152

IHJ Cardiovascular Case Reports (CVCR) xxx (2017) xxx–xxx

Contents lists available at ScienceDirect

IHJ Cardiovascular Case Reports (CVCR)

journal homepage: www.elsevier.com/locate/ihjccr

Contrast induced acute pancreatitis following coronary intervention

Jamshed J. Dalal*, Aarti Digrajkar

Kokilaben Hospital, Andheri, Mumbai 400053, India

ARTICLE INFO

Article history:
Received 5 April 2017
Received in revised form 29 May 2017
Accepted 26 June 2017
Available online xxx

Keywords:
Contrast
Coronary intervention
Pancreatitis

ABSTRACT

Contrast induced pancreatitis following coronary procedures is rare, but has been reported. It is important to consider this possibility when a patient presents with abdominal pain or multi organ manifestations post procedure. We present here such a case and possible mechanisms for the complication. Awareness would allow early diagnosis and appropriate treatment.

Case

A 75-year-old man, suffering from hypertension and diabetes, presented with non-ST elevated acute coronary syndrome. He was treated elsewhere with medical therapy and came to us a week later. He was stable with a left ventricular ejection fraction of 35% with anterior wall hypokinesis. His coronary angiogram showed a 95% lesion in LAD with multiple moderate lesions in other vessels. After discussing all options an angioplasty was performed with a drug eluting stent implanted in the severe LAD lesion. He was discharged after 48 hours with a creatinine of 1.3 mg/dL, a value same as pre procedure.

He came back to the casualty three days later with a history of severe nausea and dyspnea. There was no pain or fever. His heart rate was 120/min, blood pressure of 100/70 mm Hg, and he was drowsy. His white cell count was 10,180 mcl, hemoglobin was 10.2 g/dl, SGOT was 30 U/L, and SGPT 132 U/L. Blood sugars, platelet counts, troponin and CPK were normal. However his serum creatinine had shot up to 8.8 mg/dL with serum potassium of 8.1 mmol/L. He was severely acidotic and rapidly deteriorated and was put on a ventilator, and underwent immediate hemodialysis.

A clinical diagnosis of contrast induced, or cholesterol embolic acute renal injury with metabolic acidosis and shock was made. Also possibility of mesenteric ischemia was considered.

He was started on IV Meropenem along inotropic support, with dopamine and noradrenaline and continued on hemodialysis. To our surprise, serum amylase was reported as 1023 U/L, on day one, which rose to 1575 U/L the next day. His serum lipase was more than 3000 units/L on the first day and remained high in this range over the next many days. This raised the possibility of acute pancreatitis as the primary problem, with renal and hemodynamic manifestations being secondary to the pancreatitis. His abdominal USG did not visualize the pancreas well and a CT scan showed peripancreatic fat stranding, medical renal disease, and fatty liver with mild ascites but no gallstones. Contrast injection during CT scan was avoided due to the present problem being related to use of contrast. He had no fever and complement and Pro calcitonin levels were normal ruling out sepsis. No other cause of pancreatitis was found. With standard treatment of hydration, inotopic support and correction of fluid and electrolyte balance, the patient recovered over the next few days and was discharged ten days later.

Discussion

The question raised in this case was whether pancreatitis was induced by the contrast media (CM) used during the intervention. We have never encountered this situation and though it has been described in literature, there is little discussion on this subject.

Only a few cases of contrast agent induced pancreatitis have been reported in literature.[1–4] It is a rare complication of angiography with a possible frequency of less than 0.1%.

There are two possible theories for the mechanism of this problem

1) One mechanism postulates that pancreatitis and its severity may be related to the degree of pancreatic infarction from the cholesterol emboli. Autopsies from some these patients have

* Corresponding author.
E-mail address: jjdalal@hotmail.com (J.J. Dalal).

http://dx.doi.org/10.1016/j.ihjccr.2017.06.002

Please cite this article in press as: Dalal JJ, Digrajkar A, Contrast induced acute pancreatitis following coronary intervention, *IHJ Cardiovasc Case Rep* (2017) http://dx.doi.org/10.1016/j.ihjccr.2017.06.002

demonstrated significant cholesterol embolization to the liver, kidneys, and spleen, leading to this hypothesis.

2) A second mechanism suggested by Gorges (1) suggested that the contrast agent leads to impaired circulation similar to the pathophsiology behind contrast induced renal injury and hypothesizes that ischemia induced by viscous contrast could be contributing to the development of pancreatic necrosis. This theory gets support from the fact that iodixanol is more likely to cause pancreatitis than iohexol. This difference might be related to the dissimilarities in viscosity and/or osmolality of these two agents. Mufaddal et al suggest that iohexol being less viscous appears to be the safer contrast media.(4) At room temperature, the viscosity of iodixanol- 320 and iohexol-300 are 11.8 and 6.3 cp, respectively (for comparison, human plasma viscosity at hematocrit 0.43 is 1.72 cp).

