MINISTRY OF HEALTH OF UKRAINE
"Ukrainian Medical Stomatological Academy"

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on meeting the
department of Pediatric Surgery
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The Head of the department
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METHODICAL INSTRUCTIONS

FOR STUDENTS` SELF-WORK

WHILE PREPARING FOR PRACTICAL LESSONS

Educational discipline	Pediatric Surgery
module №3	Urgent Pediatric Surgery
Theme of the lesson	Gastrointestinal bleeding. Portal hypertension
Course	V
Faculty	foreign students preparation

POLTAVA 2020

1. The topic basis: the topic “Gastrointestinal bleeding. Portal hypertension” is very important for future doctors in their professional activity, positively influences the students in their attitude to the future profession, forms professional skills and experience as well as taking as a principle the knowledge of the subject learnt.

2. The aims of the training course:

1. To master the list of diseases which cause bleeding from upper and lower parts of the digestive tract.
2. To recognize the basic clinical manifestation of bleeding from upper and lower parts of the digestive tract.
3. To differentiate bleeding depending on the reason of its origin.
4. To interpret the auxiliary methods of research: USD, gastroduodenoscopy, X-ray, fibrocolonoscopy, rectoromanoscopy, laboratory and biochemical analyse, indices of haemodynamic.
5. To show stomach intubation, rectal examination, describe gastric content and emptying.
6. To identify the features of the course of separate diseases of the digestive system accompanied bleeding.
7. To analyse the cause-effect relationships of the origin of bleeding for separate patients, to ground and formulate a previous clinical diagnosis.
8. To offer the algorithm of the actions of the doctor at bleeding and tactics of treatment.
9. To interpret general principles of treatment of diseases which are accompanied by bleeding and to define indications for surgical treatment.

3. Basic knowledge, skills, habits necessary for studying the subject (interdisciplinary integration).

Names of previous disciplines	Obtained skills
1. Anatomy	Describe the structure of the abdominal organs. To assess the features of possible variants of the anatomical structure of the abdominal cavity organs.
2. Histology	Describe the structure of the abdominal organs. To assess the features of possible variants of the anatomical structure of the abdominal cavity organs.
3. biochemistry	Conduct laboratory methods of examining a child with bleeding from the digestive tract. To evaluate the data of clinical and biochemical analyzes: glucose in blood tests, urine; Protein in the blood serum, urine; Microelements, hepatic indices.
4. Physiology.	Describe the physiology of the gastrointestinal tract. Determine the features of the digestive system in children of different age groups.

5. 5. Pathological physiology	Describe the pathological changes in diseases of the gastrointestinal tract with bleeding. Identify the main points of etiology, pathogenesis in diseases of the digestive tract, which are complicated by bleeding.
6. Pathanatomy	Identify pathological changes in diseases of the gastrointestinal tract, which are complicated by bleeding. To determine the peculiarities of pathologic anatomical changes in bleeding from the digestive tract.
7. Operative surgery	To depict schematically the peculiarities of surgical interventions in children. To determine the features of the topographic anatomy of the abdominal cavity in children of different age groups; To justify operative dissections and interventions depending on the pathology and age of the child
8.Propedeutics of childhood diseases	Possess the technique of examining a child with bleeding from the digestive tract. To assess and demonstrate the knowledge of clinical and laboratory studies, the main symptoms of inflammatory diseases of the abdominal cavity at which bleeding from the digestive tract is observed.
9.Infectional diseases	Compare the symptoms of infectious diseases with which you need to conduct differential diagnosis of diseases of the abdominal cavity with bleeding from the digestive tract. Carry out a differential diagnosis of inflammatory diseases and surgical pathology of the abdominal cavity with bleeding from the digestive tract.

