GALLBLADDER ULTRASOUND EVALUATION, ROLE IN THE DIFFERENTIAL DIAGNOSIS OF ASCITES FLUID OF PORTAL AND PARANEOPLASTIC HYPERTENSION ETIOLOGY

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Abstract: *Ultrasound investigation is an easy and readily accessible method to assess liver and the biliary pathology. Due to the continuously increasing performance of the method, any information provided by this method and its interpretation in the clinical context is useful. Descriptive study on a sample of 73 patients (47 patients with ascites fluid of sinusoidal portal hypertension etiology and 26 patients with neoplastic etiology ascites). We determined the gallbladder wall thickness and its size and the possible correlations between gallbladder size and ascites ethiology. The average thickness of the wall of gallbladder in the context of cirrhosis of the liver was 5.7 mm, while peritoneal average thickness was 2.5 mm.*

Cuvinte cheie: *ascită, colecist, hipertensiunea portală, neoplazie, ecografie*

Rezumat: *Ecografia reprezintă o metodă simplă şi uşor accesibilă pentru evaluarea patologiei hepatice şi a celei biliare. Performanţele metodei crescând continuu, este util de folosit orice informaţie furnizată de aceasta şi interpretarea ei în context clinic. În acest sens, am evaluat ecografic un număr de 47 de pacienţi cu lichid de ascită de cauză hipertensiv portală sinusoidală, respectiv 26 de pacienţi cu ascită de etiologie neoplazică. S-a determinat grosimea pereţilor colecistului precum şi dimensiunea acestuia în toate cazurile. Grosimea medie a pereţilor colecistului în context de ciroză hepatică a fost de 5,7 mm, în timp ce în carcinomatoza peritoneală grosimea medie a fost de 2,5 mm.*

INTRODUCTION

Ascites is the accumulation of fluid in the peritoneal cavity. Among the pathological conditions, the most commonly incriminated are liver cirrhosis and neoplastic carcinomatosis. Abdominal ultrasound has a high sensitivity in the evaluation of gallbladder, including the determination of its wall thickness. In essence, it is about the indirect interpretation, through ultrasound, of colloid osmotic pressure changes through edema and decreasing protein levels, consecutively for liver cirrhosis versus inflammation and effusion that occurs at the peritoneum level in case of neoplasia. The factors involved in the formation of ascites are multiple: disruption of renal metabolism of sodium and water, local circulatory changes, decreased albumin synthesis, altered fluid reabsorption from the peritoneal space.

The occurrence of ascites may have a sudden onset, suggesting the Budd-Chiari suprahepatic vein thrombosis or portal vein thrombosis. Also, ascites is formed in the case of liver function deterioration that can occur in infections, shock, high intake of alcohol or other toxic substances, as well as in the rapid lowering of blood albumin, a process encountered after the upper gastrointestinal bleeding, acute liver failure. Insidious onset is characteristic of heaptic cirrhosis, when there is a progressive increase of the abdomen, accompanied by shortness of breath, frequently preceded by irreducible abdominal flatulence.

Symptoms: abdominal distension, patients having bloated feeling; pain is different depending on the condition in which ascites is present - the hepatic cirrhosis, ascites occurrence is not accompanied by pain only in the case of spontaneous peritonitis or hepatocellular carcinoma; abdominal pain can be caused by liver distention or heart failure, thrombosis of the inferior vena cava, suprahepatic vein, portal vein or neoplastic invasion of the peritoneum; dyspeptic disorders - indigestion, loss of appetite, nausea, vomiting (gastroenteritis dissinergy consecutive to increased intra-abdominal pressure); pyrosis through gastroesophageal relfexus, consecutive to decreased lower esophageal sphincter tone; dyspnea (orthopnea) - by abnormal ventilatory mechanics (lifting diaphragm domes), hydrothorax.

Evolution of the disease: in ascites mechanism of formation, there occur factors such as hypoalbuminemia, portal hypertension, hydrosodium retention, lymph circulation disorder and increased permeability of subperitoneal capillaries.

Hypoalbuminemia is decreased serum albumin. Albumin is the major protein component of the plasma, cerebrospinal fluid and urine. In plasma, albumin is primarily responsible for maintaining oncotic pressure; It is also involved in the transportation of various compounds (free fatty acids, bilirubin, hormones, drugs and metal ions). Albumin is a global indicator of the nutritional status of the body, especially in the elderly with various chronic diseases. It was thus found that in the hospitalized elderly, hypoalbuminemia is an independent risk factor in terms of mortality.

Portal hypertension is involved in the production of ascites by two mechanisms: the increase in pressure in the splanchnic capillaries, the result being the passage of a greater amount of fluid from the arterial segment and the decrease of reabsorption in the venous segment of the capillaries and the formation of portal and renal anastomosis with the secondary hypertension in the renal vein territory, which will lead to increased tubular reabsorption of sodium and water.

Hydrosodium retention is one of the main factors in the formation of ascites. In vasculary decompensated cirrhosis, there is a constant vascular volume expansion, which is

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unevenly distributed: on one hand, there is an excessive amount of fluid in the abdomen and legs, and on the other hand, the effective volemia in certain sectors (kidney and brain) is diminished. Effective and diminished blood volume leads to a decrease of renal blood flow at kidney level, which by stimulating the juxtaglomerular system, leads to increased secretion of renin and secondary of aldosterone, favoring tubular reabsorption of sodium in exchange of hydrogen and potassium ions; at brain level, by volume-receptor excitation, an antidiuretic hormone hypersecretion occurs, which is only partially metabolized by the sick liver; the resulting hydrosaline retenion is a self-sustaining process, since the accumulation of sodium and water will be at the expense of the extravascular space – first, the abdominal cavity - while the effective amount of the kidney and the brain is still reduced.

