

## A diabetic patient with abdominal pain and lactescent serum

一名腹痛及血清乳白色的糖尿病患者

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We present the case of a poorly controlled diabetic patient suffering from acute onset of abdominal pain and vomiting. A blood sample taken at the bedside revealed lactescent serum and provided an important clue to the patient's status of having severe hypertriglyceridaemia. The clinical diagnosis of hypertriglyceridaemia-induced acute pancreatitis was correctly made in the accident and emergency department. Clinical photos of the lactescent serum are shown. The incidence, risk factors and management of this particular type of acute pancreatitis are discussed. (*Hong Kong j.emerg.med.* 2008;15:139-142)

我們描述一名糖尿病控制得很差及患上急性腹痛及嘔吐的病人個案。床旁抽取的血液樣本顯示乳白色的血清，提供病人有嚴重高三酸甘油酯血症狀況的重要線索。在急症室臨床正確地診斷為高三酸甘油酯血症引發急性胰腺炎。我們展示乳狀血清的臨床相片，並討論這特別類型急性胰腺炎的發病率，風險因素及治理。

**Keywords:** Diabetes mellitus, hyperlipidaemias, hypertriglyceridaemia, pancreatitis

**關鍵詞：**糖尿病，高脂血症，高三酸甘油酯血症，胰腺炎

### Introduction

The most common identifiable causes of acute pancreatitis are cholelithiasis, cholechololithiasis and alcoholism. Only about 1.3 to 3.8% of the cases of acute pancreatitis with identifiable cause have been ascribed to severe primary or secondary hypertriglyceridaemia as the aetiology.<sup>1,2</sup>

### Case presentation

A 27-year-old moderately obese lady was brought in by ambulance because of severe back and abdominal pain with repeated vomiting for a few hours. She had a history of type 2 diabetes treated initially with oral hypoglycaemic agents but currently switched to insulin injection therapy. Her diabetic control was all along poor with Hb<sub>A1C</sub> level persistently above 10% and complicated with diabetic nephropathy with proteinuria. She had a history of loin pain for years. Previous ultrasound examination revealed hepatosplenomegaly but she refused further investigation. She was found on screening three years ago to have high serum triglyceride level (TGL) of 14 mmol/L and had therefore been given oral gemfibrozil treatment since then. However, her drug compliance was poor and her serum TGL remained at high level between

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10 to 32 mmol/L. She was a non-drinker. She had no previous history of pancreatitis and had no family history of hyperlipidaemia or pancreatitis.

On physical examination, she was afebrile with stable vital signs except mild tachycardia of 109 beat/min. She was in distress with sweating and she maintained a leaning forward posture because of abdominal pain. The abdominal examination revealed epigastric tenderness without guarding nor rebound tenderness. The bedside glucose level was 18 mmol/L. Urine ketone and pregnancy tests were negative. Further blood chemistry investigations were conducted. On venous blood sampling, a grossly pinkish blood was obtained (Figure 1). On standing the blood tube upright for five minutes, a milky white (lactescent) supernatant (serum) was notably seen on top of the blood clot or cell sediment (Figure 2). We made a provisional diagnosis of hypertriglyceridaemia-induced pancreatitis based on the clinical presentation and

lactescent serum. She was admitted to the surgical department for further management.

Initial blood investigations revealed compensated metabolic acidosis. The amylase level was 323 U/L. The adjusted calcium level was 1.63 mmol/L. Liver and renal function tests were essentially normal. There was a remarkably elevated serum TGL of 77.1 mmol/L. After admission, she was managed as suffering from acute pancreatitis. The serum amylase level was monitored and it peaked at 883 U/L. Urgent abdominal ultrasound revealed a swollen pancreatic head with peripancreatic fluid and fatty liver. A gallstone of 0.5 cm in diameter was noted. Both the pancreatic duct and common bile duct were not dilated. Endoscopic retrograde pancreaticocholangiography was attempted but failed because of swollen duodenal mucosa and ampulla. She received active supportive treatment and her condition gradually improved. Gemfibrozil was started and her TGL dropped to 9.5 mmol/L before discharge on day 10 after admission.



Figure 1. Pinkish colour blood sample.



Figure 2. Lactescent (miky white) serum seen after 5 minutes.

