Acute necrotizing pancreatitis following coronary artery angiography: A case report

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Abstract

BACKGROUND: Acute pancreatitis has different etiologies from biliary stone to metabolic disturbances. Coronary angiography is one of the newly understood etiologies.

CASE REPORT: This paper is about a women suffering from acute pancreatitis after coronary angiography.

CONCLUSION: Embolization of cholesterol crystals due to vessel wall trauma during coronary angiography as well as contrast medium are responsible for such side effect.

Keywords: Pancreatic Diseases, Coronary Angiography, Contrast Media

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Introduction

Acute pancreatitis is defined as inflammation of pancreas, with or without tissue fibrosis.1 Biliary stone and alcohol are the most common cause of acute pancreatitis.2 In this paper we report a case of acute pancreatitis following angiography, a rare cause.

Case Report

The patient was a 71 years old woman, presenting to our emergency department with acute, severe, continuous and positional epigastric pain, accompanied with nausea and non-bilious, non-bloody vomiting containing ingested food. There was no itching, icterus and anorexia. She was hospitalized in a cardiology center for chest pain, undergoing angiographic procedure about 48 hours before admission to our emergency department. Angiography was done by catheterization of femoral artery and injection of about 100 cc of Visipaque™ (GE Healthcare, Cork, Ireland). Coronary artery stenosis was ruled out and the patient was discharged with medical treatment.

The patient was under medical treatment with aspirin, allopurinol, metoprolol and spironolactone for several months before angiography and after that, without any significant adverse effect. On arrival, vital signs were stable and except severe epigastric tenderness nothing was detected. Lab test showed a high serum amylase level (more than 500 IU/l). Abdominal sonography reveals several hypoechoic zones in pancreas head and neck with surrounding edema. Pancreatic duct had normal size, common bile duct (CBD) was mildly dilated (10 mm), and no stone or mass was detected. Magnetic resonance cholangiopancreatography (MRCP) showed mild dilatation of CBD, pancreatic head enlargement and mild effusion in hepatorenal pouch (Figure 1).

Figure 1. Magnetic resonance cholangiopancreatography shows mild dilatation of common bile duct, pancreatic head enlargement, and mild effusion in hepatorenal pouch

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There were no signs of stone, sludge or mass in the biliary tract. Therefore, patient was admitted to the gastroenterology ward with the diagnosis of acute pancreatitis, received conservative treatment and was discharged with marked improvement after five days.

Discussion

Acute pancreatitis (inflammation of pancreas) has different etiologies which in order of frequency are biliary stones, alcohol, trauma, infection, hypertensive episodes, hypertriglyceridemia, hereditary and metabolic disturbance, etc.\(^3\) New and rare etiologies of acute pancreatitis, which are truly affecting morbidity and mortality of patients, have been introduced recently. Drugs, intravenous radiocontrast agents (used during angiography or other imaging modalities),\(^4\) peripheral vascular disease and atherosclerosis\(^5\) are some of them.

In this patient angiography was suggested as the most probable cause due to its temporal relationship with the occurrence of acute pancreatitis and also the absence of other risk factors.

During angiography, both contrast media and cholesterol crystals embolization (atheroembolism) can be responsible for necrosis of pancreas.\(^6\).\(^7\) Several cases of pancreatitis due to contrast media consumption have been presented.\(^9\),\(^10\) Recently Jin et al. reported that contrast media consumption leads to acute pancreatitis because of changing some cellular calcium signaling pathways.\(^11\)

Visipaque\(^\text{TM}\) (iodixanol: C\(_{35}\)H\(_{44}\)I\(_6\)N\(_6\)O\(_{15}\), 100 cc) was the contrast agent used in this case. It is an isosmolar, water soluble and nonionic agent, with a molecular weight of 1550.20. Its iodine content is 49.1%. Occurrences of acute pancreatitis after Visipaque\(^\text{TM}\) consumption is a novel finding, not reported in the past. On the other hand, presence and moving of cholesterol crystals through the blood vessels (atheroembolism) as the result of atherosclerotic vessel wall traumatization during angiography, is questionable, too. Since our patient did not have typical feature of atheroembolism such as blue toe or renal failure, and regarding to previous similar reports about necrotizing pancreatitis caused by contrast agents, it is assumed that contrast agent used during angiography was responsible for necrosis of pancreas in mentioned patient, not atheroembolism.

Pancreatitis is a known complication of angiography that occurs due to atheroembolism or contrast agents. There are a few documents about Visipaque\(^\text{TM}\) and its inflammatory mechanism, too. There are not enough evidences to indicate whether using non-ionic or low osmolality contrast agent can prevent pancreatitis.\(^12\) Well-designed clinical trials are needed to answer this question.

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None.

Conflict of Interests

Authors have no conflict of interests.

References

1. Bradley EL 3\(^{\text{rd}}\). A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. Arch Surg 1993; 128(5): 586-90.
2. Renner IG, Savage WT 3\(^{\text{rd}}\), Pantoja JL, Renner VJ. Death due to acute pancreatitis. A retrospective analysis of 405 autopsy cases. Dig Dis Sci 1985; 30(10): 1005-18.
3. Opie EL. The etiology of acute hemorrhagic pancreatitis. Bull Johns Hopkins Hospital 1901; 12: 182-8.
4. Runzi M, Layer P. Drug-associated pancreatitis: Facts and fiction. Pancreas 1996; 13(1): 100-9.
5. Ramirez G, O'Neill WM Jr, Lambert R, Bloomer HA. Cholesterol embolization: A complication of angiography. Arch Intern Med 1978; 138(9): 1430-2.
6. Moolenaar W, Lamers CB. Cholesterol crystal embolization to liver, gallbladder, and pancreas. Dig Dis Sci 1996; 41(9): 1819-22.
7. Orvar K, Johlin FC. Atheromatous embolization resulting in acute pancreatitis after cardiac catheterization and angiographic studies. Arch Intern Med 1994; 154(15): 1755-61.
8. Drost H, Buis B, Haan D, Hillers JA. Cholesterol embolism as a complication of left heart catheterisation. Report of seven cases. Br Heart J 1984; 52(3): 339-42.
9. Blasco-Perrin H, Glaser B, Pienkowski M, Peron JM, Payen JL. Gadolinium induced recurrent acute pancreatitis. Pancreatology 2013; 13(1): 88-9.
10. Schenker MP, Solomon JA, Roberts DA. Gadolinium arteriography complicated by acute pancreatitis and acute renal failure. J Vasc Interv Radiol 2001; 12(3): 393.
11. Jin S, Orabi AI, Le T, Javed TA, Sah S, Eisses JF, et al. Exposure to Radiocontrast Agents Induces Pancreatic Inflammation by Activation of Nuclear Factor-kappaB, Calcium Signaling, and Calcineurin. Gastroenterology 2015; 149(3): 753-64.
12. Sherman S, Hawes RH, Rathgaber SW, Uzer MF, Smith MT, Khusro QE, et al. Post-ERCP pancreatitis: Randomized, prospective study comparing a low- and high-osmolality contrast agent. Gastrointest Endosc 1994; 40(4): 422-7.

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