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Portal Vein Thrombosis Associated with Acute Cholecystitis In A Child

Çocukta Akut Kolesistit İlişkili Portal Ven Trombozu

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ÖZET

Portal ven trombozu (PVT), portal venin kısmen ya da tamamen tıkanması olarak tanımlanır. Akut kolesistit ile ilişkili PVT nadirdir ve literatürde sadece birkaç olgu sunumu bildirilmiştir. Akut kolesistit tedavisine yanıt alınamıyorsa; PVT gibi nadir ve ciddi komplikasyonlar gelişmiş olabilir. Bu olgu sunumunda çocuk hastada akut kolesistit ile ilişkili portal ven trombozu sunuyoruz.

Anahtar Kelimeler: Portal ven, tromboz, akut, kolesistit, çocuk

ABSTRACT

Portal vein thrombosis (PVT) is defined as a complete or partial occlusion of the portal vein due to a thrombus. The association of PVT with acute cholecystitis is rare, and only a few cases have been reported in the literature. If there is no response to treatment of acute cholecystitis; should be considered serious and rare complications such as PVT. In this case report, we present portal vein thrombosis associated with acute cholecystitis in children.

Key words: Portal vein, thrombosis, acute, cholecystitis, child

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INTRODUCTION

Portal vein thrombosis (PVT), is defined as a complete or partial occlusion of the portal vein due to a thrombus (1). Portal vein thrombosis is divided based on etiology into three main groups; cirrhosis-associated PVT, malignancy-associated thrombosis, and non-malignant non-cirrhotic PVT. This disease is most commonly secondary to cirrhosis or liver cancer (2). Non-malignant non-cirrhotic PVT is rare, it can be seen due to local inflammatory causes or acquired/ congenital causes of thrombophilia (3). Local inflammatory causes are cholecystitis, cholangitis, appendicitis, diverticulitis, hepatitis, and pancreatitis. The association of PVT with acute cholecystitis is rare and only a few cases have been reported in the literature (4). In this case report, we present a rare case of portal vein thrombosis associated with acute cholecystitis..

CASE REPORT

A previously healthy 12-years-old male patient presented to the pediatric emergency department with complaints of abdominal pain and vomiting for several hours. The pain was mostly localized in the right upper quadrant. He reported that he had vomit with bile for ten times in the past few hours. On physical examination, body temperature was 38°C, blood pressure was 100/75 mmHg, heart rate was 87 beats/min, respiratory rate was 19 breaths/min, and oxygen saturation was 100%. Abdominal examination showed diffuse tenderness, and Murphy's sign was positive. Other system examinations evaluated as normal.

Laboratory examination, revealed leukocytosis (22.000 /mm3) and an increase in C-reactive protein level (148 mg/L). Other complete blood count parameters were normal. Abdominal ultrasonography (USG) has reported that; "consistent with bile/sludge concentrated in the gallbladder lumen, the thickness of the gallbladder wall has increased. There is no dilatation in the intrahepatic bile ducts." We considered the diagnosis of acute cholecystitis. We started piperacillin-tazobactam and ursodeoxycholic acid treatment.

Abdomen computed tomography (CT) was performed because of the increase in CRP and sensitivity in abdominal examination despite treatment. Abdomen CT has a compatible appearance with acute cholecystitis, and there is a perfusion difference due to inflammation in the liver parenchyma around the gallbladder. A partial thrombus was observed in one of the right anterior branches of the portal vein, main portal vein, and right and left main branches open. (Figure 1). The hepatic venous system is normal. We thought that these findings were due to portal vein thrombosis related to acute cholecystitis.

Laboratory tests for the etiology of thrombosis were performed on our patient. D-Dimer values of 2,1 mg/L (0-0.55 mg/L) and fibrinogen 568 mg/L (200-400 mg/L) were

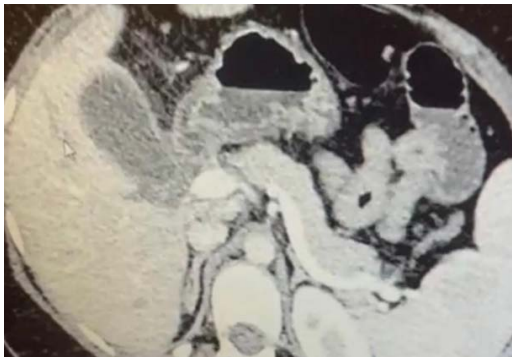

Figure 1. The arrow indicates a partial thrombus in one of the right anterior branches of the portal vein.

increased. Protein- S value decreased by 32% (60-130%). Protein-C was 83.8% (70-150%), Anti-Thrombin III was 97% (83-128%) and Homocysteine levels were normal [9.7 µmol/L (6 – 15µmol/L)]. Flow cytometric analysis was performed on the patient's erythrocytes, monocytes, and granulocytes. İn the paroxysmal nocturnal hemoglobinuria (PNH) clon; CD4, CD14, CD59, CD64, and FLAER deficiencies were not detected.

