LETTER TO THE EDITOR

Increased pancreatic enzymes and inflammatory bowel diseases: What correlation?

Dear Sir,

In the course of Inflammatory Bowel Disease (IBD), the possibility of an increase of pancreatic enzymes has been described, almost always justified with their release by the inflamed bowel. Unusually an acute pancreatitis presents itself as a pathology associated to the IBD, probably with a common pathogenetic mechanism.

We report the case of a 25 year old man with fever, epigastralgia and a mild blood-free diarrhea. At palpation there was pain in the right lower quadrant of his abdomen. Diarrhea stopped within the first day, while fever and epigastralgia in the third day. His parasitological examination and fecal culture were negative; the blood exams showed rising values of CRP, fecal calprotectin, amylase, pancreatic isoamylase and lipase, without risk factors for acute pancreatitis. His liver and renal functions were normal. Notwithstanding the complete normalization of symptoms and inflammation indices decrease, in the fourth day pancreatic enzymes reached typical rates of acute pancreatitis (Table 1). Transabdominal ecography, echoendoscopy and CT scan didn’t show alterations neither of liver and pancreas, but unexpectedly appeared congestion and thickening of the right colon region, indicative of IBD and likely connected to the patient abdominal pain. Colonoscopy described an active Ulcerative Colitis (UC) confirmed by the histological exam and the elevation of fecal calprotectin up to ten times the normal rates. Tests for HAV, HBV, HCV, thyroid panel and screening for celiac disease were normal and the patient did not present any other clinical manifestation. In the fifth day UC therapy was started according to the guidelines. Two weeks after clinical overture CT showed normal epatic and pancreatic morphologies and the decrease of flogosis signs of right colon. The cholangio-pancreatic magnetic resonance is negative for alterations of bilio-pancreatic ducts; the echoendoscopy described pancreas normal in dimensions and revealed the presence of hypoechogenuous focal nodules likely related to a recent flogosis. Pancreatic enzymes normalized slowly, while the fecal calprotectin later (Table 1).

Our patient at the beginning presented few symptoms sparely indicative either for UC (three or four bowel movements lasted just a day) or for Idiopathic Acute Pancreatitis-IAP-(mild epigastralgia). The laboratory exams oriented merely towards a diagnosis of IAP1 (documented only after the echo-endoscopy) but the instrumental enquiries were realized for studying IAP brought casually to a diagnosis of UC with a mild clinical symptomatology but confirmed by the endoscopic and histological examination and the rising of fecal calprotectin.

Literature describes the possible elevation, in course of IBD,1–3 of serum pancreatic enzymes coming from the bowel inflamed tissues and, according to some Authors,4 this increment (specially of amylase) is directly proportional to the extension and activity of disease. Sometimes serum concentrations of amylase and lipase can rise because of pharmacological treatment of UC.3 However, some Authors5 assert that, in course of IBD, we must play attention when serum pancreatic enzyme values became three times higher than the normal rates, because they are strongly suggestive of a real acute pancreatitis such as accompanying manifestation in IBD. Unusually a paucisymptomatic IBD is diagnosed casually at the clinical onset of a IAP. In our patient the IAP manifested itself more frankly compared with the UC but, above all, they had a completely independent course, since

<table>
<thead>
<tr>
<th>Laboratory parameters</th>
<th>Normal values</th>
<th>2nd day</th>
<th>4th day</th>
<th>8th day</th>
<th>13th day</th>
<th>22nd day</th>
<th>60th day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total amylase</td>
<td>&lt;140 IU/l</td>
<td>156</td>
<td>252</td>
<td>208</td>
<td>195</td>
<td>104</td>
<td>39</td>
</tr>
<tr>
<td>Pancreatic isoamylase</td>
<td>&lt;46 IU/l</td>
<td>131</td>
<td>232</td>
<td>186</td>
<td>167</td>
<td>66</td>
<td>16</td>
</tr>
<tr>
<td>Amylasuria</td>
<td>&lt;450 IU/l</td>
<td>1162</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Lipase</td>
<td>&lt;57 IU/l</td>
<td>445</td>
<td>1036</td>
<td>511</td>
<td>506</td>
<td>124</td>
<td>41</td>
</tr>
<tr>
<td>Fecal calprotectin</td>
<td>&lt;15 mg/kg</td>
<td>28</td>
<td>118</td>
<td>137</td>
<td>251</td>
<td>17.3</td>
<td></td>
</tr>
<tr>
<td>F elastase</td>
<td>&gt;200 μg/g</td>
<td>288</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&gt;500</td>
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<tr>
<td>CRP</td>
<td>&lt;0.5 mg/dl</td>
<td>16.3</td>
<td>9.0</td>
<td>0.4</td>
<td>0.2</td>
<td>0.1</td>
<td>0.2</td>
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</tbody>
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UC quickly developed into clinical remission. Our report confirms the possibility that a real IAP may occur in course of IBD despite the lack of pre-existing risk factors. The differences, both in clinical onset and course, between IAP and UC, confirm that they are two distinct pathologies probably having, if coexisting, just the same etiopathogenic basal disorder.\textsuperscript{5}

Finally, we shouldn't undervalue the eventual rise of pancreatic enzymes in course of IBD but, even if risk factors for acute pancreatitis are lacking, this event should always bring to scrupulously research a coexisting real pancreatic damage (IAP), that should be followed up and treated specifically and regardless of treatment and course of IBD.

References


C.E. Di Bartolo\textsuperscript{*}
V. Leonardi
C. Dell'Aera
E. Puntorieri
C. Zuppardo
G. Mileto

*Department of Internal Medicine and Gastroenterology, University of Messina, Messina, Italy*

*Corresponding author. Tel.: +39 3477072355.
E-mail address: elisabettadibartolo@libero.it* (C.E. Di Bartolo).

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