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Case Report

A Case of Acute Pancreatitis Following Computed Tomography Scan

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ABSTRACT

Background Acute pancreatitis is a common cause of hospitalization among gastrointestinal disorders and its frequency has been rising in the past few years. The majority of cases are due to alcohol use, gallstones and hypertriglyceridemia. However, there still remain a significant number of cases in which no causative factor can be found and therefore called idiopathic. Contrast induced pancreatitis is a rare cause pancreatitis and there are only a few cases reported so far. Here we presented a case of mild acute pancreatitis following iodinated contrast exposure.

Case Report A 42-year-old female patient with a history of lymphoma was admitted to our clinic with severe abdominal pain and nausea. Her blood tests revealed elevated pancreatic enzyme levels and mildly elevated liver function tests. Upper abdomen magnetic resonance imaging revealed pancreatic inflammation without any sign of necrosis. Since her complaints began after a computed tomography scan that she had earlier that day for the evaluation of lymphoma and no other cause could be found, iodinated contrast was thought to be the cause of acute pancreatitis in this patient.

Conclusions Contrast agents seem to be a rare cause of acute pancreatitis, however taking the increasing availability of procedures involving radiocontrast agents into consideration, it is important to keep in mind that clinicians may come across more cases of contrast-induced acute pancreatitis in the future.

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Introduction

Acute pancreatitis (AP) is a common cause of hospitalization among gastrointestinal disorders and its frequency has been rising in the past few years. Pathophysiology of AP involves both the localized destruction of pancreas and systemic inflammatory response. The severity of AP varies widely and it is classified based on Revised Atlanta Classification 2013 as mild, moderately severe and severe AP. Severe AP results in persistent organ failure and death in approximately 20% of the cases.[1] The majority of cases are due to alcohol use, gallstones and hypertriglyceridemia. However, there still remain a significant number of cases in which no causative factor can be found and therefore called idiopathic. Although the frequency of drug induced pancreatitis is very low, it should be considered when other common causes of pancreatitis are ruled out and therefore a detailed history of drug intake should be taken from every AP patient.[2] Contrast induced pancreatitis is a rare cause of drug induced pancreatitis and there are only a few cases reported so far. Here we presented a case of mild acute pancreatitis following iodinated contrast exposure.

Case Report

A 42-year-old female patient was admitted to our clinic with severe abdominal pain and nausea. She had a history of non-Hodgkin lymphoma (NHL) that had been diagnosed last year and she had received 4 cycles of R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, prednisone) so far. She received the fourth cycle approximately 20 days ago. Her pain began a few hours ago and was first located in the epigastrium and right upper quadrant. It progressively increased in severity and began to radiate towards her lower back.

Upon admission, her vital signs were normal with a body temperature of 36.5 °C, a heart rate of 83 beats per minute, a blood pressure of 125/70 mmHg and a respiratory rate of 16 breaths per minute. Mild tenderness was present in her epigastrium and right upper quadrant. Her blood tests revealed elevated pancreatic enzyme levels (amylase: 2,672 U/L, pancreatic amylase: 1,896 U/L, lipase: 3,781 U/L) and mildly elevated liver function tests (ALT: 128 U/L, AST: 196 U/L, ALP: 72 U/L, GGT: 109 U/L) *(Table 1).*

A diagnosis of acute pancreatitis was made, and the patient was hospitalized. Intravenous hydration was initiated while her oral intake was discontinued. Upper abdomen magnetic resonance imaging along with magnetic resonance cholangiopancreatography was conducted and pancreatic inflammation was detected without any sign of necrosis *(Figure 1).* No gallstones were present. The patient did not have a history of alcohol consumption. Neither hypercalcemia nor hypertriglyceridemia was detected in her blood tests. She has not recently used any new drugs except for the iodinated contrast that was administered to her earlier that day, for the computed tomography scans that were performed to evaluate the status of her NHL. Abdominal

Table 1. Laboratory parameters of the patient at admission, in 48 hours and at discharge

Parameter	At admission	In 48 hours	At discharge
Hematocrit (%)	37.3	35.7	36.4
Leukocyte count (x10^3/μL)	5.9	3.3	7.2
Creatinine (mg/dL)	0.7	0.54	0.5
Blood urea nitrogen (mg/dL)	13.31	5.24	9.1
ALT (U/L)	128	63	28
AST (U/L)	196	32	14
Amylase (U/L)	2,672	276	88
Pancreatic amylase (U/L)	1,896	190	52.7
Lipase (U/L)	3,781	96	28
Calcium (mg/dL)	8.7	9.78	8.69
Triglyceride (mg/dL)	60		

