

Solid pseudopapillary neoplasm of the pancreas in a young woman

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Introduction

A 30 years old female, with no significant past medical history, presented to our institution with two days of moderate lower abdominal pain, cramping in quality with no radiation and associated nausea and vomiting. Past surgical history revealed a C-section and tubal ligation. Patient denied subjective fever and reported use of nonsteroidal anti-inflammatory drugs (NSAIDs) multiple times daily for the past eight months. Complete blood count showed leukocytosis. Patient was treated with oral phenergan with mild improvement of symptoms. Computed Tomography of the abdomen and pelvis with contrast was requested (Figure 1). Based on the imaging findings, Surgical Oncology was consulted and patient underwent an elective laparoscopic distal pancreatectomy and splenectomy, with subsequent biopsy of the pancreatic parenchyma (Figure 2). The surgery was unremarkable without any complications and minimal blood loss. The patient's condition was stable and appropriate for discharge.

Discussion

Solid Pseudopapillary Neoplasm (SPN) of the pancreas was first described by Frantz in 1959, who named it papillary tumor of the pancreas [1,2]. This entity is most frequently seen in young women with a mean age between 22 and 28 years [3]. The predominant female preponderance has been hypothesized to be secondary to the close proximity of the ovarian ridge to the primordial pancreatic cells in the prenatal development [3,4]. Zhang et al. identified that epidermal growth factor receptor (EGFR), protooncogene tyrosine protein kinase Fyn (FYN), cJUN (JUN), glucagon (GCG), cMyc (MYC) and CD44 are key genes in the progression of SPN [5]. Pancreatic SPN has a slowly growing pattern and the majority of patients are asymptomatic. Clinical manifestations are secondary to mass effect and include nonspecific abdominal pain, nausea and vomiting [6]. Our patient had a history of lower, abdominal pain for the past eight months refractory to NSAIDs oral treatment.

