Case Reports

Extremely rare complication after percutaneous treatment of renal hydatid cyst: Pancreatitis

Guven Tekbas MD[1], Aslan Bilici MD[1], Selvi Kelekci MD[2], Faysal Ekici MD[1], Hakan Onder MD[1], Murat Kemal Cigdem MD[3]

Department of Radiology[1], Department of Pediatrics[2], Department of Pediatric Surgery[3], Dicle University Medical School, 21280 Diyarbakir, Turkey

Corresponding Author: Dr. Guven Tekbas, Dicle Universitesi Tıp Fakültesi Radyoloji AD, 21280 Diyarbakir, Turkey. E-mail: guvenmakale@gmail.com

Abstract

Pancreatitis is necroinflammatory disorder that has higher prevalence of morbidity and mortality. Hereditary pancreatitis is the rare disorder of pancreas which is seen in children. Main symptoms are the same with sporadic pancreatitis. Exact differentiation with sporadic form is not possible who requires 2 patients within one generation or more than 2 patients in more than one generation for the diagnosis. In this paper we report a 13- year- old boy who had an attack of pancreatitis after the percutaneous treatment of giant cyst hydatid of kidney.

Keywords: hereditary pancreatitis; hydatid cyst; intervention

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Introduction

One of the necroinflammatory diseases of the pancreas is acute pancreatitis which has many etiologic causes such as common bile duct stones, alcohol, trauma, medications, toxins, and ductal defects [1]. Unlike in adults the causes of pancreatitis in children are more varied however the most frequent cause is systemic disorders [2]. Here in we report a case of acute pancreatitis in a pediatric patient after percutaneous hydatid cyst treatment of kidney with a rare etiology.

Case presentation

A 13-year-old boy presented with asymptomatic giant left renal cyst hydatid. Cyst had been found incidentally, which had been evaluated with ultrasonography before the operation of adenoid. He was referred to our center for percutaneous drainage (PT). Before the drainage he was evaluated with ultrasonography and giant, exophitic, Gharbi type II cyst which was originated from mid portion of the left kidney was seen (Figure 1). The other intraabdominal organs were normal. At physical examination no pathologic findings were obtained. Heart rate and breath and cardiac sounds were normal. Indirect mal test for hydatid cyst was positive in a titer of 1/1650. The other blood tests and lung X-ray were normal. He was prepared with sterile fashion and cyst was punctured under sedoanalgesia. The standard catheterization technique had been performed with 20% hypertonic sodium. 8F drainage catheter indwelled in cyst 850 cc

fluid was drained. No major side effect was observed, 24 hours later, he had mild epigastric pain and several vomiting attack. Ultrasonography was revealed that, mild edematous pancreas and intraabdominal fluid collection. CT was performed and edematous pancreas, peripancreatic fluid collection and collapsed cyst were seen (Figure 2). According to these radiological findings, blood test was performed for the pancreatitis. Amylase count was 3000 U/L, sedimentation rate was increased. In his past medical history, his brother (9 years old) and sister (15 years old) were treated in the hospital with the diagnosis of pancreatitis one and two times in last three years, respectively. His parents were relevant. After 15 days, amylase level decreased (150 U/L) and CT revealed that normal pancreas and peripancreatic tissue (Figure 3). The catheter was pulled out when the amount of drained fluid fell below 10 cc, and the patient was discharged. A month later, amylase was measured 100 U/L. But the patient did not have any complaint. At sonography, pancreas was normal and no intraabdominal fluid collection was detected. He looked healthy.

Two months later when this paper was written the patients' brother presented with abdominal pain and vomiting. In blood test, amylase was measured 1200 U/L and he was admitted to hospital and diagnosed acute pancreatitis and his treatment was continued.

