LETTER TO THE EDITOR

Vanishing Pancreas (or Autopancreatectomy) Following Extensive Pancreatic Necrosis with Lack of Regeneration

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Patients of severe necrotizing pancreatitis who survive the initial phase of inflammatory response may go on to develop walled off necrosis [1]. About 50% of WON may remain asymptomatic and fail to follow up. Hence, the natural history of long standing sterile WON is not well documented. Vitellas et al. have shown that in most of these cases the necrosis remains stable over a period of time and later resorbs leaving a scar [2]. The residual pancreas may regrow to a significant extent [3]. However, there are not many literature reports of longterm followup of such cases. We wish to share our experience with a patient of extensive WON who followed up over 4 years. Patient developed an episode of severe necrotising pancreatitis in January 2008 and was treated conservatively. Imaging done during the course of illness revealed a large WON involving the entire neck, body and tail and most of the head of pancreas (Figure 1). A follow up scan during the next 2 years showed stable WON. The patient followed up thereafter in April 2012 with 2 episodes of significant rectal bleed in the preceding week. A Contrast Enhanced Computed Tomography (CECT) scan to rule out a pseudoaneurysm of the splenic artery revealed striking absence of pancreatic parenchyma. A small remnant of the head/uncinate process of the pancreas was seen. The splenoportal confluence was bare anteriorly (Figure 2). A tiny residual WON was seen in relation to the posterior wall of the descending colon (Figure 3). A Magnetic Resonance Cholangiopancreatography (MRCP) corroborated the CT findings (Figure 4). No definite cause for the rectal bleed could be ascertained either on imaging or on endoscopy.

The capacity of pancreatic parenchyma to regenerate after pancreatitis as well as pancreatectomy has been documented in experimental studies [4, 5]. Increased expression of pancreatic duodenal homeobox -1 or PDX-1, the genetic factor responsible for the proliferation of the ductal progenitor cells and their differentiation into acinar cells is seen in pancreatic tissue after necrotizing pancreatitis [6]. In our patient there is complete absence of any regeneration. Whether this is due to genetic deficiency of PDX-1 or because the remnant head is fulfilling the physiologic requirements is a matter of speculation. The

Figure 1. Initial Contrast enhanced Computed Tomography done in March 2008 showing walled off pancreatic necrosis (short arrow) involving the entire pancreatic parenchyma with minimal sparing of the head (long arrow).

Figure 2. Follow up Contrast enhanced Computed Tomography done in April 2012 showing the bare splenoportal junction with complete absence of the pancreas. Small amount of pancreatic mass seen in region of head (arrow).
reported rates of recovery of exocrine and endocrine function in such patients are variable [7, 8]. Uomo et al. have documented complete recovery of endocrine and exocrine function in conservatively managed patients. Our patient was a controlled diabetic and had no obvious exocrine dysfunction.

Near complete disappearance of the WON is also very surprising. Spontaneous colonic or duodenal fistulisation of infected necrosis with or without massive rectal bleed has been reported [9]. However, spontaneous extrusion of sterile WON with complete disappearance of the WON sac on follow up imaging has never been reported.

Complete resorption of WON without any pancreatic regeneration is possible as seen in our patient. Documentation of such findings adds to our understanding of the disease process and its evolution.

Conflict of Interest

The authors have no conflict of interest to declare

References