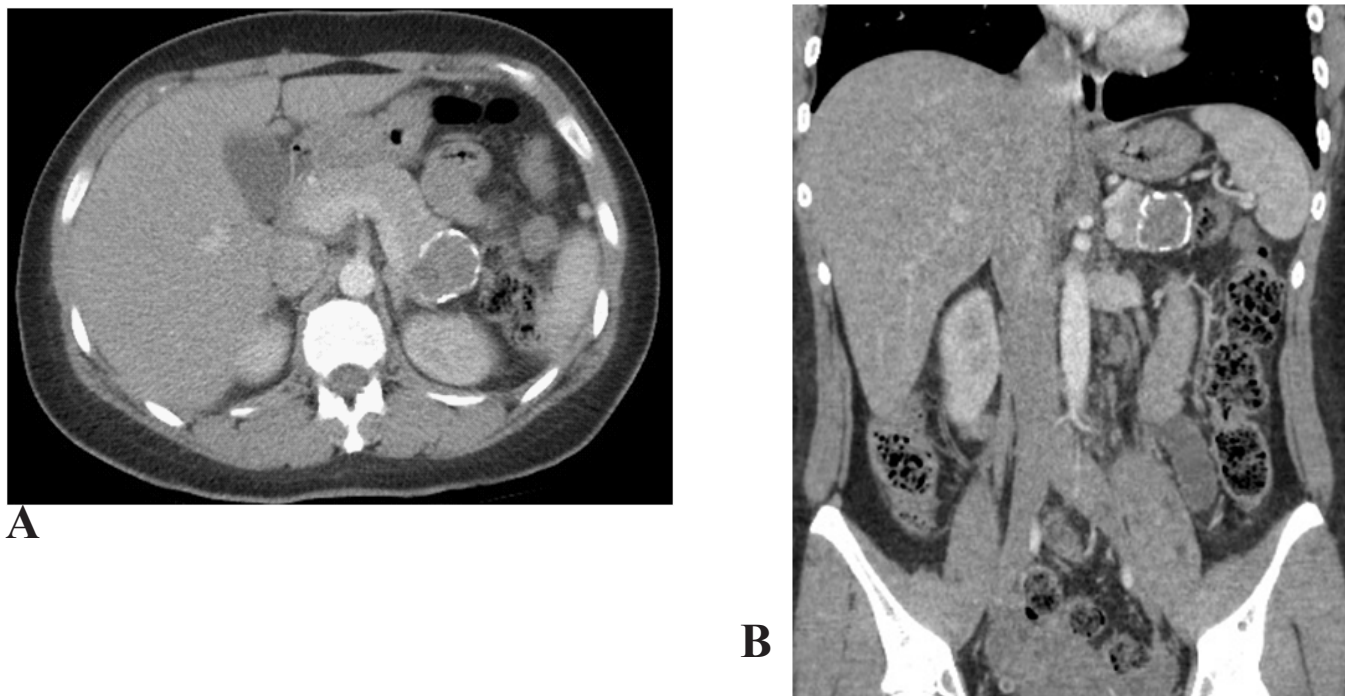


Figure 1: Computed Tomography of the abdomen and pelvis with contrast. (A) Axial and (B) Coronal projections revealed a 3.3 (AP) x 2.8 (TRV) x 3.3 cms hypodense, well defined, peripherally calcified lesion at the pancreatic tail. (arrows). Finding were consisted with a biopsy proven diagnosis of Solid Pseudopapillary Neoplasm of the Pancreas.

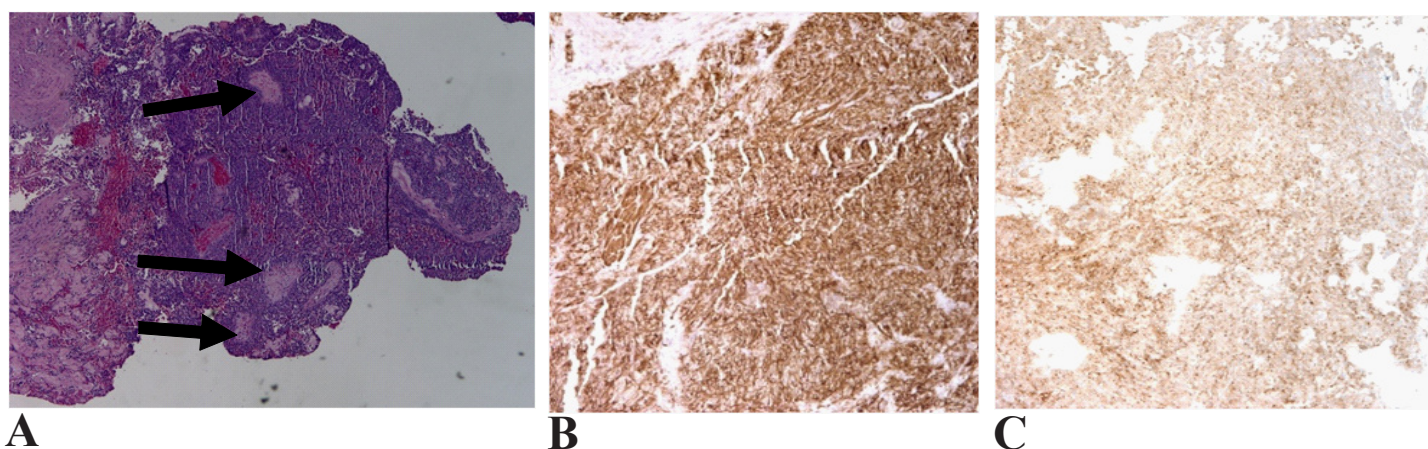


Figure 2: (A) Hematoxylin & Eosin stain of the pancreatic parenchyma demonstrated pseudopapillae arranged radially around blood vessels resembling rosettes in cross section (arrows). Immunostains for (B) CD56 and (C) CD10 are diffusely positive in the pancreatic parenchyma. The histopathological findings were considered most likely to be related with a Solid Pseudopapillary Neoplasm of the Pancreas.

Imaging studies to best characterized pancreatic SPN are Computed Tomography (CT) and Magnetic Resonance Imaging (MRI). Findings on CT include a well-defined, encapsulated mass with areas of necrosis, hemorrhage or peripheral calcifications. SPN may demonstrate similar peripheral enhancement with the surrounding pancreatic parenchyma during arterial and venous phases [6,7]. MR findings include early peripheral heterogeneous enhancement on dynamic imaging and heterogeneous high signal intensity on T2 [2,6,8].

The most common immunohistochemical stains associated with SPN of the pancreas are CD10, CD56, alpha-antitrypsin, Neuron Specific Enolase (NSE) and progesterone [3].

Pertinent differential diagnoses for pancreatic SPN include nonfunctioning islet cell tumor, mucinous cystic neoplasm, microcystic adenoma, calcified hemorrhagic pseudocyst and pancreatoblastoma [3].

Management of pancreatic SPN is surgical resection. Distal pancreatectomy and splenectomy are the modalities of choice in young patients with SPN located in the tail of the pancreas. SPN has a low rate of recurrence in young individuals [8].

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