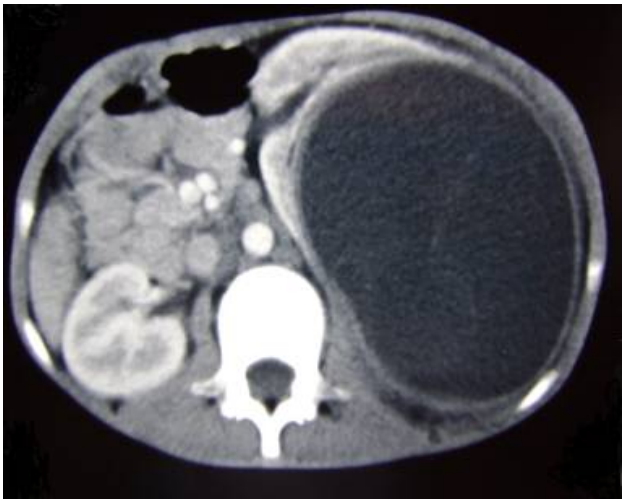

Figure 1. Abdominal computed tomography shows, large cyst compresses left kidney parenchyma and pushes kidney to the midline.

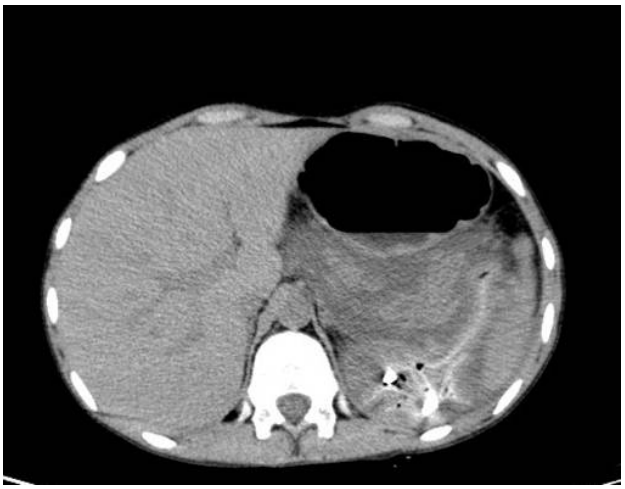

Figure 2. Abdominal non contrast computed tomography shows, edematous pancreas, peripancreatic fluid collection and collapsed cyst.

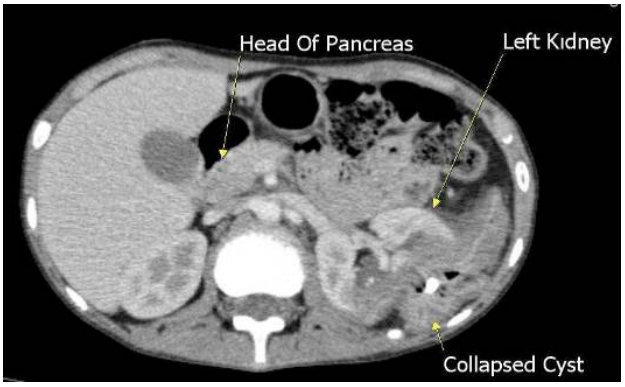

Figure 3. Post contrast abdominal computed tomography shows, normal pancreas and peripancreatic

Discussion

Acute pancreatitis is a rare life-threatening inflammatory disorder whose incidence is of approximately 1 in 50,000 in USA [3, 4].The causes include biliary tract disease, medications, trauma, systemic, viral, metabolic, and genetic syndromes [3] and moreover it can also be triggered by environmental risk factors that include infections, trauma, smoking, and alcohol [5].

The complications are such as urticaria, pain, fever, anaphylaxis, fistulas, suppurations, fever, and infection, may be seen after percutaneous treatment of hydatid cyst treatment [6, 7]. As a rare complication in some cases, pancreatitis had been reported due to hydatid liver disease [8]. However to the best of our knowledge

reported case is the first pancreatic attack after the renal hydatid cyst treatment.

