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CASE REPORT

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Ureteric obstruction as an unrecognised cause of acute pancreatitis in the emergency setting

Mariolyn Raj, MBBS

Spencer Murray, MBBS

Department of Surgery, Royal Hobart Hospital, Tasmania, Australia

ABSTRACT

Objectives

Acute severe pancreatitis has a high mortality rate (10 to 30 %) and the aetiology of the pancreatitis itself influences the severity assessment and ultimately the treatment.¹ Up to 20% of cases of acute pancreatitis are said to be due to unusual causes such as viral infection, trauma and drugs. However to date, there have been no reports in the literature of ureteric obstruction and infection causing acute pancreatitis.

Methods

We report on a case of a 77-year-old male who presented to the Emergency Department with acute pancreatitis occurring in the setting of an obstructed and infected right urinary system.

Results

We outline the pathway to diagnosis and management in this patient and highlight the difficulties associated with reaching an accurate diagnosis in the acute setting.

Conclusions

We report on this unusual case of acute pancreatitis in the emergency setting. By illustrating this case we aim to promote awareness of and encourage clinicians to consider ureteric obstruction as a possible cause of acute pancreatitis, in their workup of patients in the emergency department.

Keywords: *emergency department; pancreatitis; pyelonephritis; ureteric calculus; management*

Objectives

A 77 year old man presented to the Emergency department with a 24-hour history of acute right-sided abdominal pain radiating to the back, nausea and vomiting. On physical examination he had central and right sided abdominal tenderness and was hypovolemic.

Laboratory results showed elevated lipase of 1058 IU/L and amylase of 351 IU/L and impaired renal function with (Creatinine 159 μ mol/L, Urea 11.1mmol/L, eGFR 37mL/min). The WCC was 4/nL and liver function tests, serum calcium and lipids were normal. Urine output was adequate and dipstick urine was positive for ketones and trace blood.

A diagnosis of severe acute pancreatitis was made based on the clinical presentation and elevated pancreatic enzyme level. Ransom score was 5. The aetiology of the pancreatitis was however unclear. Routine initial investigations showed a normal chest X-ray and hepatobilliary ultrasound. There was no history of recent alcohol use or trauma.

Within 6 hours the patient became septic with a temperature of 38°C, hypotensive with blood pressure of 80/60 mmHg and had a respiratory arrest.

An urgent computer tomography (CT) scan was performed to further investigate for possible causes of the patient's pancreatitis. Due to his renal impairment no oral contrast was used. CT scan revealed a 6x9mm calculus at the right pelvoureteric junction with moderate right hydronephrosis and perinephric stranding, consistent with right-sided ureteric obstruction (Figure 1). Blood and urine cultures returned positive for growth of *E.coli*. The patient's WCC rose to 19/nL.

Figure 1: 6x9mm calculus at the right pelvoureteric junction with moderate hydronephrosis and perinephric stranding of right kidney.

A diagnosis of ureteric obstruction with associated pyelonephritis was made. His serum pancreatic enzyme level remained elevated. Immediate decompression of the obstructed urinary system via an 8.5Fr percutaneous nephrostomy treated the sepsis. Subsequently there was rapid improvement in the pancreatitis. Serum amylase and lipase levels returned to normal within 24 hours.

Three weeks later the patient underwent shock-wave lithotripsy as definitive treatment for his right obstructing ureteric calculus.

Discussion

Acute pancreatitis is a common presentation to emergency departments in Australia with an annual incidence of 5.4 to 80 per 100 000.² Gallstones and alcohol use account for 70% to 85% of cases: other recognised causes include drugs, viral infections, tumours, hyperlipidemia, hypercalcemia, trauma, iatrogenic injury and pancreatic duct anomalies (Table 1).³

Table 1: Causes of Acute Pancreatitis³

CAUSES
Biliary tract disease (approximately 40%):
Alcohol (approximately 35%):
Post-ERCP (approximately 4%)
Trauma (approximately 1.5%)
Drugs (approximately 2%)
Infection (<1%) Viral causes: mumps, EBV, coxsackievirus, echovirus, varicella-zoster, measles. Bacterial: <i>Mycoplasma pneumoniae</i> , <i>Salmonella</i> , <i>Campylobacter</i> , <i>Mycobacterium tuberculosis</i> .
Hypercalcemia (<1%)
Developmental abnormalities of the pancreas (<1%)
Hypertriglyceridemia (<1%)
Tumor (<1%)
Toxins (<1%)
Postoperative (<1%)
Vascular abnormalities (<1%)
Unknown (idiopathic).

Severe pancreatitis has a high mortality rate (10 to 30 %) and prompt recognition of the cause in the acute setting assists in facilitating appropriate treatment.¹

As illustrated, in this patient, we adopted a rational stepwise approach to investigating the likely precipitating factors causing his pancreatitis.

Initially, the patient's symptoms and significantly elevated lipase level (greater than three times normal in this case), which is highly sensitive (100%) and specific (99%) for pancreatitis, confirmed the existence of acute pancreatitis.^{4,7, 8,9}

Subsequently we investigated for the common recognised causes of acute pancreatitis; biliary disease, alcohol or trauma via hepatobiliary ultrasound, serum LFT's and clinical history taking, without any success.

