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was diagnosed as infective endocarditis and empirical intravenous ampicillin (200 mg/kg/day) + gentamicin (3 mg/ kg/day) were initiated. Although she improved clinically in a few days, sudden onset left upper flank pain developed during follow-up. Abdominal ultrasonography revealed hypoechoic regions at subcapsular regions of the spleen compatible with spleen infarct. Repeat TTE showed reduced vegetation size about 9 mm in diameter. Also Streptococcus salivarius, susceptible to ampicillin, was growing in all three blood cultures bottles. Therefore, the antibiotic therapy was restricted to only ampicillin 200 mg/kg/day. While both clinical and laboratory improvements were seen during the fourth week of antibiotic therapy, the vegetation, now 5 mm in diameter, and severe aortic regurgitation still persisted on repeat TTE. So the patient was referred for aortic valve surgery.

Infective endocarditis, an uncommon disease with high morbidity and mortality, is not a uniform disease, but presents in a variety of different forms, varying according to the initial clinical manifestation, the underlying cardiac disease, the microorganism involved, the presence or absence of complications, and underlying patient characteristics. For this reason, infective endocarditis requires a collaborative approach (1).

The most common organisms responsible for subacute bacterial endocarditis (SBE) are relatively avirulent/noninvasive pathogens, the Streptococci viridans (2). Streptococci viridans consisted of S sanguis, S intermedius, S militor, S sanguinis, S milleri, S salivarius, S mutans and others. Streptococci viridans are normal habitants of the oral fluora (3). In addition, Streptococci viridans may also be transiently present on the skin and may contaminate blood cultures. Because these organisms are relatively avirulent and non-invasive, virtually the only infectious disease that is associated with Streptococci viridans is SBE. The strains of Streptococci viridans causing SBE remain highly sensitive to penicillin and beta-lactam antibiotics (4). Monotherapy or combination therapy with a beta-lactam and aminoglycoside (eg gentamicin) is the most commonly used therapeutic approach (2). The infection is most probably due to transmission of S salivarius from the oral flora of the child in a breastfeeding woman during breast engorgement which resulted in bacteraemia and SBE on a previously unknown bicuspid aortic valve. Therefore, SBE should be kept in mind in a postpartum breastfeeding mother with breast engorgement in case of constitutional symptoms and cardiac murmur.

**Keywords:** Breast engorgement, breast feeding, lactating mothers, mastitis, *Streptococci viridans* 

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## Ampullary Pseudotumour: An Endoscopic Clue to Autoimmune Pancreatitis

The Editor.

Sir,

A 63-year old man with Type 2 diabetes mellitus for approximately 10 years presented with epigastralgia, anorexia and weight loss of 16 kg over four months. He had been diagnosed with pancreatitis four months previously, which became inactive after supportive management. His initial blood tests including liver and pancreatic biochemistries were grossly unremarkable. Panendoscopy for the upper gastrointestinal symptoms yielded a smooth-surfaced pseudotumour (black arrow) around the major duodenal papilla (white arrowhead) at the ampulla of Vater (Fig. 1). The subsequent abdominal computed tomography displayed a 1 cm nodular lesion in the ampullary area (black arrowhead), and a diffusely enlarged pancreas (white arrowheads) with an extreme protrusion in the pancreatic head [white arrows] (Figs. 2 and 3). Further diagnostic approaches revealed an elevated serum immunoglobulin (Ig) G4 level of 1470 (reference 3-201) mg/dL and a gamma globulin level of 38.1% (reference 9–18), along with an extensive lymphoplasmacytic infiltration in the specimens of endoscopic ampullary pseudotumour biopsy. The diagnosis of autoimmune pancreatitis (AIP) was established, and oral prednisolone was prescribed which was gradually tapered from a daily dose of 40 mg to 2.5 mg as a maintenance dose. The patient got dramatic clinical and radiographical improvement which lasted during the one-year follow-up period to date.

Autoimmune pancreatitis is an autoimmune-associated entity accounting for 5–6% of chronic pancreatitis (1). It

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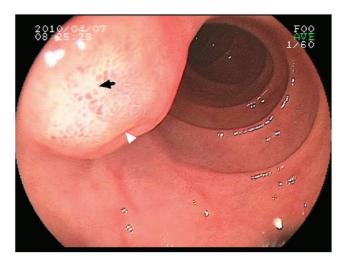


Fig. 1: Panendoscopy in the 63-year old patient showing a smoothsurfaced protrusion (black arrow) around the major duodenal papilla (white arrowhead) at the ampulla of Vater.



Fig. 2: Contrast-enhanced abdominal computed tomography displaying a 1-cm diameter nodular lesion in the ampullary area (black arrowhead), along with an extremely protrusive enlargement in the pancreatic head (white arrows).

commonly presents with jaundice, vague abdominal pain and weight loss. Major duodenal papilla swelling was reported in 24% of AIP patients (2), but protrusion as a pseudotumour is fairly unique (3). Currently, AIP is diagnosed based on a combination of imaging, serological and histologic studies. Differentiating AIP from pancreatic cancer is challenging because of the similar clinical and imaging presentations. However, AIP has a dramatic response to steroid therapy. Immunoglobulin G4-immunostaining of biopsy specimens from major duodenal papilla is supportive of diagnosing AIP

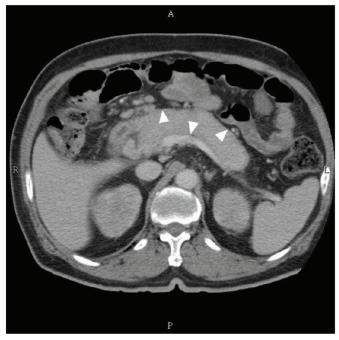


Fig. 3: Contrast-enhanced abdominal computed tomography revealing a diffuse enlargement of the pancreas (white arrowheads).

(2). Accurate diagnosis may avoid unnecessary surgery and delay in the administration of steroid therapy.

**Keywords:** Autoimmune pancreatitis, immunoglobulin G4, major duodenal papilla, panendoscopy

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