The clinical presentation of acute pancreatitis is similar to adulthood which is generally presented with sudden onset of abdominal pain, nausea and vomiting. The AP is associated with a rise of pancreatic digestive enzymes in the serum or urine with or without radiographic changes in the pancreas [2, 4]. After the attack, both exocrine and endocrine functions of the gland may recover to normal [2, 9]. In reported cases' symptoms started with mild abdominal pain and vomiting. But abdominal pain level severity disproportion with serum amylase level. After the treatment, radiologic findings were totally normal but laboratory findings were almost normal because of amylase seen 2-fold over the normal upper limit; however clinical signs were normal.

Hereditary pancreatitis is an autosomal dominant genetic disorder described an accumulation of patients with pancreatitis in one family which requires 2 patients within one generation or more than 2 patients in more than one generation [9, 10]. The clinical and pathological appearance of both acute and chronic pancreatitis in these patients is same as sporadic forms of acute and chronic pancreatitis. Mutations in the trypsinogen gene are associated with both acute and chronic pancreatitis [5] the normal product of the gene mutated in hereditary pancreatitis is likely to play a role in the protective mechanism preventing acute and chronic pancreatitis. Genetic abnormalities of the defense mechanisms whose main secretes are secretory trypsin inhibitor (SPINK1), and chymotrypsinogen C (CTRC) that result in dysfunction of these can trigger the zymogen activation cascade and lead to pancreatic autodigestion[11]. Progression to chronic pancreatitis is seen in half of the patients with acute pancreatitis and the risk of pancreatic cancer is significantly increased [10].

In conclusion, hereditary pancreatitis is a rare disease of childhood period. It may present without any risk factors. However, invasive treatment modalities such as surgery and percutaneous interventions may facilitate its occurrence. Thus, interventionalist and surgeons should be careful when managing those children who are prone to develop a pancreatitis attack. Additionally, a minimal abdominal pain after an invasive procedure should also bring mind the attack if the diagnosis is previously known.

References

1. Park AJ, Latif SU, Ahmad MU, Bultron G, Orabi AI, Bhandari V et al. A Comparison of Presentation and Management Trends in Acute Pancreatitis Between Infants/Toddlers and Older Children. J Pediatr Gastroenterol Nutr. 2010 May 12. [Epub ahead of print]PMID: 20479687
2. Lowe ME. Pancreatitis in childhood.Curr Gastroenterol Rep. 2004;6:240-246
3. Bultron G, Latif U, Park A, Phatak U, Pashankar D, Husain SZ. Acute pancreatitis in a child with celiac disease.J Pediatr Gastroenterol Nutr. 2009;49:137-138
4. Clifton MS, Pelayo JC, Cortes RA, Grethel EJ, Wagner AJ, Lee H et al. Surgical treatment of childhood recurrent pancreatitis. J Pediatr Surg. 2007 ;42:1203-1207
5. Kandula L, Whitcomb DC, Lowe ME. Genetic issues in pediatric pancreatitis. Curr Gastroenterol Rep. 2006; 8:248-253
6. Khuroo MS, Zargar SA, Mahajan R. Echinococcus granulosus cysts in the liver: management with percutaneous drainage. Radiology1991 ;180:141 -145
7. Paksoy Y, Odev K, Sahin M, Arslan A, Koç O. Percutaneous treatment of liver hydatid cysts: comparison of direct injection of albendazole and hypertonic saline solution. AJR Am J Roentgenol. 2005 Sep;185(3):727-734.
8. Zeytunlu M, Çöker A, Yüzer Y, Ersöz G, Aydın A, Tekesin O, Özütemîz Ö, Batur Y. Hydatid acute pancreatitis. Turk J Gastroenterol 2004; 15 (4): 229-232
9. Keim V. Role of genetic disorders in acute recurrent pancreatitis. World J Gastroenterol. 2008; 21:1011-1015.
10. Whitcomb DC, Somogyi L. Lessons from hereditary pancreatitis. Croat Med J. 2001;42:484-487
11. Sunnapwar A, Prasad SR, Menias CO, Shanbhogue AK, Katre R, Raut A. Nonalcoholic, nonbiliary pancreatitis: cross-sectional imaging spectrum. AJR Am J Roentgenol. 2010; 195:67-75.