This relatively higher viscosity of iodixanol compared to other contrast agents is shown to exhibit significant reduction in tissue perfusion. The intravascular use of iodixanol thus increases plasma viscosity and decreases blood flow velocity that can potentially cause ischemia to the pancreas leading to necrosis.[4] In the present case however iohexol had been used.

High CM viscosity is a key element in the pathophysiology of kidney injury also, because the hyperosmolar environment of the renal medulla results in CM enrichment in both the tubules and the vasculature. Thus, fluid viscosity increases and flow through medullary tubules and vessels decreases. Reducing the flow increases the contact time of cytotoxic CM with the tubular epithelium and vascular endothelium. This triggers a vicious circle of cell injury, medullary vasoconstriction, and hypoxia. Moreover, glomerular filtration declines due to congestion of highly viscous tubular fluid.[5]

Iohexol, because of its hyperosmolality (844 mosm/kg), increases the intravascular oncotic pressures. This potentially causes intravascular fluid expansion and maintains perfusion, thus preventing ischemic injury to the pancreas. This may be one of the other reasons for the safety of iohexol CM.

As in this case, patients in previous reports have also presented within a few days of CM exposure and responded to usual care with appropriate antibiotics, hydration and maintenance of hemodynamics.

Experimental studies

In a study on rats, Schmidt et al concluded that radiographic contrast medium aggravates the impairment of pancreatic microcirculation in experimental necrotizing pancreatitis.[6] Another experimental study by Jin and coworkers, has also demonstrated radio contrast induced pancreatitis in mice. This was shown to be caused by activation of NF-kB, calcium signaling and calcineurin. A calcineurin inhibitor, FK506 prevented this pancreatic inflammation.[7] Whether calcineurin inhibitors would have a role in preventing post contrast pancreatitis in humans remains to be seen.

Computed tomography is widely used to diagnose acute pancreatitis. There is concern that the use of contrast agents is associated with worse prognosis in patients with ongoing pancreatitis. Iodinated contrast medium lengthens the duration of pancreatitis and may increase the incidence of local or systemic complications.[8]

Conclusion

Contrast induced pancreatitis is a rare disorder but considering the number of radiological and cardiac procedures being done, one must be aware of this complication. Keeping a diagnosis of acute pancreatitis when a patient presents post contrast procedure with abdominal pain, rising creatinine or multi organ failure is important. Predominantly renal or other manifestations might lead to missing acute pancreatitis as the cause of the problem. CT helps in assessing the pancreas, but the use of contrast CT in patients with suspected pancreatitis may worsen the pancreatic status. Choice of less viscous contrast agents and maintaining hemodynamics and fluid balance with good hydration when dealing with sick patients is important. Management includes early diagnosis and supportive treatment.

Conflict of interest

No author has any conflict of interest.

References

1. Gorges R, Ghalayini W, Zughaib M. A case of contrast induced pancreatitis following cardiac catheterization. *J Invasive Cardiol.* 2013;(October (10)):203–204.
2. Bateman K, Brown R. An unusual source of abdominal pain. *J Hosp Med.* 2014;9(Suppl. 2).
3. Kasi VS, Estrada CA, Wiese W. Association of pancreatitis with administration of contrast medium and intravenous lipid emulsion in a patient with the acquired immunodeficiency syndrome. *South Med J.* 2003;96(January (1)):66–69.
4. Kheda MF, Szerlip HM. Two cases of iodixanol induced pancreatitis. *Nephrol Dial Transplant Plus.* 2008;5:296–299.
5. Seeliger E, Lenhard DC. Contrast media viscosity versus osmolality in kidney injury: lessons from animal studies. *BioMed Res Int.* 2014;2014:358136.
6. Schmidt J, Hotz HG, Foitzik T, Ryschich E. Intravenous contrast medium aggravates the impairment of pancreatic microcirculation in necrotizing pancreatitis in the rat. *Ann Surg.* 1995;221(March (3)):257–264.
7. Jin S, Orabi AI, Javed TA, et al. Exposure to radiocontrast agents induces pancreatitic inflammation by activation of nuclear factor – kB, calcium signaling and calcineurin. *Gastroenterology.* 2015;149(September):753–764.
8. Kasi VS, Estrada CA, Wiese W. Association of pancreatitis with administration of contrast medium and intravenous lipid emulsion in a patient with the acquired immunodeficiency syndrome. *South Med J.* 2003;96(1).

Please cite this article in press as: Dalal JJ, Digrajkar A, Contrast induced acute pancreatitis following coronary intervention, *IHJ Cardiovasc Case Rep.* (2017) http://dx.doi.org/10.1016/j.ihjccr.2017.06.002