Recurrent pancreatitis in an icodextrin-based peritoneal dialysis patient. Yet another case report

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Acute pancreatitis (AP) is an infrequent and severe complication in peritoneal dialysis (PD) patients especially those using icodextrin exchanges. Establishing the diagnosis of acute pancreatitis in PD patients is difficult because the clinical features of AP are very similar to that of PD associated peritonitis in addition to unreliability of serum amylase as marker in diagnosing AP in these patients.

There are very few published case reports of AP in peritoneal dialysis patients using Icodextrin in the literature. Here we present a case of recurrent pancreatitis in a patient on PD using icodextrin.

Case report

A 27-year-old obese female with end stage renal disease (ESRD) secondary to IgA nephropathy presented to the Emergency department with complaints of diffuse, persistent non-radiating pain abdomen associated with nausea and vomiting without any history of fever or loose motions.

She has been on peritoneal dialysis since June 2007 with a regimen consisting of continuous cycling PD (CCPD) alternating between 1.5% and 2.5% dextrose solutions plus a daytime long dwell using 7.5% icodextrin solution. Her transporter status is high average.

On examination she was haemodynamically stable, with tenderness over the epigastric area. Her PD fluid was clear with no evidence of infection. Her amylase levels were persistently normal 11–20 units/litre (U/L; normal <100 U/L). Her upper gastrointestinal endoscopy was normal. A CT scan of the abdomen revealed Necrotising Pancreatitis. Serum lipase done later was elevated at 529 U/L, with normal ranges 8–78 U/L.

She was managed conservatively for acute pancreatitis and was discharged home after about one and a half weeks of hospital admission. The patient had no history of smoking, alcohol or illicit drug abuse and a review of medical records didn’t reveal any evidence of hypercalcemia or hypertriglyceridaemia. Her symptoms resolved rapidly and she tolerated food intake well and she was discharged home.

The patient was re-admitted 3 weeks later with identical symptoms. Her total amylase was low throughout the whole presentation, between 9–22 U/L, however her lipase was elevated at >600 U/L. A repeat CT scan abdomen revealed a necrotising pancreatitis with pseudocyst formation. MRI scan confirmed the CT abdomen findings of necrotising pancreatitis with pseudocyst formation.

She had a prolonged hospital stay with insertion of the pancreatic drain. We failed to transfer her over to haemodialysis because of inability to secure a vascular access. She had a previously failed left arm brachiocephalic AVF with moderate stenosis of both
of her internal jugular veins related to her previous catheters for interim haemodialysis.

She was discharged on her usual PD prescription and had three more admissions thereafter with recurrent pancreatitis over a period of 6 months with similar chief complaints of abdominal pain nausea and vomiting without any evidence of peritonitis and negative fluid cultures and elevated lipase levels. After each admission, her GI symptoms will resolve rapidly with conservative measures.

Following her last admission we entertained the idea of icodextrin-induced pancreatitis. The patient was subsequently discharged home with elimination of icodextrin from her CCPD regimen. During the further outpatient follow-up, she remained free of recurring symptoms of nausea, vomiting and abdominal pain now for last 4 months.

Since she is on nocturnal intermittent peritoneal dialysis now with a short daytime dextrose dwell, she is not meeting her targets for dialysis adequacy. She is booked for review with vascular surgeon for creating a possible vascular access as soon as possible.

**Discussion**

AP is a serious and an infrequent complication in PD patients. The clinical picture of AP in PD patient often resembles that of infectious peritonitis, abdomen pain, vomiting and cloudy dialysate can occur in both the cases, furthermore, the serum amylase is not a reliable marker for AP in patient with icodextrin-based PD, because it competitively interacts with the substrate in the amylase assay and lead to a dose dependent decrease in amylase activity in such patients.\(^1\)--\(^6\)

The diagnosis of AP with renal failure is confounded by the observation that serum concentration of amylase and lipase are generally elevated in ESRF in absence of AP. Thus an abnormally high levels of pancreatic enzymes does not necessarily mean pancreatic pathology.

Schoenicke et al reported that serum amylase activity was reduced by 90% in PD patients using icodextrin compared to those using glucose and lipase activity was not significantly altered after adding varying concentration of icodextrin to serum samples from control patients.\(^4\)

Current literature suggest that PD is a risk factor for AP. Quraishi et al. presented data suggesting that adult patients on PD were 15 times more likely to develop AP compared to the general population in the state of Michigan.\(^3\) Although number of mechanism were postulated for increased incidence of AP in PD patients, one of the mechanisms could be composition and volume of the fluid administered causing chemical irritation. Furthermore calcium in the peritoneal dialysate could diffuse through the peritoneum causing local hypercalcemia of the pancreas even if the systemic calcium levels are within normal limits.

Thus in this subset of patient population the development of acute pancreatitis may represent the cumulative result of multiple coexisting noxious insults—i.e. uraemia, secondary hyperparathyroidism with hypercalcemia, hypertriglyceridaemia, polypharmacy, or the dialysis process itself.
Our patient had always used dialysate with 1.5 mmol/l calcium bath, and both her systemic calcium and calcium × phosphate product remained under good control. Our patient showed no major risk factor for acute pancreatitis with elevated lipase levels and negative imaging studies and so far no more acute pancreatitis for last 4 months made us consider the possibility of a chemically-induced pancreatitis and potential for a causal link between AP and icodextrin use.

Icodextrin was first used in late 1980s, is a relatively novel colloid osmotic agent, predominantly used for long daytime dwells in patients with low net ultra filtration and high peritoneal transport properties. With an average molecular weight of about 17,000 Da, Icodextrin shows very little diffusion across the peritoneal membrane. However it is absorbed into the systemic circulation mainly via lymphatics. 

Generally icodextrin is well tolerated its major side effects, as described in the literature include cutaneous allergic reactions and metabolic derangements such as hyponatremia and decreased plasma amylase activity. Sporadic cases of abdominal pain and sterile peritonitis have been described in the literature, but those episodes have been the result of manufacturing defects or microbial contamination.

In last couple of years few case have been reported related to the higher incidence of recurrent pancreatitis in PD patients using icodextrin, however no cause and effect has been documented neither any pathophysiologic process contributing to AP in these patients is known.

Conclusion

Chemically–induced pancreatitis should be considered in PD patients with recurrent unexplained pancreatitis with non-contributory imaging studies, and peritoneal dialysis fluid negative for infection. It is worthwhile discontinuing icodextrin in these patients before a cause and effect can be established.

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References: