# An 8½ year old girl presented with pain abdomen with hypertriglyceremia

**Luthfun Nahar, A. S. M. Bazlul Karim, Md. Rukunuzzaman and Afsana Yasmin**

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Department of Pediatric Gastroenterology and Nutrition, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Bangladesh

**For Correspondence:**
Luthfun Nahar
dr.mukta27@gmail.com

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## Presentation of Case

*Dr. Zannatul Ferdous Sonia:* A 8 ½ year old girl, 1st issue of non-consanguineous parents, from Norshingdi immunized as per EPI presented at the outpatient department with the history of abdominal pain for 5 days. The pain was located in the epigastric region and dull in nature. There was no aggravating or relieving factor and no radiation and persisted all the days. The pain had no relation with the food. She had also the history of vomiting for several times for the same duration which occurred usually after feed and contained food particle and not mixed with blood or bile and it was not projectile. She had no history of fatigue, weight loss, polyuria, polyphagia, polydypsia, respiratory distress or constipation. She had the history of similar type of attacks for repeated times for the last 7 months. The pain was so severe that she needed hospitalization for several times. Her younger brother is healthy and there is no family history of such type illness. On examination, she was afebrile, anicteric, mildly pale, and vitals were within the normal limit. Anthropometrically she was normal and the BCG mark was present. Abdominal examination showed epigastric tenderness. There was no organomegaly and ascites was absent. Other systemic examinations showed normal findings.

For her complaints she was admitted in a private hospital and underwent some investigations like serum electrolytes, amylase, lipase, alkaline phosphatase, alanine aminotransferase, calcium, random blood sugar, fasting triglyceride, ultrasonography of the whole abdomen, etc (Table I). Her reports showed severe hypertriglyceremia with raised serum amylase, lipase and blood sugar. The ultrasonography report showed a single calcification in the right lobe of liver measuring 6.6 mm. She was treated with intravenous fluid, injectable proton pump inhibitor and ceftriaxone but her condition didn't improve and was then referred to this tertiary level hospital for better management.

After admission in the Bangabandhu Sheikh Mujib Medical University, the blood was sent for complete blood count, fasting lipid profile, serum $T_4$, TSH, plain X-ray of abdomen in erect posture, chest X-ray and Mantoux test (Table I).

## Provisional Diagnosis

Hypertriglyceremic acute pancreatitis

## Differential Diagnosis

*Dr. Afsana Yasmin:* As peptic ulcer diseases also present with similar type of clinical features, I thought it could be a case of peptic ulcer disease.

### *Peptic ulcer disease*

Peptic ulcer diseases can occur in children, but it is uncommon in pediatric age group. In children, it may be due to primary or secondary causes. Among the primary causes *Helicobacter pylori* infection is the commonest. Other primary causes are Zollinger–Ellison syndrome, G-cell hyperplasia, systemic mastocytosis, short bowel syndrome, hyperparathyroidism, etc. Secondary ulcers are more common than primary which have bad prognosis. Secondary ulcer may develop due to stress, sepsis, trauma, burn, type 1 diabetes mellitus, drug, etc.[1] Among the drugs NSAID, steroid, immunosuppressive drugs, etc can cause ulcer.[2] Stress ulcer is more common in children of less than 4 years of age and the primary ulcer is more common above 4 years of age and primary ulcer may recur even after treatment.[3-4] Cushing ulcer associated with brain tumor or brain injury which are typically single, deep which are prone to perforation. Stress and spicy food don't cause ulcer but exacerbate the symptoms. The common symptoms are gas, bloating, nausea, vomiting, epigastric pain, abdominal discomfort which usually increase at empty stomach or after meal.[2] Gastrointestinal bleeding may occur with long standing epigastric pain but painless bleeding may be the only manifestation of ulcer.[5] Diagnostic examinations include urease breath test, stool tests for *H. pylori* detection, anti-HP antibody ($I_gG$) and upper gastrointestinal tract endoscopy.[2, 6] Primary peptic ulcers in children usually presents between the ages of 8 to 17 years, commonly around 12 years, usually the cause is

**Table I**

**Laboratory investigations of patient on admission**

| Parameter | Patient | References |
|---|---|---|
| Hemoglobin (gm/dL) | 10.3 | 13.5 ± 1.3 |
| TC of WBC (×10⁹/L) | 9 | 7.0 ± 3 |
| *Differentials* | | |
| Neutrophil | 63% | 40-80% |
| Lymphocyte | 30% | 20-40% |
| Eosinophil | 05% | 1-06% |
| Monocyte | 02% | 2-10% |
| Platelet count (x10⁹/L) | 170 | 150-400 |
| ESR (mm in 1st hour) | 55 ↑ | 0-10 |
| Fasting blood sugar (mmol/L) | 4.3 | 3.5-6.0 |
| Random blood sugar (mmol/L) | 11.8 | <7.8 |
| Serum triglyceride (mg/dL) | 2650 ↑↑ | <150 |
| Serum amylase (U/L) | 240 | 25-115 |
| Serum lipase (U/L) | 160 | Up to 60 |
| *Serum electrolyte: (mmol/L)* | | |
| Sodium ($Na^+$) | 134 | 136-148 |
| Potassium ($K^+$) | 3.80 | 3.5-5.2 |
| Chloride ($Cl^-$) | 98 | 98-108 |
| Carbon dioxide ($CO_2$) | 25 | 21-32 |
| Serum alanine aminotransferase (U/L) | 74 | 30-65 |
| Serum alkaline phosphatase (U/L) | 190 | 50-136 |
| Serum calcium (mg/dL) | 10.3 | 8.1-10.4 |
| Serum $T_4$ (nmol/L) | 120 | 54-173 |
| Serum TSH (mIU/L) | 5.04 | 0.47-5.01 |
| *Serum lipid profile (mg/dL)* | | |
| Total cholesterol | 148 | <200 |
| HDL cholesterol | 17 ↓ | >40 |
| Triglyceride | 894 ↑ | <150 |
| Anti *H. pylori* antibody | Negative | |
| Plain X-ray abdomen | Normal | |
| Chest X-ray | Normal | |
| Mantoux test | 3 mm (negative) | >10 mm |
| Ultrasonography of whole abdomen | Calcification in right lobe of liver measuring about 1.12 cm and soft calculus or sludge in the gall bladder | |
| Upper GI endoscopy | Normal | |

unknown and may present with long-term history.[5,7]

In this patient, epigastric pain with no radiation along with vomiting of undigested food and epigastric tenderness goes in favor of peptic ulcer disease, but no relation with food and not relieved by antiulcerant goes against the peptic ulcer disease.

To exclude peptic ulcer disease, we have done anti-*H. pylori* antibody (IgG) and upper GI endoscopy. Her anti-*H. pylori* antibody (IgG) was negative and upper gastrointestinal endoscopy findings were normal. So, it was not a case of peptic ulcer disease.

***Diabetic ketoacidosis***

Type 1 diabetes may cause diabetic ketoacidosis which is a life threatening condition and a common cause of death in diabetic child. Majority of type 1 diabetes patients unfortunately present with diabetic ketoacidosis as initial presentation. Diabetic ketoacidosis is characterized by hyperglycemia, acidosis, with ketoacid accumulation in the blood due to insulin deficiency. The mean age of presentation of diabetic ketoacidosis is around 9 years. The biochemical criteria for the diagnosis of diabetic ketoacidosis are: a) blood glucose: >200 mg/dL or ≥11.1 mmol/L, b) venous pH: <7.3 or bicarbonate: <15 mmol/L, c) ketonemia and ketonuria.

Insulin plays the main role for the production and utilization of ketones. Normally insulin inhibits lipolysis, oxidation of free fatty acids, and increases oxidation of ketones in the peripheral tissues. So, both overproduction and under utilization of ketones occur in insulin deficient state as diabetic ketoacidosis.[8] A triad of acute pancreatitis, hypertriglyceremia and diabetes is also reported at the same time.[9] The clinical features of diabetic ketoacidosis varies according to severity and associated comorbidities. Polyuria with polydipsia are the common presenting symptoms including weight loss, fatigue, dyspnea, vomiting, abdominal pain, polyphagia, dehydration, unconsciousness, poor skin turgor, dry mucous membranes, orthos-tatic hypotension, deep (Kussmaul) respirations and a fruity smell on the patient's breath due to increased acetone.[10] Among them common presen-ting symptoms are abdominal pain, vomiting and dehydration.[11]

Study suggested that girls are at more risk for diabetic ketoacidosis than boys; consanguinity, family history of diabetes are also risk factors. It is recommend to exclude diabetes in all cases of acute abdomen in children.[11]

Diabetic ketoacidosis cases can be roughly divided in to the following grades based on pH and bicarbonate.[12] Mild: pH 7.2–7.3, bicarbonate 10–14; moderate: pH 7.1–7.3, bicarbonate 5–9, and severe: pH <7.1, bicarbonate <5.

The severe complication of diabetic ketoacidosis is cerebral edema. Other complications in children

include hypokalemia, hypoglycemia, acute renal failure, shock, rhabdomyolysis, thrombosis, stroke, pneumomediastinum, prolonged corrected QT interval, pulmonary edema, decreased cognitive function, etc.[10]

This 8½ year old girl presented with severe upper abdominal pain and vomiting which goes in favor for diabetic ketoacidosis but there is no history of fatigue, weight loss, poyluria, polyphagia, polydypsia, respiratory distress and patient was conscious, respiratory pattern was normal and repeated attacks of same type illness goes against diabetic ketoacidosis.

*Dr. Luthfun Nahar:* After evaluating the patient's presenting features, physical findings and initial investigation results (Table I). Report showed raised serum amylase and lipase with very high level of fasting triglyceride. Severe recurrent epigastric pain and vomiting along with high serum amylase and lipase are in favor of acute pancreatitis. Presence of high level of serum triglyceride indicates hypertriglyceremia as the underlying cause of pancreatitis.

*Acute pancreatitis:* Acute pancreatitis is a reversible process characterized by the histological evidence of inflammation of pancreatic parenchyma, interstitial edema, infiltration of inflammatory cells with variable degrees of cellular apoptosis, necrosis and hemorrhage.[13]

Acute pancreatitis is diagnosed by the presence of at least two of the following three criteria[14-17]:

1. Characteristic abdominal pain of acute onset, severe dull ache, especially in the epigastric region may radiate to back, aggravated by eating or drinking, usually after taking fatty food and relieved by lean forward or knee-chest position.[18]

2. Serum amylase and /or lipase level at least more than 3 times of upper normal limit.

3. Positive imaging findings.

There are several etiologies of acute pancreatitis. Among the metabolic causes hypertriglyceremia is one of established cause of pancreatitis.

***Hypertriglyceridemia***

Hypertriglyceridemia indicates increased level of plasma fasting triglyceride concentration above 95$^{th}$ percentile for age and sex. A triglyceride level of ≥100 mg/dL is considered as hypertriglyceremia in children of 0-9 years of age and a level of ≥130 mg/dL for children of 10-19 years of age. Hypertriglyceridemia results from either increased triglyceride production or reduced triglyceride clearance. Hypertriglyceridemia means increase level of circulating triglyceride rich lipoproteins which is produced by the liver or absorbed from food. Dietary fat converts into chylomicron within enterocyte and VLDL synthesis occur in liver. Both of them are degradated by endothelial and hepatic lipoprotein lipase to free fatty acid and monoglyceride.[19]

Grossly the triglyceride level are:

Normal: <150 mg/dL; (1 mmol = 88.6 mg/dL)

Mild hypertriglyceremia: 150-199 mg/dL; Moderate hypertriglyceremia: 200-999; Severe hypertriglyceremia: 1000-1999 mg/dL; Very severe hypertriglyceremia: ≥2000 mg/dL.

The risk of AP with serum triglyceride of >1000 mg/dL is about 5% and >2000 mg/dL is 10-20%. It is not established that HTG pancreatitis is more severe than due to other causes.[20, 21]

The complication of HTG are pancreatitis, cardiovascular diseases, non alcoholic fatty liver disease etc.[22]

HTG is a well established risk factor for pancreatitis and the risk is about 1-4%. Usually primary lipid disorder is due to genetic defect of triglyceride synthesis and metabolism. According to Friedrickson's classification of hyperlipoproteinemia, there are 5 types of primary lipid disorder: Type I and V only presence of chylomicrons in plasma.[23-24]

Type I hyperlipoproteinemia indicates the presence of chylomicrons, normal total cholesterol with very high level of triglyceride; whereas type V hyperlipoproteinemia is defined as the presence of chylomicrons and very low density lipoproteins with increased level of cholesterol and very high level of triglyceride.

Type 1 hyperlipoproteinemia is mainly due to genetic deficiency of lipoprotein lipase (LPL) or other related proteins as apolipoprotein C2, A5, lipase mutation factor 1 (LMF1) and glycosyl-phosphatidylinositol-anchored high-density lipoprotein binding protein 1 (GPIHBP1. It is diagnosed by genomic DNA analysis for APOA5, APOC2, LMF1 and GPIHBP1 and immunoblotting method to detect serum LPL autoantibody.

This patient presented with recuurent attacks of acute abdominal pain favorable for pancreatitis and no suggestive history for peptic ulcer diseases or diabetic ketoacidosis. Biochemically she had raised serum amylase, lipase and severe hypertriglyceremia. As there is no evidence of secondary causes of hypertriglyceremia; so, probably it is a case of primary hypertriglyceremia.

In this patient, triglyceride level was very high with normal total cholesterol and LDL couldn't be detected by Friedewald's formula, HDL was normal. She had acute pancreatitis. So, probably it is Type 1 hyperlipoproteinemia. But we couldn't confirm it due to lack of facilities to do genetic and antibody analysis.

## Dr. Nahar's Diagnosis

Acute pancreatitis due to primary hypertriglyceremia

## Discussion

*Dr. A. S. M. Bazlul Karim:* The term "recurrent acute pancreatitis" was first introduced in 1948 in medical literature by Henry Doubilet.[25] According to INSPIRE criteria it is defined by at least two separate episode of acute pancreatitis with absence of irreversible, structural changes in the pancreas. The patient must have to meet the criteria of acute pancreatitis after the first attack.[26]

Most common causes of recurrent acute pancreatitis are bile duct stone or sludge, sphincter of oddi dysfunction, anatomical abnormality of pancreatic tree or ductal stone, genetic mutation, hyperlipidemia, hypercalcemia, ascariasis, autoimmune pancreatitis, drug such as azathioprine, mercaptopurine, sulphonamide, etc. Even organophosphorus compounds can cause acute pancreatitis.[25, 27-29]

The girl had similar type of attacks before admission but there is no biochemical proof of pancreatitis. From history it is assumed that she could have previous attacks of pancreatitis. Then the case can not categorized as acute recurrent pancreatitis.

Treatment of acute recurrent pancreatitis is the same as acute pancreatitis. Initial treatment include rest to pancreas by limiting oral intake, aggressive intravenous hydration, and pain management; along with treatment of underlying cause and complications.[20]

*Dr. Md. Rukunuzzaman:* Type 1 hyperlpoproteinemia also called familial hyperchylomicronomia. It may be due to environmental influence or may be genetic. The disorder tends to run in families. We have done fasting lipid profile of patient's mother and it was near normal limit.

Figure 1: Lipemic serum

The presentation of type 1 hyperlipoproteinemia are recurrent acute pancreatitis, eruptive xanthomas, lipema retinalis, hepatosplenomegaly, abnormal liver enzymes, etc.[30]

When patient presents with severe hypertriglyceremia, serum turn to creamy appearance which is called lipemic serum.[31]

Lipema retinalis is a transient change of retina as creamy appearance of retinal blood vessels due to lipid infiltration and may decrease visual acuity. Lipid lowering therapy may turn to normal fundus and visual acuity.

This patient attended to us on 3rd day. She had lipemic serum (Figure 1) and eye findings were normal

*Dr. Md. Wahiduzzaman Mazumder:* Hypertriglyceremia lead to triglyceride rich chylomicron sludge in the capillary bed which cause ischemia of pancreas and pancreatic lipase release from damaged pancreatic acini. Further production of FFA leads to free radical damage and inflammation which cause premature activation of trypsinogen to trypsin and activation of other enzymes resulting pancreatitis. Noncompliance of low fat diet may cause recurrent pancreatitis in type 1 hypertriglyceremia. Recurrent pancreatitis can cause chronic exocrine and endocrine pancreatic insufficiency.[19]

There may have relationship with some genetic predisposition to hypertriglyceremia induced pancreatitis as like mutations in cationic trypsinogen (PRSS1), serine protease inhibitor Kazal type 1 (SPINK1), cystic fibrosis transmembrane conductance regulator (CFTR), and tumor necrosis factor superfamily member 2 (TNF2).[20]

*Dr. Karim:* Acute management of severe hypertriglyceremia is done by fasting, insulin or heparin infusion or plasmapheresis.[32]

A patient of severe hypertriglyceremia should be kept nothing by mouth with intravenous fluid. Diet increases further chylomicron production and exacerbate hypertriglyceremia. So, fasting initially prevent chylo-micron production and is helpful for gradual clearance of chylomicron and significant reduction of hypertriglyceremia within one or two days. When triglyceride level is at 1,000 mg/dL and there is no abdominal pain then fat free oral diet can be started.[32]

Insulin infusion reduce the triglyceride level rapidly, it can be given by continuous or subcutaneous infusion. Intravenous insulin infusion along with fasting will reduce triglyceride level up to 80% in first 24 hours. Continuous insulin can be given at 0.1-0.3 U/kg/hours in dextrose infusion to maintain blood glucose level between 140-180 mg/dL. In diabetic patient continuous insulin infusion at 0.5 to 1 IU/kg/hours can be used with others supportive management.[32]

A case report of nondiabetic adolescent patient with severe hypertriglyceremia, subcutaneous infusion of bolus dose of regular insulin at 0.1 U/kg decreased triglyceride level rapidly.[33]

Heparin also release lipoprotein lipase from muscle, adipose tissue and endothelium to circulation and hydrolyze the triglyceride. This effect is short lived and is quickly followed by increased hepatic LPL degradation. Therefore heparin is not routinely recommended to treat hypertriglyceremia in children.[32, 33]

Plasmapheresis is another option to treat severe hypertriglyceremia. In children, there are only few

case reports to use this option. It can reduce triglyceride level about 70%. It may be used in patient with severe hypertriglyceremia with acute pancreatitis complicated by lactic acidosis, severe hypocalcemia, organ failure and with acute respiratory distress syndrome.[32]

Long-term management of hypertriglyceremia needs lifestyle changes including dietary restriction of fat <10-15% along with weight reduction, increasing physical activity and pharmacotherapy.

Fibrate (gemfibrozil and fenofibrates) are the first line drugs to treat hypertriglyceremia. Gemfibrozil 600 mg twice daily can be used. Adverse effect may be cholesterol gall stone. Fibrate is contraindicated in renal impairment and gall bladder diseases.[19, 20, 32]

Nicotinic acid or niacin is a second line drug. But child may develop flushing, abdominal pain, vomiting, headache and increase liver enzymes.[19, 20, 32]

Statin (atrovastatin, simvastatin, rosuvastatin) is a weak triglyceride lowering drug and can be used as combined therapy with fibrate.[19, 20, 32]

*Omega 3 fatty acid:* It is a polyunsaturated essential fatty acid containing eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) which is usually present in fish oil and seafood. The dose at 2 gm/day reduces the triglyceride level rapidly. It should be used with other drugs as combined therapy but not so effective as monotherapy.[19, 20, 34]

*Cholestyramine:* It is a bile acid sequestrant, which binds bile in the gastrointestinal tract and prevent its absorption. It removes bile acid from the body by forming insoluble complex and excreted in intestine. As a result conversion of cholesterol into bile lowers the plasma cholesterol level.

*Dr. Sayma Rahman Munmun:* What is the mechanism of insulin to reduce triglyceride level?

*Dr. Rukunuzzaman:* Insulin increase the activity of peripheral lipoprotein lipase and helps to reverse hepatic insulin resistance. So, triglyceride breaks down to free fatty acid and monoglycereide. Insulin is very effective in patient with poorly controlled diabetes with hypertriglyceremia.[19, 20]

*Dr. Shuborna Rani Das:* How will we confirm that hypertriglyceridemia is the cause of pancreatitis?

*Dr. Karim:* Triglyceride level ≥1,000 mg/dL provides strong support for hypertriglyceremia-induced pancreatitis if there is no underlying etiology for acute pancreatitis. Triglyceride level of ≥500 mg/dl is a high degree of suspicion for acute pancreatitis. Triglyceride should be estimated within 24 hours of presentation and triglyceride level will fall rapidly in the fasting state in case of hypertriglyceremia-induced pancreatitis.[20]

*Dr. Archana Shrestha Yadav:* How we can prevent further episode of pancreatitis?

*Dr. Md. Wahiduzzaman:* Triglyceride level <500 mg/dL is the safe therapeutic target to prevent recurrent attack. So lifestyle modification with weight loss, restricted fat, consumption of simple carbohydrate, control risk factors of secondary causes, discontinuation of offending drugs and sometime pharmacotherapy as fibrate along with other drugs may be helpful to prevent recurrence. Multi-faceted approach is required in patients with hypertriglyceridemia pancreatitis to prevent recurrences.[20]

*Dr. Hazera Akter:* What treatment modalities you have applied for this patient?

*Dr. Nahar:* This patient was treated initially in a private clinic with nothing by mouth, intravenous fluid, injectable antibiotics, injection tramadol hydrochloride and proton pump inhibitor. After admission in our hospital fat free diet we allowed with other conservative management of acute pancreatitis. For hypertriglyceremia subcutaneous insulin 0.1 unit/kg was started with monitoring of blood glucose. Her triglyceride level dramatically reduces from 2,650 to 838 mg/dL (Figure 2). At the same time we continued fibrate (gemfibrozil) and niacin. Subsequently we added omega 3 fatty acid, anti-oxidant, ursodeoxycholic acid and cholestyramine. Blood sugar level was monitored during insulin therapy (Figure 2).

*Dr. Parisa Marjan:* What drug was advised during discharge?

*Dr. Nahar:* We advised to restrict dietary intake of fat, increase physical activity along with pharmacotherapy as fibrate, niacin, omega 3 fatty acid, cholestyramine and anti-oxidant.

*Dr. Mohua Mondol:* What is the explanation of hepatic calcification in this case?

*Dr. Nahar:* The most common cause is hepatic tuberculosis. Others etiologies are sarcoidosis, hemangioma, echinococcal cyst, hepatic adenoma, etc. She had no history of fever, weight loss, contact with TB patient and her chest X-ray was normal and Mantoux test was negative. Hepatic calcification in this case may be due to a dilated intrahepatic bile ductule.

*Dr. Kamrun Nahar:* Why there is gall bladder sludge and is it may the cause of pancreatitis?

*Dr. Nahar:* This patient was initially kept nothing by mouth. So, less contraction of gall bladder may the cause for gall bladder sludge. She received injectable Ceftriaxone for two days in a private clinic. Ceftriaxone also may be responsible it. She had also severe hypertriglyceremia. So, I think sludge is not the underlying cause.

*Dr. Habibur Rahman:* Why initially patient had hyperglycemia and what measures were taken?

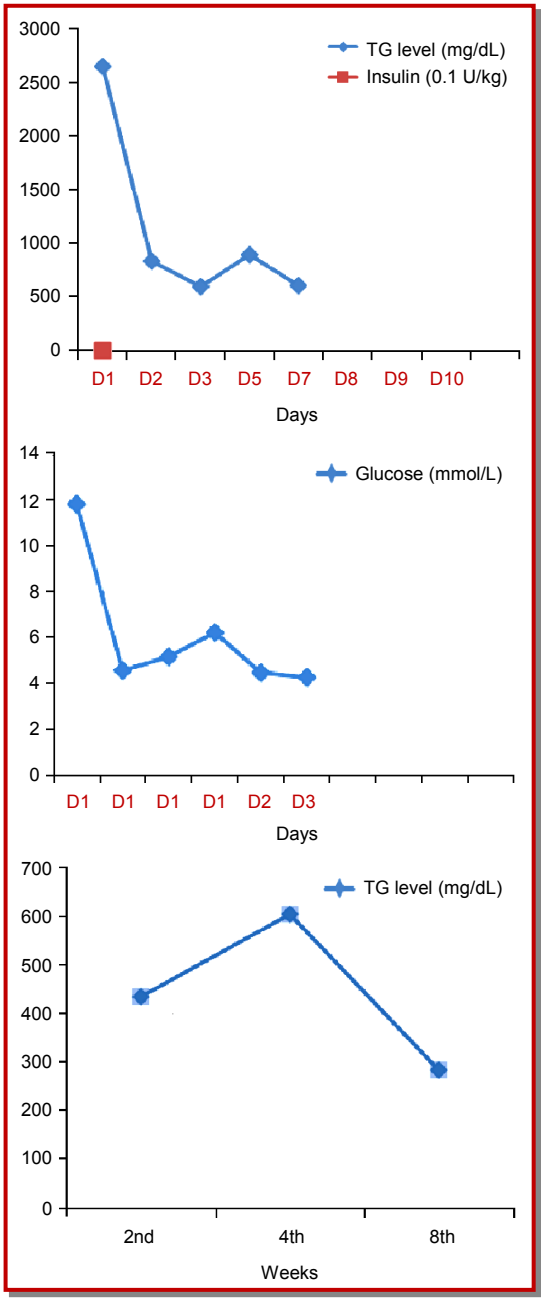


Figure 2: Triglyceride level (upper), random blood sugar (middle) during hospital period and triglyceride during follow-up after discharge (lower)

*Dr. Nahar:* Though acute pancreatitis is a stressful condition. Human body regulates the stress response by hypothalamic pituitary adrenal axis and releases more adrenaline, catecholamine, cortisol to reduce the mortality. At the same time, body maintains glucose intolerance and insulin resistance. So, this patient had hyperglycemia and no measures were taken to reduce glucose level. On subsequent follow-up, it was within normal limit.

## Follow-up

Acute recurrent pancreatitis need long-term follow-up. Our plan to follow-up the patient 2, 4 and 8 weeks after discharge by doing fasting lipid profile (Figure 2).

## Final Diagnosis

Acute pancreatitis due to primary hypertriglyceremia

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