Lymph circulation disorder – in liver cirrhosis, a part of the lymph flows into the peritoneal cavity.

The increase in subperitoneal capillary permeability causes the water and proteins to pass into the peritoneal cavity, for instance in the tuberculous peritonitis, cirrhosis of the liver, peritoneal metastases.

The main etiologies and their characteristics:

1. Liver diseases:

a) Cirrhosis, Budd-Chiari syndrome: the fluid is citrine, the proteins are <3 g / l, leukocytes are <200 mm^3, there are some rare RBCs.

b) Liver carcinoma: the liquid is citrine, hemorrhagic or chilous proteins are> 30 g / l, red blood cells and leukocytes are increased, there are malignant cells on the direct examination.

2. Carcinomatous pathologies:

a) Intraperitoneal malignancies (stomach, colon, uterus, ovaries) and secondary cancer of the peritoneum: the liquid is citrine, hemorrhagic or chilous, the proteins are> 30 g / l, red blood cells and leukocytes are increased, there are cell carcinoma.

3. Infectious pathologies:

a) Peritoneal tuberculosis: the liquid is citrine, hemorrhagic or chilous, the proteins are> 30 g / l, leukocytes> 1,000 / mm^3 with 70% lymphocytes, positive Lowenstein culture.

b) Suprainfection of the cirrhotic ascites: the liquid is cloudy or purulent, an exudate or transsudate may occur, a germ will be isolated by culture.

c) Bacterial peritonitis: secondary to cholecystitis, appendicitis, a diverticulitis, a perforation of the digestive tract has the above features.

In peritoneal carcinomatosis, the mechanisms present in turn some peculiarities: peritoneal carcinomatosis is actually the local dissemination of digestive, gynecological malignancies, or of another cause, with or without systemic metastases. Peritoneal carcinomatosis from digestive tumours has an incidence of 15-30% of patients. The most common cause of presentation is ascites. The main locations for metastases detection are the greater omentum, right subphrenic space, pouch of Douglas and the diaphragm area. Carcinomatosis evolution is driven mainly by physical factors rather than the biological ones and the way of occurrence depends on the type of neoplasia. Nonmucinous gastrointestinal adenocarcinomas have a tumour dependent penetrating mechanism up to serum level. Later, cancer cells adhere to the peritoneum.

Malignant neoplasms of the stomach, colon, pancreas and ovary, penetrating beyond the organ involved, can spread directly along the adjacent peritoneal viscera surfaces and may involve other structures. Neoplastic propagation along the peritoneal surface may include the bowel, at distance from the main tumour. Transverse mesocolon can serve as a major route for dissemination, from the stomach, colon and pancreas. Issues of peritoneal division are also brought into discussion, as well as the positioning of the ligaments and mesenteries that remain interconnected as propagation ways of neoplasia.

METHODS

Between 2010 and 2013, I assessed by ultrasound 47 patients with chronic cirrhotic ascites, regardless of its etiology, as well as a number of 26 patients with chronic neoplastic ascites. All patients have already the diagnosis confirmed. There were not included the patients with gallstones gallbladder. In patients with neoplastic etiology, on the first place, there were the patients with gastric cancer (10), followed by those with colon cancer (8), ovarian cancer (6) and two cases with clear cell renal cancer. Ultrasound evaluation was performed with a 3.5 MHz transducer, all patients being preprandially evaluated.

RESULTS

The average age of the patients with cirrhosis was 54 years old, with predominance of males (64%). The average size of gallbladder ranged normal. The average thickness of the wall of the gallbladder was 5.7 mm.

The mean age of the patients with neoplastic pathology was 60 years, in a relatively uniform relation between the two genders. The average thickness of the wall of the gallbladder was 2.5 mm. The average size of the gallbladder was increased in the upper abdominal malignancies, with a plus in the malignancies accompanied by cholestasis.

CONCLUSIONS

The average thickness of the gallbladder wall in the patients with liver cirrhosis as against those with neoplastic etiology ascites is almost double (5.7 mm versus 2.5 mm), being undoubtedly very useful in the clinical practice, ultrasound contributing a lot to this.

There is no correlation between the etiology of ascites and gallbladder size, except for the extrahepatic cholestases determined by the neoplasm.

Gallbladder wall thickness in the patients with liver cirrhosis evolves in parallel with increasing the amount of ascites fluid and therefore, with the stage of cirrhosis.

There is no precise correlation between the size of the portal vein and gallbladder wall thickness in the patients with cirrhosis, a probable explanation being the portal variability vascularisation.

REFERENCES

1. Wang T, Hwang S et CO. Gallbladder wall thickness in pacient with liver cirrhosis Journal of Gastroenterology 1997 Jun;12(6):445-449.
2. Brogna A, Bucceri AM et co. Ultrasound demonstration of gallbladder wall thickness,as a method to diferentiate cirrhotic ascites – Investigational radiology 1996 Feb 31 (2) 80-83.
3. Afshin Mohammadi, Mohammad Ghasemi-Rad, Mehdi Mohammadifar – Medica (Buchar) 2011 October;6(4):282-288.
4. Loreno M, Travali S, Buccer Am. Ultrasound study of gallbladder wall thickness and emptying in cirrhotic patient without gallstones.-Gastroenterology Practice 2009; 683040.
5. Kok T, van der Jagt EJ, Haagsma EB, Bijleveld CM, Jansen PL, Boeve WJ. The value of Doppler ultrasound in cirrhosis and portal hipertension.- Scand J Gastroenterology Suppl 1999;230:82-88.
6. Tiutiuca RC, Tiutiuca I. Profilul carcinomatozei peritoneale în adenocarcinoamele pancreatice. Jurnalul de Chirurgie, Iaşi 2009;5(4).