A case of carbamazepine-induced pancreatitis

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Abstract
There are many etiologies of acute pancreatitis, including gallstone, hyperlipidemia, drugs, alcohol, trauma, etc. In the clinical work, drug-induced acute pancreatitis cases are so many, but the etiology is often ignored by doctors. This paper reports a case of lipid metabolic abnormalities caused by carbamazepine, which also led to hyperlipidemia, so as to induce acute pancreatitis.

Pancreatitis
Acute pancreatitis is one of clinical common acute abdominal pain. Severe acute pancreatitis in the whole is accounted for 10% to 20% of acute pancreatitis, with a greater risk of disease, with many complications and progress rather quickly, high mortality; and clinical often is with systemic inflammatory response syndrome and multiple organ failure.

Of the various etiologies of abdominal pain, pancreatitis represents a specific pathology with various causes. A rare yet often overlooked cause of pancreatitis known as drug-induced pancreatitis is described by the author herein.

Case study
Chief complaint
A 52-year-old man was with the severe left upper quadrant (LUQ) abdominal pain ten hours.

History of present illness
The patient complained of pain that differentiated between sharp and dull aching in the LUQ abdominal area for 10 hours’ duration. The patient also complained of consistent fevers ranging between 36.2°C (97.2°F) and 37°C (98.6°F). The patient stated the pain increased after any food or oral intake and was mildly relieved with positional change (sitting up). The patient stated she was unable to sleep in bed for the past ten hours secondary to being unable to lie down because of sharp pain. The patient said he had the same symptoms 2 years prior that self-resolved within 7 days [1-6].

Past medical history
In 1995, this patient was with diagnosis of epilepsy, but did not cure. Since 2002, he had been using carbamazepine to control epilepsy, which dosage was one tablet each time, 3 times a day.

Past medical history
Allergies: The patient denied any allergies to food and medications.
Immunizations: The patient’s history of vaccination was unknown.
Past hospitalizations/surgeries: In 2002 for the right occipital arteriovenous fistula in patients was with surgical treatment. In 2006, he was with diagnosis of gallbladder stone, and did cholecystectomy.

Social history
The patient worked at government in a small town. He got married at the age of 23. So far, he had got smoking and drinking 17 years, but his drinking could’ t be more than one hundred milliliter every day, his smoking rarely was more than ten cigarettes every day. He had given up smoking and drinking for thirteen years.

Family history
The patient’s parents had no history of diabetes, hypertension, and epilepsy. The patient was unsure of past familial cancer history.

Review of systems
A review of systems can be found in Table 1.
Physical examination

The patient was awake, alert, and oriented to time, name, and location. The patient was sitting in the chair beside the stretcher secondary to pain when lying down. The patient was actively guarding the left upper quadrant.

Vital signs

Temperature 36.7°C (98.1°F). Blood pressure was 139/85 mmHg and heart rate was normal at 70 beats per minute. The patient’s respiratory rate was 20. Pulse oximetry was 100% on room air. Pertinent physical examination findings are shown in Table 2 [11-15].

Differential diagnoses

Differential diagnoses for left upper quadrant pain and fever include bowel obstruction, splenic infarct, infectious mononucleosis, splenic rupture, gastritis, gastric carcinoma, hiatal hernia, pancreatitis, pancreatic cancer, pancreatic cyst, pyelonephritis, renal calculus, and peripnephric abscess (Collins).

Review of this patient with hyperlipidemic pancreatitis

When patient was with abdominal pain clinic, checked blood triglycerides tendency for 15.3 millimoles per liter. So, we determined the cause of the patients with acute pancreatitis was hyperlipidemia.

Clinical presentation

When this patient arrived at our hospital, he was nausea, vomiting, and unbearable abdominal pain, at the same time there was shit unanswered and less urine volume. The patient admitted to hospital after 1 hour, had epileptic seizures. In addition, this patient was with no other symptoms.

Table 1. Patient review of systems.