Thereotical questions for the lesson:

1. Name the basic clinical manifestations of bleeding from upper and lower parts of the digestive tract.
2. What are the main reasons of bleeding from the digestive system?
3. Give estimation of gastric content and emptying depending on the height of bleeding sources.
4. Give interpretation of general principles of bleeding treatment .
5. What are the features of controlling clinical course of bleeding from a digestive system? Define indications for surgical treatment.
6. Formulate indications for conservative and operative treatment of bleeding from a digestive system .

4. Maintenance of the subject:

GASTROINTESTINAL BLEEDING

Since many of the specific entities resulting in gastrointestinal bleeding are discussed elsewhere, only general comments will be made here. Gastrointestinal bleeding raises many questions of diagnosis and management, the resolution of which depends on the severity and duration of bleeding, associated symptoms, site of origin, and the age of the child. Of first importance are estimating the amount of bleeding and assessing its impact

on the child. Pulse rate and blood pressure must be measured at frequent intervals, rising pulse rate and falling blood pressure being signs of major blood loss. If major bleeding is suspected or possible, hospitalization may be indicated. Blood of an appropriate donor should be typed and cross-matched, and hemoglobin level and hematocrit determined to establish baseline levels. It may take several days for the hematocrit to stabilize after an acute hemorrhage. Placement of a catheter for measurement of central venous pressure may be useful to follow the need for blood replacement in intensive bleeding. Some intravenous line should be readily available. Diagnostic maneuvers are then necessary to locate the site of bleeding and identify its source. Passage of a nasogastric tube may help to establish that bleeding is occuring in stomach or duodenum, but observation may be confusing, and the procedure is arduous for the child. Arteriography has been used to locate severe bleeding of unexplained origin. The passage by rectum of bright red blood does not rule out a lesion of the upper gastrointestinal tract, but the passage of altered blood is more frequent in such lesions.

The age of the child has considerable diagnostic significance in gastrointestinal bleeding. Below 1 year of age intussusception, Meckel's diverticulum, volvulus and gangrene of the small bowel and hiatal hernia are important causes of intestinal hemorrhage (Fig. 11-9), but anal fissure, also more common under 1 year of age, is the most frequent cause of rectal bleeding in both infancy and childhood. In newborn infants hemorrhagic disease of the newborn and the "swallowed blood syndrome" may result in the passage of either bright red or altered blood. Polyps, esophageal varices and ulcerative colitis occur beyond 1 year of age. Localized lesions are responsible for about 50 per cent of cases of gastrointestinal bleeding, and systemic disturbances (sepsis, hemorrhagic diseases, allergy) are responsible for 10 to 20 per cent. About one third of cases of gastrointestinal bleeding are not etiologically identifiable, even after exhaustive investigation. About half of the localized lesions causing bleeding from the intestinal tract are in the anus, rectum or colon; about one third are in the small intestine, and only about 10 per cent are above the ligament of Treitz.

HEMATEMESIS. Hematemesis in childhood may result from blood swallowed during epistaxis or after a surgical or dental procedure. Such bleeding or accumulation of a large amount of blood in the gastrointestinal tract may be responsible for leukocytosis or fever. Blood-tinged vomitus and minor hematemesis are quite frequent after repeated vomiting of any origin. Otherwise, hematemesis suggests upper gastrointestinal lesions such as esophageal varices, erosive esophagitis secondary to gastroesophageal hiatal hernia, peptic ulcer, poisons and drugs such as aspirin, or, rarely, hemangioma or aberrant gastric mucosa in the esophagus.

RECTAL BLEEDING. Gross rectal bleeding may result from anal fissure, peptic ulcer, marginal ulcer associated with a Meckel's diverticulum or an intestinal duplication, gangrenous bowel secondary to volvulus, intussusception, gastrointestinal infection, ileitis, polyps, ulcerative colitis or amebiasis. Tarry stools may result from peptic ulcer, esophageal varices or bleeding diatheses. The association of vomiting, abdominal pain or shock with rectal bleeding suggests the possibility of a surgical emergency. Rectal bleeding with diarrhea suggests infectious enterocolitis, ulcerative colitis, amebiasis or intussusception. Minimal or microscopic bleeding with recurrent or chronic anemia suggests Meckel's diverticulum, amebiasis, ulcerative colitis, infectious enterocolitis, hiatal hernia, or polyps; in infants, *cow's milk allergy* is an occasional cause of such bleeding.

