ADPKD with CAPD related pancreatitis

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Abstract

Pancreatitis is a very rare adverse effect of Continuous Ambulatory Peritoneal Dialysis (CAPD), especially in individuals complicated with autosomal dominant polycystic kidney disease (ADPKD). We reported a case of recurrent acute pancreatitis in a 63-year-old male with ADPKD receiving CAPD and review the literature with regards to CAPD induces pancreatitis. We also highlighting the fact that those with ADPKD undergoing CAPD may have a higher risk of developing pancreatitis. High degree of suspicion is warranted on the part of physicians to diagnose CAPD induce pancreatitis in patients presenting with gastrointestinal symptoms. Once CAPD is suspected as the causative agent then consider to switch into hemodialysis rather than restart CAPD.

Keywords

Acute pancreatitis, Continuous Ambulatory Peritoneal Dialysis (CAPD), autosomal dominant polycystic kidney disease (ADPKD).

Introduction

CAPD is one of the most common choices for renal replacement therapy throughout the world. The leading complication of peritoneal dialysis is peritonitis [1]. Pancreatitis is a very rare complication and was first published in a case report in 1985[2]. Concerns about CAPD-induced acute pancreatitis have recently been raised [3, 4].We report a case of ADPKD with recurrent acute pancreatitis associated with CAPD therapy.

Case Report

A 63-year-old male patient presented to the emergency department with sudden attack of epigastric pain. He had been undergoing CAPD since 3 years ago, because of autosomal dominant polycystic kidney disease (ADPKD), and had suffered three outbreaks of acute pancreatitis in six months. Abdominal

ultrasound, computer tomography (CT) and magnetic resonance cholangiopancreatography (MRCP) were performed during previous episodes and no structural abnormality was found.

On examination the patient had normal vital signs. The abdominal examination was significant for mildly distended abdomen with severe epigastric and left upper quadrant tenderness. There was no rigidity and muscle guarding and bowel sound were hypoactive. There were no palpable abdominal mass. The patient had no fever and jaundice. The serum amylase was elevated to 737 IU/L (Reference range: 36-128), serum lipase to1188 IU/L (Reference range: 22-51). The liver function tests and lipid profile were all normal. The serum calcium level was 10.3 mg/dL (Reference range: 8.9-10.3). Other laboratory examination including peritoneal dialysate analysis and it revealed no evidence of peritonitis, hypercalcemia, hyperlipidemia or autoimmune pancreatitis. Abdominal ultrasound, magnetic resonance cholangiopancreatography (MRCP) and triphase dynamic computer tomography of the abdomen demonstrated no dilatation of intrahepatic ducts or common bile duct, no stones, no pancreatic enlargement or necrosis, and no fluid accumulation. Drug history has also been reviewed and there were no changes of his regular medication within the recent 2 years. Because no causal factor for pancreatitis was found after the initial work-up, acute pancreatitis induced by CAPD was suspected. The patient was treated with conservative management and he switch to hemodialysis by our advice. He discharged after his symptoms were relief and normalized of serum pancreatic enzymes. At a 12-months follow-up visit, the patient remained healthy. We advised our patient to avoid CAPD and hence there has been no recurrent to date.

Discussion

Acute pancreatitis is an acute inflammatory of the pancreas which is usually associated with acute upper abdominal pain and elevated levels of pancreatic enzymes in the blood. It can range from almost asymptomatic to severe consequences and even mortality. Differential the possible cause of acute pancreatitis is extremely important. Published literatures conclude that patients on peritoneal dialysis (PD) are at a higher risk for acute pancreatitis as compared to patients receiving hemodialysis (HD) [2, 3, 5]. The exact reason for CAPD induce acute pancreatitis is still not totally clear. A few mechanisms had been proposed. A possible mechanism is the peritoneal dialysate diffused through the peritoneum of the lesser sac to the anterior surface of the pancreas, causing chemical irritation [3]. Another putative potential mechanism

is the calcium in the peritoneal dialysate and it could diffuse through the peritoneum causing “local hypercalcemia” at the pancreas even though the systemic calcium levels may still remain normal.

Elevated intraabdominal pressure may be another potential mechanism that makes the pancreas more susceptible to parenchymal damage and it also impaired the microvascularisation leading hypoxaemia which may induce premature activation of proteolytic enzymes, thereby provoking acute pancreatitis. The intraabdominal pressure may be even higher in patients with ADPKD and receive CAPD. This case highlights a number of interesting points. Co-occurrence of ADPKD and CAPD related acute pancreatitis is rarely reported, to our knowledge, this has never been previously described.

The management of CAPD induced pancreatitis consists of standard treatment of pancreatitis with prompt discontinuation

In summary, we document a rare case of 63-year-old asian male with ADPKD who had recurrent acute pancreatitis associated with CAPD, which was successfully treated by supportive treatment and switching into hemodialysis.

Conclusion

Acute pancreatitis is a very rare but potentially serious adverse effect of CAPD, especially in patients complicated with ADPKD. Clinicians should be aware that acute abdominal pain and/or elevation of pancreatic enzymes in CAPD patients may be caused by acute pancreatitis. It is important to consider CAPD as a possible etiology for acute pancreatitis. Earlier diagnosis and treatment may be helpful in such patients. Once CAPD is suspected as the causative agent then it should be consider switching into hemodialysis.

Disclosures

All authors declare that there are no conflicts of interest

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