In the genetic analysis for thrombosis, Factor V (Leiden), Factor II Prothrombin, methyl tetrahydrofolate reductase (MTHFR) (677C>T), and Factor XIII (V34L) were normal. Heterozygous mutations were detected in Factor V(R2) and Plasminogen Activator Inhibitor 1 (4G/5G). In addition, we found a homozygous mutation in MTHFR (1298A>C).

Since the patient had acute PVT, enoxaparin treatment was started. Clinical findings and laboratory results improved after antibiotic and anticoagulant treatment. The patient became clinically stable and was discharged. One month later, the patient was re-evaluated. There was no abdominal tenderness, defense, or rebound. There was no pathological feature in the other system examination. The Portal vein diameter was within normal limits of 8 mm in color doppler ultrasonography of the portal vein. Hepatopetal flow was observed; thrombus was not observed in hepatic arteries and veins

DISCUSSION

In this case report, we presented a very rare case of PVT. This disease due to acute cholecystitis is rare and only a few cases have been reported in the literature (4). Abdominal pain, diarrhea, constipation, and vomiting may observed as a result of PVT. During admission, abdominal tenderness, bloating, fever, and decreased bowel sounds may be seen. If the diagnosis is delayed or the patient does not receive appropriate treatment; it can cause mesenteric ischemia, sepsis due to perforation, and septic shock (5). In a large series

of 23,796 autopsies in Sweden, the prevalence of PVT was reported as 1% when cirrhosis, tumor, and all other etiological factors were taken into account (6). Data on the frequency of non-cirrhotic PVT are less. It is known that it constitutes 5-10% of portal hypertension cases in Western societies and is responsible for one-third of portal hypertension cases in developing countries (7).

It has been determined that more than one risk factor causing thrombosis is present in at least one-third of PVT cases developing secondary to local inflammatory causes such as cholecystitis, hepatitis, cholangitis, diverticulitis, appendicitis, and pancreatitis (2). Choi et al. They retrospectively evaluated patients with transiently increased hepatic attenuation on CT scans. They reported 6 cases of PVT associated with acute cholecystitis. They did not observe any etiological factor associated with PVT in these patients. They speculated that the occurrence of PVT after acute cholecystitis may be due to inflammation or an infectious process involving the cystic vein (4). Among the systemic thrombogenic factors, Antithrombin III, protein C, and protein S deficiency are associated with a high risk of thrombosis, however, they are rarely seen in the etiology of PVT. Factor V Leiden (VFL) mutation, prothrombin mutations, and MTHFR homozygous mutations have a lower coagulation risk compared to anticoagulant protein deficiencies, but the incidence is higher in the etiology of PVT. Systemic acquired risk factors are primary myeloproliferative diseases, anti-phospholipid syndrome, PNH, cancers, inflammation, oral contraceptive drugs, pregnancy/postpartum period, and hyperhomocysteinemia due to vitamin deficiency (5). In this case, genetic mutations are minor risk factors for thrombosis. Therefore, we think that portal vein thrombosis developed in the patient as a complication of cholecystitis.

In the venous thromboembolism management guideline published by the American Society of Hematology in 2018, unfractionated heparin, low molecular weight heparin, fondaparinux, or vitamin K antagonists are mentioned as anticoagulant treatment options for the treatment of pediatric venous thromboembolism; therefore this anticoagulants can safely be used in children. This guide also sought to answer the question of whether anticoagulants should be used in PVT. Thrombosis is known to regress spontaneously in some patients with portal vein thrombosis. Guidelines support the use of anticoagulants in the case of occlusive and idiopathic PVT but not the use of anticoagulants in cases of portal hypertension due to chronic PVT (7). Enoxaparin was started in the patient. In the first month of treatment doppler USG was performed and complete regression of the thrombus was observed. Because of the patient genetic test results; we planned to continue anticoagulant treatment for at least three months.

CONCLUSION

Nonspecific symptoms may be observed during the PVT. If the diagnosis is delayed or the patient does not receive appropriate treatment; it can cause mesenteric ischemia, sepsis due to perforation, and septic shock. For his reason, if there is no adequate response to the treatment of acute cholecystitis, serious complications such as PVT should be considered.

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