If the blood is bright red, diagnosis of the cause of rectal bleeding begins with careful examination of the anus for a fissure or "sentinel tag." If any one of these is found, and the bleeding is minor or consists chiefly of blood-streaked stools or blood spots on the paper used to wipe the anus after defecation, particularly if there is a history of hard, large or painful bowel movements, the blood may be presumed to be from an *anal fissure.*

Treatment consists in meticulous cleansing of the anus with soap and water after each defecation and in use of mineral oil to lubricate passage of stool until the lesion heals.

If an anal lesion is not found, thrombocytopenia, hypoprothrombinemia and other bleeding disorders should be ruled out; the rash associated with anaphylactoid purpura usually serves to identify it as a cause of intestinal bleeding. Sigmoidoscopic examination is useful to identify ulcerative colitis or polyps in the distal colon. Barium studies can serve to diagnose duodenal ulcer (because of the relative shortness and motility of the child's intestine, blood from a bleeding ulcer may be passed in virtually unaltered form), ileitis, ulcerative colitis, polyps, intussusception and volvulus; they are rarely informative for Meckel's diverticulum or duplication. Occasionally air contrast study of the colon is useful in the diagnosis of polyps. Pigmented lesions on the lips suggest polyposis of the small bowel as part of the *Peutz-Jeghers syndrome.* Exploratory laparotomy may be indicated in selected cases of severe bleeding without historical, physical, laboratory or roentgenographic findings to explain the bleeding.

PORTAL HYPERTENSION

ETIOLOGY**.** In children, in contrast to adults, obstruction of the portal vein exceeds cirrhosis as a cause of symptomatic portal hypertension. Thrombosis may be secondary to omphalitis in the neonatal period or to cannulation of the umbilical vein for exchange transfusion or other purposes. The development of collaterals in the connective tissue around the thrombosed portal vein is a response to obstruction rather than a malformation.

At the time of birth the ductus venosus branches off the left portal vein to enter the inferior vena cava. The umbilical vein is in continuity with both the ductus venosus and the left branch of the vein. Sepsis in the umbilical region may, therefore, spread along the umbilical vein to the left branch of the portal vein and then to the main portal vein. Umbilical infection may also spread to the hilus of the liver, producing portal vein obstruction there. Rarely, the normal obliterative process involving the umbilical vein and ductus venosus may extend to the portal vein. Anomalies of the portal venous system are rare: obstructive valves in the portal vein have been reported, as well as fistulas between the hepatic artery and portal vein and between the splenic artery and vien. Obstruction of the portal vein by neoplastic invasion is uncommon.

Portal hypertension is a common sequel to the cirrhosis which follows extrahepatic biliary obstruction or to postnecrotic hepatocellular disease. Because of the poor prognosis in these conditions, documentation of the presence of portal hypertenension is often of only academic interest.

Acute thrombosis of the portal vein is usually followed by spread to the mesenteric veins, with diarrhea, peritonitis and intestinal gangrene.

CLINICAL MANIFESTATIONS. In portal vein thrombosis, hepatic function is usually normal and presenting signs are those resulting from portal hypertension. Hematemesis is common and is often accompanied by the passage of bright red blood per rectum.

The opportunities for development of collateral communications between the portal and systemic circulations are many. They include: from vessels of the liver, esophagus and cardia of the stomach to diaphragmatic and intercostal veins; from vessels in the falciform ligament to umbilical veins; and from the surfaces of abdominal organs to vessels in the abdominal wall and in the retroperitoneal tissues; and from hemorrhoidal and portal veins to the azygos and bronchial ones. Because many of the collateral vessels enter the azygos vein, a widened mediastinum may be demonstrated by tomography.

