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# Acute pancreatitis secondary to hypertriglyceridemia

**A man presents with acute severe abdominal pain and triglycerides 5,250 mg/dL**

A 42-year-old man with obesity, type 2 diabetes mellitus, hyperlipidemia, depression, asthma, and obstructive sleep apnea presents with severe abdominal pain, nausea, and vomiting. He says the pain started 18 hours ago and has steadily worsened. He describes the pain as 8 on a scale of 10, intermittent, aching, and mainly localized to his epigastric area. It does not radiate and is relieved with simethicone. He also has had some nonbloody diarrhea since the pain started. He recalls a similar episode 2 years ago and was told it was pancreatitis, and it resolved.

He has had no recent trauma and has not traveled or started new medications. He says he does not use tobacco or illicit drugs, and he consumes alcohol occasionally, the last time 3 days ago, when he had approximately 3 glasses of wine.

He says he has recently started eating more fried and unhealthy foods. He admits to not complying with his medications, which include metformin, albuterol, simvastatin, and escitalopram. His diabetes is not under control, but he has not been hospitalized in the last 2 years for hyperglycemia. His family history is significant for hyperlipidemia and diabetes in his father and brother.

## INITIAL EVALUATION

The patient's body mass index is 36.5 kg/m$^2$ and his waist circumference is 44 inches. His temperature is 98.1 °F (36.7 °C), heart rate 90 beats per minute, blood pressure 148/92 mm Hg, respiratory rate 20 breaths per minute, and oxygen saturation 98% on room air.

On physical examination, he has mild discomfort because of the pain, and his abdomen is mildly tender to palpation near the epigastric region. Bowel sounds are diminished in all 4 quadrants. The abdomen is not visibly distended, and the skin is without jaundice, pruritus, or xanthomas.

### Laboratory test results

- Hematocrit 45% (reference range 41–53)
- Glucose 347 mg/dL (70–110)
- Lipase 19,889 U/L (31–186)
- Triglycerides 5,250 mg/dL (35–160)
- White blood cell count 9.8 × 10$^9$/L (4.5–11)
- Bicarbonate 28 mmol/L (21–28)
- Sodium 138 mmol/L (136–142)
- Potassium 4.2 mmol/L (3.5–5)
- Blood urea nitrogen 16 mg/dL (6–24)
- Serum creatinine 1.1 mg/dL (0.6–1.2).

### Imaging and electrocardiography results

Abdominal ultrasonography shows an enlarged fatty liver and a heterogeneous pancreas with some mild peripancreatic fluid surrounding it. His gallbladder has a wall thickness of less than 3 mm and no cholelithiasis.

Computed tomography (CT) shows hepatomegaly with fatty liver and moderate pancreatitis.

Electrocardiography shows normal sinus rhythm.

## DIAGNOSING ACUTE PANCREATITIS

Acute pancreatitis is one of the more common diagnoses in patients hospitalized for epigastric abdominal pain. It is an inflammatory process that begins with the acinar cells in the pancreas, initiating a systemic inflammatory

The authors report no relevant financial relationships which, in the context of their contributions, could be perceived as a potential conflict of interest.

doi:10.3949/ccjm.87a.19156

response. In severe cases, this inflammatory process can lead to necrosis of the pancreas and multisystem organ failure, resulting in a high risk of death. To help reduce the risks, it is essential to recognize acute pancreatitis early and initiate timely treatment.

Guidelines recommend abdominal CT if the diagnosis is unclear or if the patient's symptoms have not improved after 2 or 3 days of treatment.[1] Thus, this patient underwent CT to confirm the cause of his pain, and it showed moderate pancreatitis. He now fulfilled all 3 of the diagnostic criteria for acute pancreatitis (**Table 1**)—abdominal pain in the epigastric region, significantly elevated serum lipase, and pancreatitis on imaging.

The severity of his acute pancreatitis and the risk of complications is assessed using the Bedside Index for Severity in Acute Pancreatitis (**Table 2**).[2] Our patient's score is 0, indicating a low mortality risk—less than 1%.

**TABLE 1**

**Diagnostic criteria for acute pancreatitis**

**Must have at least 2 of these 3 criteria:**

Abdominal pain

Serum amylase or lipase level at least 3 times the upper limit of normal

Ultrasonography, computed tomography, or magnetic resonance imaging that shows irritation of the pancreas

Based on information in reference 8.

## NEXT STEP: IDENTIFY THE CAUSE OF ACUTE PANCREATITIS

**1** What is the most likely cause of this patient's acute pancreatitis?

- ☐ Cholelithiasis
- ☐ Alcohol use
- ☐ Hypertriglyceridemia
- ☐ Hereditary pancreatitis
- ☐ Pancreatic duct variants or anomalies
- ☐ Autoimmune pancreatitis
- ☐ Pharmaceutical agents

### Cholelithiasis

Cholelithiasis is one of the most common causes of acute pancreatitis in the United States and needs to be looked for in patients with acute pancreatitis. To identify it as the instigating factor, there has to be cholelithiasis or choledocholithiasis on radiologic imaging with no signs of other risk factors.[3] The patient's liver enzyme panel also must be assessed. Small stones and sludge have been hypothesized to irritate the wall of the gallbladder and contribute to causing some cases of idiopathic acute pancreatitis. This patient's history of diabetes also increases his risk of biliary sludge.[4]

Patients with signs of ductal obstruction secondary to cholelithiasis should undergo endoscopic retrograde cholangiopancreatography (ERCP), which has been shown to decrease overall morbidity and mortality in patients with ductal obstruction.[5] It is warranted if acute pancreatitis worsens or there are clinical signs of ductal obstruction, including an increase in total bilirubin, aspartate aminotransferase, alanine aminotransferase, or ductal dilation on imaging.[1,5,6]

Magnetic resonance cholangiopancreatography (MRCP) has been shown to be a helpful tool to diagnose acute pancreatitis, but it has no benefit in the clinical management of acute pancreatitis because it is an imaging modality only; it does not support treatment. The advantage of MRCP is its low risk of adverse effects compared with ERCP, which has been associated with inducing acute pancreatitis, bleeding, and pain. However, MRCP is purely diagnostic, whereas ERCP can be both diagnostic and therapeutic.[6]

Endoscopic ultrasonography also has been proposed as a standard in approaching acute pancreatitis secondary to biliary issues. One of its main benefits is it can be performed at the bedside to evaluate the size of the obstruction to determine if ERCP is warranted.[7]

Patients with acute pancreatitis secondary to gallstones should undergo cholecystectomy to prevent future occurrences. This surgery should be performed during the same hospitalization unless the patient develops severe acute pancreatitis with complications such as pancreatic necrosis. Patients who do not qualify for surgery because of age or comorbidities may undergo biliary sphincterotomy, which can help reduce the frequency of acute pancreatitis.[1]

**Common causes of pancreatitis: gallstones, alcohol use, hypertriglyceridemia**

TABLE 2

**Bedside Index of Severity in Acute Pancreatitis**

| Clinical factor | Score<br>Yes = 1 point<br>No = 0 points |
|---|---|
| Blood urea nitrogen > 25 mg/dL | |
| Abnormal mental status (Glasgow Coma Score < 15) | |
| Evidence of systemic inflammatory response syndrome[a] | |
| Over age 60 | |
| Imaging tests showing pleural effusion | |
| Total points[b] | |

[a]Requires at least 2 of the following: temperature < 36°C or > 38°C, respiratory rate > 20 breaths per minute or $PaCO_2$ < 32 mm Hg, pulse > 90 beats per minute, and white blood cell count < 2.0 or > 12.0 × $10^9$/L or > 10% immature bands.
[b]0–2 points = low mortality risk (< 2%); ≥ 3 points = high mortality risk (> 15%).

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Our patient did not display any signs of cholelithiasis or choledocholithiasis in any of the abdominal imaging studies. However, that does not rule out the possibility of microlithiasis or biliary sludge, and it requires further investigation with a liver enzyme panel and, possibly, endoscopic ultrasonography.

**Triglycerides > 1,000 mg/dL typically cause pancreatitis symptoms**

### Alcohol use

Alcohol is another major cause of acute pancreatitis. Alcohol-induced acute pancreatitis occurs with frequent intake of alcohol or episodes of heavy drinking (> 80 mL of alcohol in 24 hours).[8]

Even though the patient admitted to drinking 3 days before his symptoms started, he denied daily alcohol use or binges, making alcohol use less likely to be the main cause of his acute pancreatitis.

### Hypertriglyceridemia

Pancreatitis induced by hypertriglyceridemia is a well-known phenomenon that typically goes underreported. It is the third most common cause of acute pancreatitis, but is relatively rare, accounting for 7% of cases.[9]

Establishing hypertriglyceridemia as the cause of acute pancreatitis can be challenging because most cases of acute pancreatitis have other causes. Moreover, triglyceride levels can be mildly or moderately elevated with these other causes as well: the 2 main causes of acute pancreatitis, alcohol and gallstones, can both cause acquired hypertriglyceridemia.

Both biliary obstruction by gallstones and alcohol consumption increase the levels of triglycerides because of their secondary effects on fatty metabolism. Biliary obstruction from gallstones increases the trapping of triglycerides in the serum, causing a rise in their levels. Alcohol increases very low-density lipoprotein (VLDL), which causes an increase in the release of fatty acids from both the liver and adipose tissue, also causing a rise in triglycerides in the serum. Hypertriglyceridemia should therefore be considered only after heavy alcohol consumption and gallstones have been ruled out due to the false rise that can be seen in the other 2 primary causes of acute pancreatitis.

Serum triglyceride levels need to be evaluated early in the disease in these patients because levels can fall drastically with fasting, and hypertriglyceridemia can be missed. Patients with hypertriglyceridemia-induced pancreatitis typically have serum triglyceride levels above 1,000 mg/dL and no signs of gallstones or alcohol-induced acute pancreatitis.

Compared with the common causes of acute pancreatitis, hypertriglyceridemia has a more complicated and severe medical course with a higher mortality rate.[10]

This patient's triglyceride level in the emergency department was 5,250 mg/dL, which supports the diagnosis of hypertriglyceridemia as the instigating factor for his acute pancreatitis.

### Hereditary pancreatitis

Hereditary pancreatitis is an uncommon cause of acute pancreatitis, but it must be considered in patients who have multiple episodes of acute pancreatitis. Patients with hereditary pancreatitis-induced acute pancreatitis typically present with multiple episodes by age 30. They have a higher risk of pancreatic adenocarcinoma and require surveillance for it.

Hereditary pancreatitis also increases a patient's risk of developing chronic pancreatitis, which can cause fibrosis and strictures in the pancreatic ducts that can lead to pancreatic

**TABLE 3**

**Revised Atlanta classification of severity of acute pancreatitis**

| Mild | Moderate to severe | Severe |
|---|---|---|
| Absence of organ failure | Organ failure for < 48 hours | Persistent organ failure for > 48 hours |
| Absence of local complications[a] | Local complications[a] | |

[a]Local complications: interstitial edematous pancreatitis, necrotizing pancreatitis, pancreatic pseudocyst, necrotic collection, and pleural effusion.

Based on information in references 8 and 14.

exocrine and endocrine insufficiency. Genetic testing and visualization of the pancreas for signs of acute or chronic pancreatitis can help identify patients with hereditary pancreatitis.[11]

This patient had no knowledge of a family history of pancreatitis or of having pancreatitis before age 30, making hereditary pancreatitis less likely.

### Pancreatic duct variants or anomalies

Abnormalities in the pancreatic duct can lead to recurring acute pancreatitis that usually requires surgical management. Many times, congenital abnormalities in the pancreatic duct can go undetected until adulthood, when they are incidentally found during abdominal imaging. If pancreatic duct abnormalities are suspected, the best initial test is MRCP.[12]

In our patient, no abnormalities were seen on CT of the abdomen or ultrasonography, but those results did not rule out the possibility of an abnormality in the pancreatic duct.

### Autoimmune pancreatitis

Autoimmune pancreatitis, also known as idiopathic duct-destructive pancreatitis, is being clinically recognized more often because of technologic advances in imaging, which may reveal an enlarged pancreas, often with a hypoattenuated rim or narrowing of the main pancreatic duct.

To differentiate autoimmune pancreatitis from a malignancy, a fine-needle biopsy is required. The symptoms most associated with autoimmune pancreatitis are jaundice, weight loss, and epigastric pain. It can also present alongside other autoimmune diseases.[13]

Even though this patient has epigastric pain, he does not have jaundice or weight loss. On imaging, the pancreas appeared to be irritated but not enlarged.

### Drugs

Certain medications can cause acute pancreatitis, including statins, selective serotonin-reuptake inhibitors, and metformin. Medication-induced pancreatitis tends to be mild to moderate but it can be severe.

The diagnosis of medication-induced pancreatitis depends on ruling out the more common causes such as biliary stones or obstructions, alcohol use, and hypertriglyceridemia. The best course of action if medication-induced pancreatitis is suspected is to stop the offending drug and see if the symptoms of acute pancreatitis resolve.

Our patient admits to not taking his medications for the last 2 months. Also, 2 of the more common causes of acute pancreatitis have not been ruled out yet, which make this diagnosis less likely.

**Triglycerides > 2,000 mg/dL are classified as very severe hypertriglyceridemia**

## THE NEXT STEP

**2** What is the most appropriate next step in this patient's workup?

- ☐ Assess his risk of complications of acute pancreatitis
- ☐ Determine if there is a possibility of biliary sludge or microlithiasis
- ☐ Identify the cause of his hypertriglyceridemia

The revised Atlanta classification for acute pancreatitis provides guidance for determining its severity and identifying complications attributed to it (**Table 3**).[14] Organ failure, a key component of the classification criteria, is assessed using the modified Marshall scoring system (**Table 4**).

TABLE 4

## Modified Marshall scoring system for organ failure

| | Respiratory | Renal | Cardiovascular |
|---|---|---|---|
| **Score**[a] | $Pao_2/Fio_2$[b] | Serum creatinine (mg/dL) | Systolic blood pressure (mm Hg) |
| 0 | > 400 | ≤ 1.4 | > 90 |
| 1 | 301–400 | 1.5–1.8 | < 90 and responding to fluid resuscitation |
| 2 | 201–300 | 1.9–3.5 | < 90 and not responding to fluid resuscitation |
| 3 | 101–200 | 3.6–4.9 | < 90 with pH < 7.3 |
| 4 | ≤ 100 | ≥ 5 | < 90 with pH < 7.2 |

[a]A score of 2 or more indicates organ failure. Persistent failure is considered organ failure lasting longer than 48 hours.
[b]Ratio of partial pressure of arterial oxygen to fractional inspired oxygen.

Based on information in references 8 and 14.

TABLE 5

## Criteria for clinical diagnosis of hypertriglyceridemia

| Degree of hypertriglyceridemia | Serum triglycerides (mg/dL) |
|---|---|
| Mild | 150–199 |
| Moderate | 200–999 |
| Severe | 1,000–1,999 |
| Very severe | ≥ 2,000 |

Based on Endocrine Society clinical practice guidelines, reference 18.

During the second week after the onset of acute pancreatitis symptoms, patients may develop local complications that can manifest clinically as unremitting pain, sepsis, elevation in pancreatic enzymes for a second time, or organ dysfunction. These patients should undergo contrast-enhanced CT, contrast-enhanced magnetic resonance imaging (MRI), or non-enhanced MRI to identify the local complications. Imaging done during the first 5 to 7 days of symptom onset does not help detect necrosis in the pancreas or local complications because these complications don't typically arise, or are undetectable, until the second week.[14,15]

Correctly identifying the trigger for the acute pancreatitis is important for long-term management of the patient, in order to prevent future episodes of acute pancreatitis. Unfortunately, some cases might remain as idiopathic acute pancreatitis, in which case the patient should be advised of the most common causes of acute pancreatitis and certain risk factors that can potentiate another episode of it.

## CASE CONTINUED

The patient is admitted to the intensive care unit, where he is placed on nothing-by-mouth orders and given intravenous fluids. Laboratory tests are ordered.

### Laboratory test results

- Low-density lipoprotein cholesterol (LDL-C) 170 mg/dL (reference range 50–130)
- High-density lipoprotein cholesterol (HDL-C) 31 mg/dL (40–75)
- Aspartate aminotransferase 34 U/L (10–40)
- Alanine aminotransferase 18 U/L (9–46)
- Alkaline phosphatase 80 U/L (40–115)
- Total bilirubin 0.7 mg/dL (0.2–1.2)
- Indirect bilirubin 0.6 mg/dL (0.2–1.2)
- Direct bilirubin 0.1 mg/dL (< 0.2)
- Total protein 6.9 g/dL (6.1–8.1)
- Albumin 4.2 g/dL (3.6–5.1)
- Globulin 2.7 g/dL (1.9–3.7)
- Hemoglobin $A_{1c}$ 9.1% (4–5.6).

## MANAGING HYPERTRIGLYCERIDEMIA-INDUCED ACUTE PANCREATITIS

The higher the triglyceride level, the greater the risk of acute pancreatitis and secondary cardiovascular complications.[16,17] Patients who display signs of acute pancreatitis caused by hypertriglyceridemia typically have serum triglyceride levels above 1,000 mg/dL, classified as severe hypertriglyceridemia by the Endocrine Society (**Table 5**).[18]

There are primary and secondary causes of hypertriglyceridemia (**Table 6**). Identifying the cause can help practitioners select appropriate long-term management strategies and address specific risk factors associated with the causes.

This patient's hypertriglyceridemia was most likely caused by metabolic syndrome. He fulfilled all 5 criteria for metabolic syndrome (of which only 3 are needed):

- Waist circumference significantly greater than 40 inches

- Fasting glucose level higher than 110 mg/dL
- Triglyceride level higher than 150 mg/dL
- HDL-C level less than 40 mg/dL
- Systolic blood pressure higher than 130 mm Hg.

Patients with the metabolic syndrome have an excess of visceral adipose tissue, which contributes to the increase in triglyceride levels. Many also develop insulin-resistant diabetes, resulting in increased concentrations of VLDLs and an influx of free fatty acids to the liver, which can contribute to fatty liver disease in this patient population. Having the metabolic syndrome, insulin-resistant diabetes, and elevated apolipoprotein B level can increase a patient's cardiovascular risk by up to 20 times. This risk can be reduced by lowering the triglyceride level through glycemic control and weight loss.

## TREATMENT FOR ACUTE PANCREATITIS

**3** What is the best treatment for this patient's acute pancreatitis?

☐ Insulin drip
☐ Heparin
☐ Plasmapheresis

Acute pancreatitis secondary to hypertriglyceridemia is initially managed by giving aggressive intravenous hydration, limiting food intake, and managing pain. Further management of the elevated triglycerides includes an insulin drip, plasmapheresis, or heparin.[19] There are no formal guidelines to advise the best treatment regimen, so the decision is left to practitioners' discretion, considering the risks and benefits as well as the availability of resources.

### Insulin drip

Insulin drip therapy for hypertriglyceridemia has been shown to be effective in lowering triglycerides to less than 500 mg/dL.[20] It also increases the level of peripheral lipoprotein lipase, which helps process the excess triglycerides. The dosage is 0.1 to 0.3 U/kg/hour by continuous infusion, with glucose monitoring every 30 minutes to every hour. In patients whose glucose levels are below 200 mg/dL, a separate 5% dextrose infusion can help prevent hypoglycemia. Insulin increases the metabolism of low-density lipoprotein and also accelerates chylomicron formation.[9,19,20]

**TABLE 6**

**Causes of hypertriglyceridemia**

| |
|---|
| **Primary causes** |
| Familial hypertriglyceridemia |
| Apolipoprotein C-II deficiency |
| Familial combined hyperlipidemia |
| **Secondary causes (acquired)** |
| Diseases |
| Hypothyroidism |
| Diabetes mellitus |
| Renal disease |
| Human immunodeficiency virus-associated dyslipidemia |
| Systemic lupus erythematosus |
| Nephrotic syndrome |
| Pregnancy |
| Medications |
| Beta-blockers |
| Corticosteroids |
| Thiazides |
| Protease inhibitors |
| Second-generation antipsychotics |
| Estrogen-based oral contraceptives |
| Diet |
| Excessive alcohol intake |
| High-fat diet |

Based on information in reference 16.

### Heparin

Heparin has potential benefits in that it increases lipoprotein lipase, which helps break down triglycerides. However, although heparin, like insulin, causes an initial increase in lipoprotein lipase, in the long term it reduces the activity of this enzyme. Some studies have found that the initial benefits are not worth the long-term risks, which can include an increase in the release of toxic components from triglycerides, decreased metabolism of triglycerides, and storage of triglycerides in the liver. In turn, this can cause a net increase in triglyceride levels and also increase the risk of bleeding.

**Pancreatitis caused by hypertriglyceridemia has a more complicated and severe medical course**

### Plasmapheresis

Plasmapheresis physically removes triglycerides from the blood, leading to faster lowering of levels to below 500 mg/dL.[19,21] It consists of removing some of the patient's plasma with the elevated triglycerides and replacing it with a colloid solution.

The main advantage of plasmapheresis over insulin in correcting hypertriglyceridemia is its speed. However, it is more expensive and also requires the insertion of a central line. It can be considered for patients who are at high risk of complications or have signs of organ failure and necrosis.[19,21] Our patient had no signs of organ failure nor necrosis in the pancreas on his abdominal CT.

## CASE CONTINUED

The patient is started on an insulin drip at a rate of 0.1 U/kg/hour. Because he now requires hourly glucose checks, he is kept in the intensive care unit. His triglyceride levels begin to show a downward trend, although they remain significantly above the normal range. The patient also receives heparin.

### Discussion

**Insulin drip lowers triglycerides in acute pancreatitis**

Untreated acute pancreatitis increases the risk of serious complications and death. The most commonly noted complications are pancreatic pseudocyst, peripancreatic fluid collections, and necrotic collections. In necrotizing pancreatitis, there is a collection of necrosis and fluids that can become walled off and encapsulate the material, enabling bacteria to colonize. Patients who are left untreated are also at risk for developing systemic inflammatory response syndrome and multiorgan failure.

An insulin drip has the most evidence of a true therapeutic benefit in acute pancreatitis secondary to hypertriglyceridemia. However, the evidence is inconclusive regarding its effects on mortality and in the long term.

## LONG-TERM MANAGEMENT OF HYPERTRIGLYCERIDEMIA

Treatment of hypertriglyceridemia focuses on preventing cardiovascular complications and managing its cause. For patients with a nonfasting triglyceride level above 200 mg/dL, a full lipid panel is recommended to help assess their cardiovascular risk.[18]

The initial management of hypertriglyceridemia focuses on lifestyle changes, which include losing weight, reducing alcohol consumption, increasing physical activity, and avoiding refined carbohydrates and sweetened substances with high levels of fructose.[19] Fructose increases VLDL cholesterol production and promotes triglyceride formation in the liver, which increases the serum triglyceride levels.

The American Heart Association recommends using triglyceride-lowering medications if a patient's level is above 500 mg/dL.[22] Fibrates are the first-line treatment for patients at risk for developing acute pancreatitis.[18] Fibrates can reduce triglycerides by 30% to 50% and also increase HDL, which has cardiovascular protective factors. When using fibrates, one must be cautious with the increased risk of developing cholesterol-based gallstones, which can also trigger acute pancreatitis.[18] In combination therapy with statins, fenofibrate is the preferred fibrate, owing to its low interference with the metabolism of statins.[16] Recent studies also have found that ezetimibe in combination with fibrates helps lower triglyceride and LDL levels.[18]

Niacin can also be used as a monotherapy or in combination with statins to prevent cardiovascular events due to hypertriglyceridemia.[17] Patients taking niacin should have routine liver enzyme panel testing because of the hepatotoxicity risk with niacin. The more common side effects associated with niacin include cutaneous flushing, which can be minimized with aspirin therapy. Niacin should be used with caution in patients with diabetes or a history of gout, owing to its impairment of glucose tolerance and promotion of hyperuricemia.

Omega-3 fatty acids have been shown to lower triglyceride levels by 20% to 50% at doses of 3 to 4 g/day.[23] Even with this decrease in triglyceride levels, no studies have shown a decrease in cardiovascular risk when they are used as monotherapy. When omega-3 fatty acids are combined with statins, studies have shown a 19% decrease in cardiovascular events.

Statins should not be used as monotherapy in severe hypertriglyceridemia because they lack efficacy in lowering triglyceride levels, but

a statin can be used in combination therapies, especially to reduce the cardiovascular risk.[18,23]

Rimonabant is a hunger-reducing medication that assists in weight loss and lowering triglyceride levels.[24] It has been approved by several countries but was rejected by the US Food and Drug Administration because of associated increases in suicidal ideation.

For patients with familial lipoprotein lipase deficiency, alipogene tiparvovec is a gene therapy geared to reverse this deficiency. It has been shown to help increase the production of lipoprotein lipase, which is used to break down triglycerides.[25] It is approved for use in the European Union but not in the United States.

Apolipoprotein C-III (apoC3) has a major influence on triglyceride metabolism. An increase in its function has been associated with hypertriglyceridemia, but loss of function is associated with lower triglyceride levels and fewer cardiovascular events. ApoC3 inhibits lipoprotein lipase from breaking down triglycerides. Antisense oligonucleotide is currently being investigated in clinical trials to function against apoC3 mRNA by inhibiting its translation to decrease the production of apoC3 and inherently lower triglyceride levels.[26]

## CASE CONTINUED

Our patient's long-term treatment is aimed at managing the metabolic syndrome effects by addressing his triglycerides, glucose, and weight. Fenofibrate therapy is initiated with atorvastatin to help lower his triglyceride levels and reduce his cardiovascular risk. He is also prescribed long-acting insulin to better manage his diabetes.

## TAKE-HOME POINTS

- In patients with acute pancreatitis, test for hypertriglyceridemia and manage it appropriately.
- The severity of hypertriglyceridemia is a clinical indicator of the patient's risk for cardiovascular complications and pancreatitis.
- Hypertriglyceridemia-induced acute pancreatitis has a more complicated and severe medical course than acute pancreatitis due to other causes such as alcohol use or biliary obstruction.
- Determine the cause of hypertriglyceridemia and direct long-term treatment to address it.
- Prevent and manage secondary cardiovascular risks related to hypertriglyceridemia. ■

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