

Case Report

Acute pancreatitis following coronary angiography: case report and review of contrast-induced pancreatitis

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ABSTRACT

Acute pancreatitis is a common surgical presentation with a multitude of causative factors. While the pathogenesis is not completely understood, new potential triggers have been described in recent literature. Contrast-induced pancreatitis is one of these rare phenomena. We present a case of acute pancreatitis in a patient who underwent coronary angiography and discuss the suspected pathogenesis behind contrast-induced pancreatitis. A 65-year-old man with background of cholecystectomy and UroLift procedure underwent two-stage elective coronary angiography following an episode of angina. He had been started on perindopril, rosuvastatin, aspirin and clopidogrel the week prior following first-stage percutaneous transluminal angioplasty of the right coronary artery. The patient underwent uncomplicated angiography, receiving 120 ml of Omnipaque 350. After transfer to the ward, he complained of progressive epigastric pain and nausea. On examination, the patient was afebrile, haemodynamically stable and tender in the epigastrium. His lipase was 888 U/l. Liver ultrasound showed an absent gallbladder but no other abnormalities. Total cholesterol was 2.7 mmol/l and IgG subclasses within normal range. There was no indication to perform an EUS. His symptoms resolved and his lipase normalised within three days. Although contrast-induced pancreatitis is rare, it should be considered in patients exposed to intravenous contrast who manifest symptoms. It occurs due to reduced capillary flow resulting from increased viscosity of radiographic contrast. The rate of invasive coronary investigations continues to rise with cardiovascular disease affecting one in five Australians. Therefore contrast-induced pancreatitis will likely become more common in this population hence it is important to be recognised.

Keywords: Contrast-induced pancreatitis, Coronary angiography, Pancreatitis

INTRODUCTION

Pancreatitis is an inflammatory process characterised by abdominal pain and elevated lipase. The worldwide incidence is increasing and has been reported to be 4.9 to 73.4 cases per 100,000 people.¹ It costs the health system greater than two billion dollars per year in the United States.² The majority of cases (approximately 80%) are caused by alcohol and gallstones.^{3,4} Drug induced pancreatitis is rare and accounts for less than 5% of cases, and contrast pancreatitis accounts for an even smaller proportion. However, due to the increasing availability of radiographic procedures, recognizing contrast-induced

pancreatitis as a potential complication is vital for optimising patient care.

CASE REPORT

A 65-year-old man presented for an elective coronary angiogram as part of a two-staged investigation for coronary artery disease. His only past medical history is a cholecystectomy and UroLift procedure. The patient has no other medical conditions and is allergic to penicillin. He was recently started on perindopril and rosuvastatin for risk factor modification following an episode of angina. Aspirin and clopidogrel were also commenced

the week prior following his first stage angiogram where a stent was placed in the right coronary artery.

The patient underwent coronary angiogram at midday, receiving 120 ml of Omnipaque 350. No intervention was required, and the procedure was completed without complication. After being transferred to the ward, the patient complained of epigastric discomfort that progressively intensified, reaching a peak of 10/10 at 6:00 pm with associated nausea. On examination, the patient was tender in the epigastrium but otherwise afebrile and hemodynamically stable. A full panel of bloods were taken with the only abnormality a lipase of 888 U/l. Calcium was within normal limits and total cholesterol was 2.7. The patient was a non-alcoholic and not diabetic. A CT abdomen and biliary ultrasound was also conducted which showed an absent gallbladder but no other abnormalities. The following day the patient was pain free and repeat lipase was 241 U/l. On the third day of admission, prior to discharge, his lipase had normalized to 48 U/l. IgG subclasses were all within expected range. In the absence of other precipitating factors and immediate onset of epigastric pain with elevated lipase, the patient was diagnosed with contrast-induced pancreatitis.

DISCUSSION

Contrast-induced pancreatitis is rare, with first reports dating back to 1981 following ventriculography.⁵ Since then, only a handful of case reports have been published describing this uncommon condition.⁶⁻⁹ The pathophysiology of contrast-induced pancreatitis is poorly understood, however, mechanisms to explain its occurrence have been explored.

Impaired microcirculation as a result of radiographic contrast can cause insult to the pancreas and subsequent inflammation. Schmidt et al demonstrated in animal models a significant reduction in total capillary flow following infusion of contrast media as compared with normal saline.¹⁰ The reduction in flow was largely due to a decline in pancreatic microcirculation in low-flow capillaries. They also noted capillary stasis in 3.4% of subjects injected with contrast compared to 1.2% with normal saline. Consequently, this reduction in tissue perfusion leads to impaired oxygenation and subsequent tissue injury.

The microcirculation of the pancreas can also be affected by the viscosity of radiographic contrast. These agents increase whole blood and plasma viscosity and reduce haematocrit.¹¹ Kheda and Szerlip demonstrated this concept in a series of two case reports where iodixanol-320, which has a viscosity of 11.8 cp, induced pancreatitis, whereas in the same patients, the lower viscous agent iohexol-300 (6.3 cp) did not.⁹ The authors suggested that the increased plasma viscosity with iodixanol decreased blood flow velocity leading to potential ischemic tissue injury. These cases provide an

interesting finding in that there may be potential after further studies to implement protocols where higher viscosity agents such as iodixanol are avoided, especially in patients that have had contrast-induced pancreatitis.

Studies conducted on mice by Jin et al have shown the deleterious effect of contrast media on the pancreas at a molecular level.¹² These authors were able to demonstrate that activation of NF- κ B, calcium signaling and calcineurin by contrast media leads to acinar cell injury and consequent pancreatitis. Interestingly, the use of calcineurin inhibitors, such as FK506, prevented activation of NF- κ B and subsequent pancreatic inflammation. Although the use of calcineurin inhibitors to prevent contrast induced pancreatitis has not been studied in human models, it may be a useful protective agent in the future following further research.

Even though contrast-induced pancreatitis is rare, with the increasing use of radiological investigations, it should be considered by clinicians as a potential cause of pancreatitis in patients exposed to contrast medium. Impairment of microcirculation and changes at a molecular level in the pancreas from contrast media are potential mechanisms that cause pancreatitis. Studies looking at ways to prevent contrast-induced pancreatitis through low viscosity agents or calcineurin inhibitors may be beneficial in the future.

CONCLUSION

Clinicians should consider contrast agents as a potential cause of pancreatitis. Use of radiographic contrast material can potentially exacerbate underlying pancreatitis and increase the incidence of local or systemic complications. Use of low viscosity contrast media such as iohexol-300, may reduce the risk of pancreatitis in patients undergoing investigations requiring radiographic contrast.

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