The abdominal CT scan was a useful second like test, which aided the diagnostic workup, by screening for other possible intra-abdominal causes of the acute pancreatitis, permitting identification of the obstructing right ureteric calculus.¹

One may question, like we did initially, if the finding of ureteric obstruction was merely co-incidental and not the true cause for the patient's pancreatitis.

We conducted a literature review at the time and found several reports in the literature, which outlined the occurrence of pancreatic inflammation associated with renal hydronephrosis and pyelonephritis.^{5,6}

We felt it was biologically plausible that in this patient's situation extravasation of infected urine from the obstructed right ureter into the surrounding tissues could occur due to increased pressure in the ureter proximal to the obstructing calculus. Subsequent irritation and inflammation of the uncinate process, which lies anatomically close to the right upper ureter could thereby give, rise to the clinical features of acute pancreatitis that we were observing.

In deciding to institute treatment for the ureteric obstruction we observed several effects which confirmed our suspicions that ureteric obstruction was the likely precipitating factor causing the pancreatitis in this patient.

Firstly there was rapid resolution of the patient's symptoms within 24 hours of urinary decompression. Both his renal function and serum pancreatic enzyme levels normalised within 24 hours. The rapidity of the resolution of the acute pancreatitis immediately following treatment lends support to our hypothesis regarding the cause.

Secondly, a retrospective history obtained from the patient a few days later, revealed that his initial symptoms had been acute right flank pain associated with transient dysuria before the onset of the severe central and right sided abdominal pain. In view of this history and on reviewing his clinical findings at admission: renal impairment and presence of blood in the urine, one can surmise that the ureteric obstruction was most likely present initially, but was not recognised till much later.

Moreover we thoroughly investigated for other likely causes of acute pancreatitis initially and found no other factor to explain his symptoms except for ureteric obstruction and infection. In this setting the specificity of this finding also adds weight to our conclusion that pancreatitis was most likely due to the effects of the right ureteric obstruction in this patient.

In hindsight there were several factors which confounded and maybe even delayed the diagnosis in this case. The initial clinical picture was confusing because of the non-specific and common presenting symptoms; abdominal pain and fever; which could occur in both pancreatitis and renal tract infections.^{4,5} A further confounding factor which delayed identification of the cause of the pancreatitis in this case, is that renal insufficiency is itself one of the major systemic manifestations of acute pancreatitis. Therefore the renal impairment in this patient was initially assumed to be due to the pancreatitis, rather than due to disease within the urinary tract itself.⁶

To date there have been no reports in the literature of acute pancreatitis associated with an obstructed urinary system. There is however evidence in some studies that the finding of hydronephrosis on CT scans of patients with pancreatitis is not uncommon.¹⁰ We therefore recommend that clinicians consider the entire clinical picture and not only findings of hydronephrosis on a CT scan, when attributing a renal cause to the pancreatitis.

Recognising cases of acute pancreatitis due to ureteric obstruction and infection will allow prompt intervention in the form of percutaneous ureteric decompression. This treatment rapidly alleviates symptoms thus reducing the overall morbidity and mortality for the patient.

We highlight this unusual case of acute pancreatitis occurring in the setting of ureteric obstruction, in an attempt to raise awareness of this clinical scenario. We hope this will prompt clinicians to consider ureteric obstruction and infection in their list of potential causes of acute pancreatitis in the emergency setting.

References

1. Conwell DL. Acute and chronic pancreatitis. *Practical Gastroenterology*. 2001; 1: 47-52.
2. Banks, PA. Epidemiology, natural history and predictors of disease outcome in acute and chronic pancreatitis. *Gastrointestinal Endoscopy*. 2002; 56:S226 to S230.
3. Maes B, Hastier P, Buckley MJ *et al*. Extensive aetiological investigations in acute pancreatitis: results of a 1-year prospective study. *Euro J Gastroenterol Hepatol*. 1999; 11: 891-896.
4. Koizumi M, Takada T Kawarada Y *et al*. JPN Guidelines for the management of acute pancreatitis: diagnostic criteria for acute pancreatitis. *J Hepatobiliary Pancreat Surg*. 2006; 13(1): 25-32.
5. Brandes JC, Campbell DA, Kleinman JG. Pyelonephritis complicating relapsing acute pancreatitis. *Am J of Nephrology* 1989; 9:241-243.
6. Takeyama Y, Ueda T, Hori Y, Takase K, Fukumoto S, Kuroda Y. Hydronephrosis associated with acute pancreatitis. *Pancreas*. 2001; 23:218-220.
7. Gumaste VV, Roditis, N, Mehta D, Dave, PB. Serum lipase levels in nonpancreatic abdominal pain versus acute pancreatitis. *Am J Gastroenterology*. 1993; 12: 2051-2055.
8. Chase CW, Baker DE, Russell WL, Burns RP. Serum amylase and lipase in the evaluation of acute abdominal pain. *Am Surg*. 1996; 12: 1028-1033.
9. Wilson, RB, Warusavitarne J.*et al*., Serum elastase in the diagnosis of acute pancreatitis: a prospective study. *ANZ J Surg*. 2005. 75(3): 152-156.
10. Morehouse HT, Thornhill BA, Alterman DD. Right ureteral obstruction associated with pancreatitis. *Urol Radiol*. 1985 7; 3:150-152.

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