Collateral circulation between portal and systemic vessels may produce dilated veins on the abdominal wall; such vessels radiating from the umbilical region are referred to as "caput Medusae." With development of extensive collateral circulation, venous hums may be audible over the xiphoid or umbilicus. The Cruveilhier-Baumgarten syndrome consists of cirrhosis and portal hypertension with a patent umbilical vein; this permits development of a particularly

prominent caput Medusae. The varicosities arising in the *esophagus and stomach* are the most important, owing to their tendency to bleed massively (see below).

Pancytopenia commonly results from hyper-splenism in the enlarged spleen (the Banti syndrome).

Ascites has generally not been considered to result from portal hypertension without cirrhosis, but it has been recognized in extrahepatic portal obstruction as a consequence of hypoproteinemia due to hemorrhage.

DIAGNOSIS. Radiographic examination of the esophagus for varices is the simplest and safest means of detecting collateral circulation secondary to portal hypertension. The mucosa of the normal esophagus appears on contrast study to exhibit long, narrow, evenly spaced lines; these may be displaced by varices, which appear as filling defects. Small, flexible fiberoptic esophagoscopes facilitate direct visualization; varices appear as blue, rounded projections beneath the mucosa.

Percutaneous measurement of intrasplenic pressure yields a good approximation of portal venous pressure. Portal venous pressure is normally about 7 mm Hg; presinusoidal pressure in the spleen is slightly above this value. Measurement of an elevated intrasplenic pressure provides a baseline for evaluating the effectiveness of shunting procedures.

Splenic venography should be carried out, but demonstration of collaterals alone does not establish the presence of portal hypertension, since the collaterals may effectively decompress the portal system. Demonstration of normal wedged hepatic vein pressure with elevated intrasplenic pressure establishes the site of the vascular obstruction as being in the extrahepatic portion of the portal vein. This procedure is not often necessary in children, since the distinction between intrahepatic and extrahepatic portal vein obstruction is readily made on clinical and biochemical grounds.

In a normal splenoportogram the opaque medium reaches the liver in 2 to 3 seconds, and the only vessels seen are the splenic and portal veins. The procedure is useful in investigating the cause of intestinal bleeding and essential before creating an anastomosis between portal and systemic circulations. It is also of value in the investigation of splenomegaly and in the delineation of liver masses. There is a 1 per cent risk of serious hemorrhage following splenoportography and this procedure should be undertaken only when definitive surgery is contemplated and scheduled.

Umbilical vein catheterization has been utilized as an alternate method of visualization of portal vein collaterals and determination of portal venous pressure. It can be carried out under local anesthesia and avoids the occasional splenic rupture which may follow percutaneous splenoportography.

Intrahepatic presinusoidal portal hypertension without elevation of wedged hepatic vein pressure has been encountered in schistosomiasis, in congenital hepatic fibrosis and in infiltrations of the portal tracts with primitive hematopoietic tissue, as in myeloproliferative disease, myeloid leukemia, Hodgkin's disease or sarcoid.

In cirrhosis with portal hypertension, wedged hepatic venous pressure is also elevated *(intrahepatic postsinusoidal portal hypertension).* Connective tissue proliferation in the portal tracts may lead to anastomoses between portal and hepatic vein radicles (internal Eck fistulas). In cirrhosis the main obstruction is to outflow from the hepatic vein, in which wedged pressures may remain elevated even after successful shunting procedures. In veno-occlusive disease, phlebitis of minute hepatic vein radicles also results in postsinusoidal intrahe-patic portal hypertension.

Obstruction to outflow of the main hepatic veins by cardiac failure or constrictive pericarditis results in postsinusoidal extrahepatic portal hypertension.

Primary portal hypertension has been described; it may be the result of excessive blood flow through an enlarged spleen.

