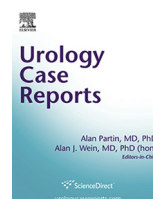


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Abdominal Compartment Syndrome and Necrotizing Pancreatitis Following Extracorporeal Shock Wave Lithotripsy

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ABSTRACT

Extracorporeal shock wave lithotripsy (ESWL) is a common procedure in the treatment of renal calculi. There have been major complications reported with ESWL such as acute pancreatitis, bower perforation, venous thrombosis, and biliary obstruction. There are few reports in the literature of necrotizing pancreatitis secondary to ESWL. We have a case report of a 29-year-old female that developed an abdominal compartment syndrome with an acute necrotizing pancreatitis hours after extracorporeal shock wave lithotripsy.

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Introduction

Extracorporeal shock wave lithotripsy (ESWL) is a widely accepted urological procedure performed for renal calculi. Nonetheless, a number of major complications have been reported in the literature. We report a rare case of a young female who developed acute necrotizing pancreatitis with an abdominal compartment syndrome following ESWL for a right renal pelvic calculus.

Case presentation

A 29-year-old female was known to have a 1.1 cm right ureteropelvic junction stone with right hydronephrosis, which she had undergone extracorporeal shock wave lithotripsy (ESWL). The procedure had been uneventful. Six hours later she presented to our emergency department complaining of diffuse abdominal pain radiating to the back. Other symptoms included nausea, non-bloody emesis, and chills. The patient had no previous history of biliary stones, alcohol consumption or abdominal surgeries.

On physical examination, the abdomen was mildly distended with mild guarding and tenderness in the epigastrium and periumbilical region. On biochemical evaluation, she had an elevated white blood cells count (18.69 K/mm^3), serum amylase of 3002 U/L

(normal range: $25\text{--}115 \text{ U/L}$) and serum lipase of $33,275 \text{ U/L}$ (normal range $73\text{--}373$). An abdominal computed tomography (CT) ([Fig. 1](#)) showed a moderate amount of abdominal ascites with fluid and edema around the pancreas without any evidence of necrosis consistent with acute pancreatitis.

She was admitted to the medical ICU for medical management. However, over the next 48 h she decompensated becoming hypotensive and oliguric. She required liters of crystalloid and intermittent pressor support. She deteriorated and was emergently intubated due to respiratory failure. She had developed significant abdominal distention, tenderness with peritonitis along with elevated peak airway pressures. She was found to have an abdominal compartment syndrome with peritonitis and was emergently taken to the operating room (OR) for a decompressive laparotomy. She was found to have necrotizing pancreatitis along with liters of ascites. An abdominal washout was performed, debridement, and abdomen was left open with placement of an abdominal wound vac. She had a complicated hospital course which required her to have multiple debridement and washouts. Her length of stay was 62 days that required a total of 12 operations. She was ultimately discharged with a closed abdomen to a rehabilitation center in stable condition with a good prognosis.

Discussion

The use of extracorporeal shock wave lithotripsy was introduced into clinical practice in the 1980s. Since then it has become accepted

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Figure 1. CT scan with extensive fat stranding and edema around the pancreas suggestive of acute pancreatitis.

as an effective, non-invasive treatment for a wide range of upper urinary tract stones.¹ Serious complications of ESWL include: pulmonary contusion, cardiac arrhythmia, new onset diabetes mellitus, gastric erosions, aortic aneurysmal rupture, portal and iliac vein thrombosis, biliary obstruction, colonic and splenic injury, bowel perforation and acute pancreatitis.

Few isolated reports of acute pancreatitis following ESWL exist in literature, and the exact mechanism of pancreatitis is unknown.¹ Several studies have evaluated the serum markers of pancreases after ESWL for renal stones, and the results are mixed. Thiel et al demonstrated no significant change in amylase and lipase levels postoperatively and 30 days after ESWL for renal stones in 16 cases.² Gunnar et al included c-peptide, insulin, and glucagon in addition to amylase and lipase and reported no significant acute changes in six patients with upper urinary tract stones.³

The risk factors for acute pancreatitis are gallstones, alcohol, smoking, drugs, hypertriglyceridemia and hypercalcemia. The sequence of development of acute pancreatitis following ESWL, in the absence of any of the predisposing factors and prior abdomen surgery, supports a causal relationship in our patient. There are multiple hypotheses as to the cause of pancreatitis in these patients. One hypothesis is the inadvertent fragmentation of gallstones or common bile duct stones causing pancreatic duct obstruction leading to acute pancreatitis. Another hypothesis is shock waves cause small hematoma and microvascular damage to the pancreas. This soft-tissue injury has been reported as a direct effect of the sonic pulsation or as a result of the process of cavitation, in which rapid expansion and collapse of tiny air bubble induced by the shock wave injure the adjacent tissue.⁴

It has also been suggested that post-procedure adhesions between the pancreas and surrounding tissue as a cause for mechanical injury resulting in pancreatitis following ESWL.² There has also been a positive correlation between the number of shock waves and rise in pancreatic enzymes following ESWL.^{2–4}

This case is particular interesting in that this patient developed abdominal compartment syndrome (ACS) secondary to severe, acute pancreatitis. The severity of the inflammatory process lead to significant “third spaced” fluid sequestration into the bowel walls and retroperitoneum. The resultant “shock bowel” and paralytic ileus increased intra-abdominal pressures leading to ACS. The development of ACS ultimately caused her to go into respiratory distress and multiple organ system failure.

The diagnosis of ACS is made with diagnostic measurement of intra-abdominal pressure (IAP) and clinical findings. Measuring the bladder pressure is an easy way to monitor IAP. A foley catheter is then inserted into the patient with a distillation of 25 mL of sterile water. The catheter is clamped. The patient is positioned supine with the transducer placed at the level of the mid-axillary line. It should be measured at end-expiration with no abdominal contractions present. ACS is defined as IAP >20 mm Hg with new organ dysfunction or failure. Treatment for patients with explicit ACS requires surgical intervention with a decompressive laparotomy.⁵

Conclusion

The risk factors for acute pancreatitis are gallstones, alcohol, smoking, drugs, hypertriglyceridemia and hypercalcemia. The sequence of development of acute pancreatitis following ESWL, in the absence of any of the predisposing factors and prior abdomen surgery, supports a causal relationship in our patient. When complicated by extensive necrosis and formation of fluid collection around the pancreas morbidity is increased, as in our case. It remains unclear as to why she developed a necrotizing pancreatitis. Abdominal compartment syndrome should be considered in any critically ill patient with abdominal distention. These patients should have their intra-abdominal pressures monitored with bladder pressures. Further work needs to be done to better understand the relationship with ESWL and pancreatitis and urologists should be aware of the complication.

Conflict of interest

There is no conflict of interest.

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