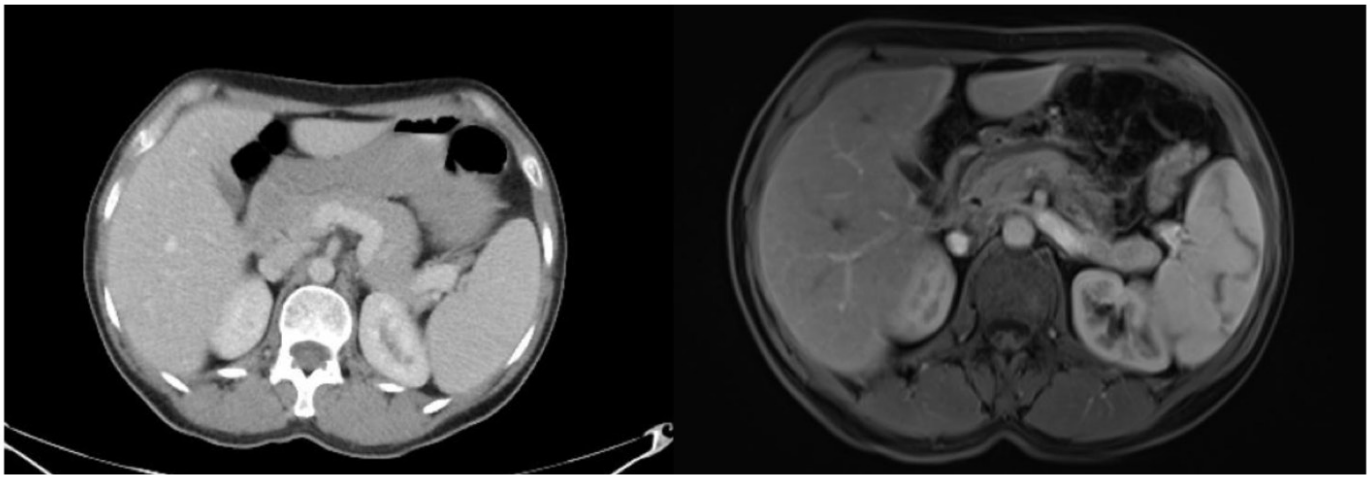

Figure 1. Magnetic resonance cholangiopancreatography images showing pancreatic inflammation without any sign of necrosis.

CT scan was examined, and no sign of pancreatic inflammation was noted. Since her complaints began only a few hours after the iodinated contrast administration, it was thought to be the most likely cause of acute pancreatitis in this patient. On follow up, her pain began to resolve, and pancreatic enzyme levels began to decrease. Her pain resolved completely on the third day of hospitalization and oral intake was initiated. She didn't have any further complaints and was discharged on the next day.

Discussion

Contrast agents are used for a variety of diagnostic and therapeutic procedures in medicine. Their usage is associated with various complications, contrast induced nephropathy being the most significant. Contrast induced pancreatitis has also been reported in the literature, but there aren't many cases. Since such procedures are becoming increasingly available worldwide, it is important for the clinicians to be aware of even the rarest complications in order to make suitable interventions.

Cases of contrast induced AP date back to 1956 when Robinson reported a case with autopsy findings of AP following translumbar aortography.[3] After that, there were several other AP cases reported following aortography and a more recent case following ventriculography in 1981 by Chin *et al.*[4] More recently, cases of AP following coronary angiography and thrombectomy have been published.[5-7] In February 2020, Mui *et al.*[8] reported a case of mild contrast induced AP, whose symptoms began right after being transferred to ward after uncomplicated coronary angiography. It has also been shown that contrast enhanced CT performed immediately after the onset of AP symptoms may further damage the pancreas.[9]

The pathophysiology of contrast induced AP is not well understood. The most recognized hypothesis is the impaired microcirculation in pancreatic tissue due to contrast exposure, similar to contrast induced nephropathy. In 1995, Schmidt *et al.*[10] examined rats with acute pancreatitis and demonstrated that contrast infusion induced a significant decrease of total pancreatic capillary flow and concluded that contrast exposure aggravated the impairment of pancreatic microcirculation in experimental pancreatitis. However, in 2005 Plock *et al.*[11] conducted a meta analysis to review whether the application of contrast enhanced CT worsens the course of AP due to impaired microcirculation in humans and found out that there were not enough data to support this hypothesis in humans.

In 2014, Jin *et al.*[12] investigated the effects of pancreas exposure to contrast in mice and human cell lines at the molecular level. They found out that incubation of mouse and human acinar cells with iohexol led to increased intracellular release

of calcium and activation of nuclear factor-kappa B. They also showed that iohexol did not result in pancreatic inflammation in calcineurin Aβ-deficient mice and concluded that calcineurin inhibitors might be used to prevent post-endoscopic retrograde cholangiography (ERCP) pancreatitis, which is a finding that needs to be studied in human models.

It is also possible to hypothesize that, chemotherapeutic agents such as cyclophosphamide and doxorubicin or corticosteroids could be the cause of AP in our patient, or they might had induced a pancreatic inflammation at first and later, AP was triggered by the iodinated contrast administration. There are several case reports in the literature that assume there is a relation between the use of these therapeutic agents and AP.[13,14] However, the diagnosis of contrast induced AP cannot still be ruled out in this patient.

Since AP is one of the leading causes for hospitalization among gastrointestinal disorders and is associated with significant morbidity and mortality, it is important to define the causative factors and take precautions against them. Contrast agents seem to be a rare cause of AP, however taking the increasing availability of procedures involving radiocontrast agents into consideration, it is important to keep in mind that clinicians may come across more cases of contrast induced AP in the future. Studies are needed to find ways to prevent this phenomenon, such as using lower volumes or lower osmolality contrast.

Conflict of Interests

Authors declare that there are none.

Authors' Contribution

Study Conception: BS, LO; Study Design: BS; Supervision: LO, EP; Analysis and Data Interpretation: BS, LO, EP; Literature Review: BS; Manuscript Preparation: BS; Critical Review: LO, EP.

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