TREATMENT. In infants and children with portal hypertension secondary to cirrhosis, the prognosis of the underlying liver disease is often so poor as to contraindicate surgical treatment. On the other hand, in patients with Wilson's disease who have massive esophageal bleeding,

amelioration of the portal hypertension by surgery may be indicated in view of expected improvement in the hepatic disease with penicillamine treatment.

Extrahepatic portal obstruction leading to symptomatic portal hypertension requires careful consideration of operative intervention. There are a number of reasons for conservatism in relation to a surgical approach. The surgical problems are more difficult in young children because of the small size of vessels available for portal to systemic vein anastomosis. If acute bleeding can be adequately controlled, the normal hepatic functions of these patients enable them to tolerate recurrent hemorrhages. A further basis for conservatism in respect to shunting procedures in children is the observation that variceal bleeding may cease spontaneously after repeated episodes. These considerations make the delay of surgical intervention until at least 4 to 6 years of age or older a reasonable course in most instances.

In most instances of portal hypertension secondary to portal vein thrombosis the vessel is unsuitable for anastomosis. In some patients proximal segments of the vessel seen on venography may be used for anastomosis to the inferior vena cava. The collateral branch most used for relief of portal hypertension in children is the splenic vein,| which is anastomosed to the left renal vein, with removal of the spleen. Unfortunately the splenic vein may be unsuitable owing to small size, extension of thrombosis, perisplenitis involving the vessels at the splenic pedicle, previous splenectomy, or thrombosis of an earlier splenorenal shunt. It should be emphasized that splenectomy without a shunt procedure should not be done for obstruction of the portal vein, because it is ineffective in relieving portal hypertension and sacrifices the vessel most useful in definitive shunting procedures. Alternative approaches must be devised when the splenic vein is unsuitable for anastomosis.

ESOPHAGEAL AND GASTRIC VARICES

The clinical features are those of gastrointestinal bleeding, with the added manifestations of portal hypertension. The bleeding may be slow, and productive of melena, anemia and increased red cell production, or it may occur as massive hematemesis; it may continue for days until controlled by treatment. Patients with intact hepatic function whose portal hypertension is caused by thrombosis of the portal vein tolerate hemorrhage much better than those with cirrhosis.

When the source of bleeding is in doubt, a water-soluble barium solution may be used even in the presence of bleeding to establish its site. The Seng-staken tube, for compression of varices, may be used diagnostically as well as therapeutically. In obscure situations splenic venography may be used to detect varices.

In emergency treatment of bleeding varices, transfusion of whole blood is indicated, preferably guided by measurement of blood volume. Intensive care may be needed to prevent brain damage from hemorrhagic shock and anoxia. Esophageal tamponade employs the Sengstaken tube, a three lumen tube connected to balloons placed in the esophagus and in the upper part of the stomach, and permitting aspiration of blood from the stomach; this will usually control bleeding. The gastric balloon is filled with radiopaque material in order to verify by radiography its proper placement. The balloons are distended to a pressure of approximately 30 mm Hg and traction is applied over a pulley. The technique has the disadvantages of being uncomfortable and giving rise at times to ulceration of the esophagus and pharynx.

5. Additional materials for the self-control

A. Clinical cases

Case 1. You are called to an 8 year-old child, whose state worsened suddenly. The patient has coffe grounds vomiting. The abdomen is soft, non-tender, during the palpation of abdomen enlargement of the spleen is found. The pulse is frequent of weak filling. Arterial pressure is 90/50 mm Hg. Hemoglobin is 86 g/l.

What diagnosis have you made?

What tactics have you developed?

Case 2. A 2 year-old child got pale; the pulse is low, and frequent. The abdomen is not distended, muscular tension, tenderness are localized in the umbilical region. Twice there was emptying with the admixture of clots of blood like the «jelly of currant».
What diagnosis have you made?

Case 3. A 5 year- old boy and his parent applied to a clinic. The boy often has with the last portion of excrement separate drops of scarlet blood, an feces is formed, constipation is marked.
What diagnosis have you made?

B. Tests

4. LITERATURE FOR STUDENTS

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