephrosis is usually detected during routine prenatal ultrasonography in the second trimester as the first (**algorithm 1**).



Algorithm 1

This algorithm is consistent with the American Academy of Pediatrics Clinical reports on serial prenatal ultrasound findings of fetal hydronephrosis and the risk of kidney disease based on an approach for monitoring outline.

GA: gestational age; PUV: posterior urethral caps; RPD: renal pelvis diameter; SFU: Society of fetal urology rating system; us: ultrasound; UTD: urinary tract dilation classification system.

* This low-risk UTD A1 properties is compatible with the class.

¶ This increased risk, the characteristics of UTD A2-A3 is compatible with the class.

Developed significant dilation in the second trimester amniotic fluid decrease and urinary tract, fetal kidney dysfunction suggests and has a poor prognosis.

*Reference:

Anthony Herndon CD, Otero HJ, D Hains and others. Perinatal urinary tract expansion: pre-natal/post-imaging, prophylactic antibiotics, and the following recommendations: a clinical report. Pediatrics 2025; 156:e2025071814.

Hydronephrosis severity of : the severity of hydronephrosis, prenatal ultrasonography renal pelvis diameter (RPD) are classified according to. The combination of RPD and other sonographic findings in the Table (**Table 1**) [1] as mentioned in congenital abnormalities of kidney and urinary tract (CAKUT) estimating the risk category determines.

During the ultrasound examination of the urinary bladder filling and discharge circular due to fetal kidney, ureters, and bladder may alter the appearance of. Therefore, more than one measurement should be taken during each examination [34].

The severity of hydronephrosis increases, increases the possibility of CAKUT [2,3,27,35]. A meta-analysis in the light of fetuses hidronefrozlu approximately 12%, middle fetus hidronefrozlu 45% of fetuses and severe hidronefrozlu in 88% of cakut was diagnosed in [2]. Average 20.6 months old until they are in a separate large case series which followed, mild

prenatal hidronefrozlu 10% of children, middle children hidronefrozlu 24% of children hidronefrozlu and severe in 63% of postnatal surgical intervention has been [27]. However, an indication for surgical intervention in children hidronefrozlu light half of vesicoureteral reflux (vur) has been; this is slightly higher than the rates reflect the current series also more surgery.

Prenatal unilateral hydronephrosis self-healing of the cases, approximately 25% to 75% is seen in [2,3,27,28] (above the 'Transient hydronephrosis see Section). Accordingly, it is more informative to estimate neonatal cakut repeated ultrasound in the third trimester.

Other ultrasound features: RPD in addition to that may help you in determining the cause and severity of hydronephrosis should be evaluated based on the following parameters. Risk factors that are associated with cakut between the correction to be more severe, bilateral hydronephrosis, urinary tract obstruction symptoms (enlarged ureter, bladder wall increased in thickness), and decreased amniotic fluid volume is located in the renal parenchyma thinning or ekogenit.

●Single-sided or double-sided involvement double – sided hydronephrosis (**Figure 2 and 3**), especially in dilated ureters, megacystis, bladder wall thickening and/or amniotic fluid decrease, together with suggests the possibility of lower urinary tract obstruction. The most likely cause in the male fetus, which is an important cause of obstructive uropathy and renal function impairment of posterior urethral covers (PUV) [36]. To identify the reliability of ultrasonography in prenatal PUV is limited. However, before the birth of a child specialist fetus is suspected of any PUV (e.g., urology, Maternal-Fetal Medicine) and be taken in consultation with the expert assessment must be made immediately after birth.

●Renal parenchyma – parenchyma my thinning and/or cortical cysts in the renal cortex is the sign of a developmental disorder or injury. Ekogenik renal cortex and/or small kidney, obstructive renal parenchymal that can occur depending on uropati hit or abnormal development (dysplasia) may show.

●Ureter – ureter dilated shoot can be compatible with. Additionally, a blockage can be caused by including the following:

•Congenital ureteropelvic junction (UPJ) obstruction (**Figure 4**).