## Discussion

The aetiology of acute pancreatitis was numerous. High serum TGL is known to precipitate an attack of acute pancreatitis and has accounted for 1.3 to 3.8% of the cases, though the pathogenesis of inflammation was unclear.<sup>1,3</sup> Hypertriglyceridaemia can be caused by both inherited and acquired disorders of triglyceride metabolism. Hyperlipoproteinaemia was classified by Frederickson into 5 phenotypes, in which type I and V are associated with lactescent blood and occurrence of acute pancreatitis without other precipitating factors.<sup>2,4</sup> Secondary hyperlipidaemia is commonly associated with obesity, diabetes mellitus, nephrotic syndrome, hypothyroidism and drugs like oestrogen, tamoxifen, beta blocker and glucocorticoid. However, very high serum TGL (>11 mmol/L) is rare and occurs in less than 1 in 5000 individuals.<sup>3</sup> A serum TGL above 11.3 mmol/L indicates an increased risk of developing acute pancreatitis with an incidence of up to 21%.<sup>2</sup> In one case series of 70 patients with hypertriglyceridaemia-induced acute pancreatitis, the mean serum TGL was 52 mmol/L. Patients typically presented with an acute onset of abdominal pain, nausea, and vomiting. Most of the cases were poorly controlled diabetic or alcoholic patients with history of hypertriglyceridaemia; and only 15-20% of the cases were non-diabetic, non-alcoholic, non-obese patients with drug/diet induced hypertriglyceridaemia. Serum amylase level is less useful in diagnosis because substantial hyperamylasaemia (> 2 times of the upper limit of normal) might not be seen in nearly half of the patients with hypertriglyceridaemia-induced pancreatitis.<sup>1</sup> Spuriously, a normal amylase level has been reported.<sup>5</sup> The underlying reason of low amylase level was unknown but could be related to the suppression of enzyme activity by a circulating inhibitor.<sup>6</sup> Therefore, the diagnosis should depend on a high index of clinical suspicion on high risk patients. For uncertain cases, ultrasound or CT scan could be very useful in confirming the diagnosis, as well as looking for the aetiology and complications.

In this reported case, the patient belonged to the high risk group of poorly-controlled diabetes with history of hypertriglyceridaemia. Her presentation was typical,

especially with her grossly lactescent blood noted on blood sampling. Based on all these information, together with a high index of clinical suspicion, a quick and correct bedside diagnosis could be arrived at without difficulty. The milky appearance of the serum was mainly due to the high level of very low density lipoprotein (VLDL) or chylomicron.<sup>3</sup>

The clinical course of hypertriglyceridaemia-induced pancreatitis is not different from pancreatitis of other causes.<sup>2</sup> During the acute phase, active supportive care should be given as in all patients suffering from acute pancreatitis. However, extra effort should be placed to reduce the serum TGL in this particular group of patients. Intravenous heparin or insulin could stimulate lipoprotein lipase activity and have been effective in reducing serum TGL and blood viscosity.<sup>7</sup> Plasmapheresis performed within 48 hours of admission was reported in one case series as helpful in reducing the serum TGL as well as improving the clinical state including pain symptom.<sup>8</sup> However, the effectiveness of plasmapheresis has not been clearly defined yet.

Recurrent attacks of acute pancreatitis could be prevented by lowering the serum TGL to less than 2.2 mmol/L. The management of hyperlipidaemia includes non-pharmacologic therapy such as weight reduction in obese patients, aerobic exercise, avoiding alcohol, strict glycaemic control in diabetics, avoidance of concentrated sugars and medications that might raise serum TGL. For pharmacologic therapy, bile acid sequestrants should be avoided because they could increase VLDL synthesis and exacerbate hypertriglyceridaemia.<sup>9</sup> A fibrate such as gemfibrozil was considered as first line therapy.<sup>10</sup> Refractory cases could try fish oil supplements (>3 g/day), which contain omega-3 fatty acids that could lower VLDL production. However, gastrointestinal and metabolic side effects limited their use and there was a concern about increase in LDL levels.<sup>11-13</sup> The addition of orlistat, which inhibited pancreatic and gastric lipases, has been successfully used as an adjunct to treat patients with sky-high serum TGL in a small case series.<sup>14</sup> For those patients with marked symptoms, anecdotal reports suggested that non-selective plasmapheresis and plasma exchange could be helpful.<sup>7,15</sup>

## Conclusion

We report a typical case of hypertriglyceridaemia-induced acute pancreatitis correctly diagnosed at bedside in the accident and emergency department. Emergency physicians should have a high index of clinical suspicion on this condition when they encounter alcoholic, poorly controlled diabetic or nephrotic patients presenting with an acute onset of abdominal pain and vomiting. Lactescent serum found on blood taking can reveal the patient's status of severe hypertriglyceridaemia and is a very useful hint of the diagnosis.

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