<table>
<thead>
<tr>
<th>General</th>
<th>Denied any weight loss or gain.</th>
<th>Cardiovascular</th>
<th>Denied any chest pain, dyspnea on exertion, orthopnea, nocturnal paroxysmal dyspnea, edema, cyanosis, hypertension, heart murmurs, varicosities, plebitis, or claudication.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Derm</td>
<td>Denied any rash, lesions, or masses.</td>
<td>Eyes/Ears/Nose/Mouth/Throat</td>
<td>Denied any headaches, dizziness, visual disturbance, epistaxis, or recent upper respiratory infection.</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Deny any chest pain, dyspnea on exertion, orthopnea, nocturnal paroxysmal dyspnea, edema, cyanosis, hypertension, heart murmurs, varicosities, plebitis, or claudication.</td>
<td>Lung sounds clear to auscultation in all fields.</td>
<td>No shortness of breath, wheezing, stridor, cough, hemoptysis, respiratory infections, or exposure to tuberculosis.</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Denied any shortness of breath, wheezing, stridor, cough, hemoptysis, respiratory infections, or exposure to tuberculosis.</td>
<td>Heart sounds.</td>
<td>Normal at 70 beats per minute.</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Appetite decreased. Intermittent abdominal pain, intermittent nausea, and no recent changes in bowel habits.</td>
<td>No seizures, tremors, incoordination, paraesthesia, difficulties with memory of speech, sensory or motor disturbances, or muscular coordination (ataxia, tremor).</td>
<td>Predominant mood “nervousness” (define), emotional problems, and anxiety.</td>
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<tr>
<td>Genitourinary</td>
<td>No urgency, frequency, dysuria, nocturia, hematuria, polyuria, or oliguria.</td>
<td>No murmur.</td>
<td>No murmurs, varicosities, plebitis, or claudication.</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>No pain, swelling, redness or heat of muscles or joints, limitation of motion, muscular weakness, atrophy, or cramps.</td>
<td>No paroxysmal dyspnea.</td>
<td>No cardiac murmurs.</td>
</tr>
<tr>
<td>Neurologic</td>
<td>No seizures, tremors, incoordination, paraesthesia, difficulties with memory of speech, sensory or motor disturbances, or muscular coordination (ataxia, tremor).</td>
<td>No cardiac murmur.</td>
<td>No cardiac murmurs.</td>
</tr>
</tbody>
</table>

Physical examination

The patient with abdominal slightly raised, abdomen was not soft. The upper abdomen was tenderness obviously, and obvious rebound tenderness. His abdomen was without shifting dullness, and bowel sounds. In addition, this patient was without any other positive signs.

Laboratory evaluation

A variety of diagnostic laboratory tests assist in the diagnosis of both acute and chronic pancreatitis. Amylase has historically been the predominant marker when assessing for assumed pancreatitis. An amylase level 3 times the upper limit of normal has a high specificity for acute pancreatitis.

Computed tomography of the abdomen

This patient did the first Computed Tomography of the Abdomen, when he could feel abdominal pain in the beginning. The first result was a Full Abdomen Scan Computed Tomography, it told us that acute pancreatitis and infiltrating fluid were around 12 bowel swelling thickening, adjacent pancreas below saw enlargement, fuzzy vessels; Splenic nodules was in shadow, properties to be determined; the liver parenchyma density decreased slightly, mild liver fatty might be infiltration, gallbladder did not see, according to the scanning inner bag slightly thickened [16-21].

Ultrasound of the abdomen

This Patients with abdominal exceeds tippeted that: liver slightly enhanced density, pancreas and liver echo echo reduce owe evenly, big spleen; In the spleen had slightly strong echo tubercle - hemangioma. Gallbladder was excision. The right side pleural had effusion. The abdominal had cavity effusion.

Discussion

Carbamazepine-induced Pancreatitis

We aimed to strengthen the follow-up of patients with oral carbamazepine, esp ecially long-term monitoring of blood lipid levels. We hoped that through our efforts could reduce carbamazepine induced acute pancreatitis, at the same time reduce other diseases caused by the carbamazepine.

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References