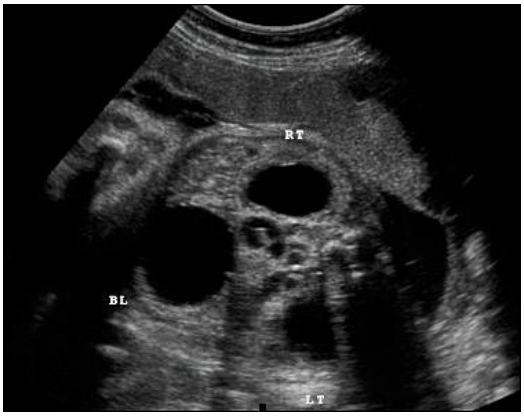
•Ectopic ureter, ureterocele (**image 5** and **Image 6**).

•Megaureter (**Figure 6**).

Bilateral ureteral dilation, PUV (male infants), urethral stenosis/atresia, bladder dysfunction or megacystis-microcolon-hipoperistalsis syndrome suggestive of lower urinary tract obstruction, such as it is.

●Bladder abnormalities bladder as the bladder and bladder wall thickening of the distal obstructive uropathy associated with trabekulasyon to the findings (e.g., in the male fetus PUV) [36]. Other symptoms that suggests proximal dilation of the urethra between PUV (keyhole Mark) (figure 5a) is located in. The expansion of the bladder, the sagittal diameter (measured in MM) plus 2 gestational age (weeks) can be defined as. For example, the expansion of the bladder to a fetus of gestational age 24 weeks, the sagittal diameter ≥26 mm (that is, 2 plus 24) is defined as [37,38].

Ureterosel can be seen in the bladder (**Figure 6**) and the settlements in the case of ectopic urethra may cause bladder outflow obstruction.



Picture 6: 30 4/7 weekly during pregnancy, the ultrasound image of the male fetus, severe hydronephrosis and bladder are seen in enlarged with thickened walls on both sides.

BL: bladder; RT: right kidney; LT: left kidney.

●Volume of amniotic fluid – amniotic fluid decrease, the amniotic fluid account for 90% of fetal urine production or excretion, which may be caused by a decrease in that cause blockage in kidney dysfunction. Amniotic fluid decrease, serious kidney disease that affects only one kidney or both kidneys of a consistent feature.

●*Acid or urine urino* : urino in the perirenal fascia is the mass of a liquid created by ekstravaze encapsulated in urine. UPJ obstruction or urinary tract obstruction is due to the increased pressure, such as PUV. Although rare, the presence of fetal urinoman 80% of the cases is associated with dysfunctional ipsilateral dysplastic kidney [39].

•Spontaneous rupture of renal calculi urinary tract obstruction and increased as a function formed as a result of pressure-induced urinary acid, rarely, rupture of the bladder due to bladder or neurogenic may occur.

PRENATAL MANAGEMENT

Objective : the majority of cases of Fetal hydronephrosis is not clinically significant. The purpose of prenatal administration, temporary and benign cases for unnecessary tests and parent/carer concerns, to avoid a serious underlying condition associated with cases at the same time to detect.

Based on the findings of the first ultrasound assessment and treatment: a treatment approach, the severity of hydronephrosis, single-sided or double-sided involvement, genitourinary anomalies, including the findings of the first ultrasound and amniotic fluid volume and the accompanying ekstrarenal depends on.

Prenatal monitoring of the expansion of the urinary tract by ultrasonography

Serial ultrasounds, prenatal: prenatal and postpartum assessments of the timing of renal pelvis diameter (RPD), and other sonographic findings depends on the risk category determined according to [1]. Fetuses at increased risk (for example, before 28 weeks RPD ≥7 mm or 28 weeks after the RPD ≥10 mm and/or other sonographic abnormalities (Table 1), a series of four-week prenatal ultrasound examination should have.

Specific ultrasound findings : results of *any of the following symptoms indicates a worse prognosis and requires individualized treatment*

●Two-sided hydronephrosis or only the affected kidney – regardless of the gestational age, two-sided hydronephrosis or RPD >4 mm and one kidney in a fetus with normal amniotic fluid volume, which is, to assess the progress of amniotic fluid volume dilatation and serial ultrasound examinations should be performed in two to three weeks after diagnosis. Track inspections, depends on the results of serial ultrasound.

●Other urological findings – Ureteral dilatation, thickened bladder and/or the presence of posterior urethral in males ureterocele caps (PUV) as sub-path are signs that may be

associated with urinary tract obstruction [36]. In some centers, predominantly in the amniotic fluid of fetal renal function to evaluate the measurement of fetal urine biochemical markers to use. However, these tests are clinically accurately predict postnatal renal function is not accurate enough. These tests are discussed separately.

●Amniotic fluid decrease – oligohidramniyo you (i.e., abnormally low amniotic fluid volume) management depends on the gestational age of the fetus, but this finding is associated with a worse prognosis. You oligohidramniyo management are discussed separately.

●Other non-genitourinary anomalies, and genetic syndromes for evaluation – signs of mild hydronephrosis, other congenital anomalies should require a detailed assessment of fetal anatomy to determine

In particular, if otherwise normal mild hydronephrosis is detected in a fetus in the second trimester, should be recommended for the assessment of Down syndrome.

Additional fetal anomalies are detected if advanced maternal age pregnancies, maternal serum screening tests or if abnormalities are found in the first or second trimester genetic counseling and testing should be provided.

In any of these cases, management decision-making, high-risk obstetrician fetal ultrasound specialist, a pediatric urologist, and the fetus of parents/care providers, a collaborative consisting of mother-fetus is shared with the team. Parents/care providers, first ultrasound evaluation of the significance of the findings and potential subsequent steps, if necessary, pre-natal and post-management description of options we offer. In particular, fetal/Neonatal prognosis is bad (for example, severe bilateral hydronephrosis, and inadequate amniotic fluid decrease with advanced renal parenchyma), some parents may want to terminate the pregnancy.

Postnatal ultrasound indications: post-natal ultrasound, depends on the risk category (Table 1) [1]' e should be made for any fetus :

●32 of pregnancy. detected after week or ongoing low-risk hydronephrosis (RPD ≥7 mm). Postnatal ultrasound for these babies, life 48. by the time six. between the first week should be done.

●During and after any period of pregnancy, even though improved, are at increased risk of hydronephrosis. Ultrasound examination for these babies after birth, ideally within 48 hours after you are discharged from the hospital birth in the hospital should be done. Also, well, men, we encourage you to meet with a child or a urologist.

The first ultrasound is normal after birth, even though it is cured after a second ultrasound to ensure that the correction should be done three to six months.

Other elements of assessment and management are discussed in a separate topic review postpartum.

Fetal surgery: Posterior urethral valve (PUV) and sometimes serious complications for the fetus, fetal intervention may be considered. The techniques and issues are discussed separately.

SUMMARY AND RECOMMENDATIONS

●Epidemiology – Fetal hydronephrosis (dilation of the renal pelvis and fetal), pregnancies 0.6 to 4.5%, as seen in prenatal ultrasonography is a common finding. 12 of pregnancy. from the week can be detected.

●Etiology

•Temporary – cases of Hydronephrosis approximately 25% to 75% are temporary. Especially in the second trimester renal pelvis diameter (RPD) are common in people hidronefrozlu light of 4 to 7 mm.

•Congenital anomalies of kidney and urinary tract (CAKUT) – CAKUT, fetal hydronephrosis (especially if there is moderate or severe) is one of the important reasons. The two most

common CAKUT ureteropelvic junction (UPJ) obstruction and vesicoureteral reflux (vur) is. These disorders may be associated with urinary tract infection and chronic kidney disease, because it should be diagnosed shortly after birth.

Lower urinary tract obstruction, especially in posterior urethral valves (PUV), while less common, neonatal survival, chronic kidney disease, and sometimes has important implications in terms of lung development.

•Associated anomalies – Hydronephrosis, in Down syndrome and other genetic syndromes is a common finding.

●Definition and classification of risk – the combination of ultrasonographic findings, and other RPD, to estimate the risk category determines the cakut (**Table 1**).

●Management Of Patients:

•Serial prenatal ultrasounds pre – natal and post-assessments of the timing depends on the risk category (Algorithm 1). Fetuses at increased risk (for example, before 28 weeks RPD ≥7 mm or 28 weeks after the RPD ≥10 mm and/or other ultrasonographic abnormalities) should have a four-week prenatal ultrasound examination series.

•Other effects of ultrasound findings – indicates a worse prognosis and individualized treatment requires any of the following symptoms:

-Severe hydronephrosis

-Two sided hydronephrosis

-Renal parenchymal abnormalities

-Symptoms of lower urinary tract obstruction (enlarged ureter, increased bladder wall thickness)

-Amniotic fluid decrease

-Unilateral renal agenesis

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