The Clue to his Pancreatitis!

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Introduction
Xanthomas are lesions characterized by accumulations of lipid-laden macrophages. They are a common manifestation of lipid metabolism disorders, including hyperlipoproteinemia (HLP). HLPs have been classified according to 5 major phenotypes described in Table 1. These phenotypes are based on specific electrophoretic patterns seen in patients with elevated lipoprotein levels. We present a case of a patient presenting with gallstones and pancreatitis, initially misdiagnosed as gallstone pancreatitis. His skin manifestations in combination with an abnormal lab specimen clued us in to the correct diagnosis.

Case
A 28-year-old male presented to the Emergency department with abdominal pain. He had a CAT scan which showed extensive peripancreatic edema and cholestasis. His amylase and lipase levels were markedly elevated, but his LFTs were normal. A diagnosis of gallstone pancreatitis was made and the surgical service was consulted. Prior to entering the room, we were cautioned that the patient had a “nasty” rash. On examination, we was found to have the lesions shown on images 1 and 2. Subsequently, we were called by the lab stating that the laboratory specimen was lipemic (Image 3), and his triglyceride level was >5000 mg/dL. This sent us to the textbooks, and we were quick to realize that he had tuberoeruptive xanthomas, most likely called by the lab stating that the laboratory specimen was lipemic (Image 3), and his triglyceride level was >5000 mg/dL. This sent us to the textbooks, and we were quick to realize that he had tuberoeruptive xanthomas, most likely

Discussion
The patient was managed in the ICU with IV fluids, pain control, gentamicin/statin, and tight glycemic control for approximately 3 weeks. He recovered from his pancreatitis and was discharged home. The plan is for an eventual cholecystectomy, but for now, he requires tight control of his HLP.

Types of Hyperlipoproteinemia

<table>
<thead>
<tr>
<th>Types</th>
<th>Causes and incidence</th>
<th>Diagnostic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>(Frederickson’s hyperlipoproteinemia, familial hypercholesterolemia)</td>
<td>• Deficient or abnormal lipoprotein lipase, resulting in decreased or absent post-heparin lipolytic activity</td>
</tr>
<tr>
<td>II</td>
<td>(Familial hypercholesterolemia, essential familial hypercholesterolemia)</td>
<td>• Deficient cell-surface receptors that regulate LDL, resulting in increased levels of plasma LDL over joints and pressure points</td>
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<tr>
<td>III</td>
<td>(Familial broad beta disease, chylomicronemia, familial xanthomia tuberosa)</td>
<td>• Unknown underlying defect results in deficient conversion of triglyceride-rich VLDL to LDL</td>
</tr>
<tr>
<td>IV</td>
<td>(Familial hyperbetalipoproteinemia, familial hyperbetalipoproteinemia)</td>
<td>• Usually occurs secondary to alcoholism, diabetes, or emotional disorders</td>
</tr>
<tr>
<td>V</td>
<td>(Familial hyperbetalipoproteinemia, familial hypertriglyceridemia)</td>
<td>• Defective triglyceride clearance causes pancreatic acinar cell damage and is usually secondary to another disorder, such as obesity or nephritis</td>
</tr>
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References