

Clinical Utility of PET/CT in Autoimmune Pancreatitis

Vien X Nguyen^{1*}, Cuong C Nguyen¹ and Ba D Nguyen²

¹Department of Gastroenterology and Hepatology, Mayo Clinic, Scottsdale, AZ, USA

²Department of Radiology, Mayo Clinic, Scottsdale, AZ, USA

Autoimmune Pancreatitis (AP) is a rare type of chronic pancreatitis with presumed autoimmune etiology characterized by pancreatic fibrosis and inflammation due to infiltration of Immunoglobulin G4 (IgG4)-positive plasma cells [1]. Beside the pancreas, this entity affects other organs (50-85%) such as the salivary glands, lungs, lymph nodes, bile duct system, kidney, retroperitoneum, and prostate by infiltration with IgG4-positive plasma cells [2]. Hence, AP is the pancreatic manifestation of a novel clinicopathological disorder called systemic IgG4-related sclerosing disease. Serum Immunoglobulin G (IgG) and IgG4 levels are frequently high. However, IgG4 is more than sensitive than total IgG for diagnosing autoimmune pancreatitis. Dense IgG4 plasma cells are observed on histo-immunostaining of the affected organs (Figure 1). When AP appears as a discrete mass (usually at pancreatic head), it can be mistaken for pancreatic cancer. Approximately 3-11% of Whipple procedures were performed on patients with autoimmune pancreatitis who preoperatively were thought to have pancreatic cancers [3].

Positron Emission Tomography (PET) is a functional imaging technique using 2-(¹⁸F)-Fluoro-2-deoxy-D-Glucose (FDG) to characterize cellular metabolism. Because PET imaging lacks of high-resolution anatomy, Computed Tomography (CT) imaging provides

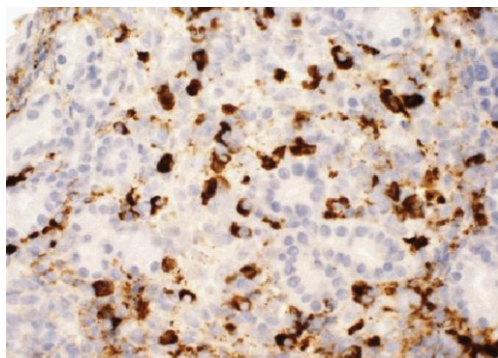


Figure 1: IgG4 immunostaining of the prostate shows a high number of IgG4-positive cells in high power field. (Magnification: 400X).

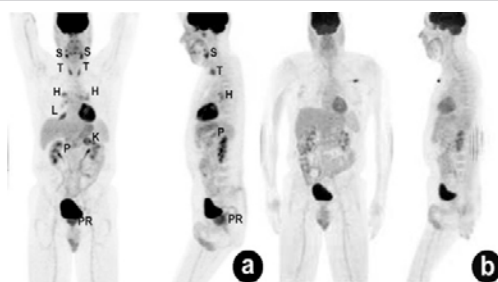


Figure 2: Serial coronal and sagittal pre-corticosterapeutic (a) and post-corticosterapeutic (b) PET Maximum Intensity Projection (MIP) images show complete resolution of the diffuse hypermetabolic lesions of systemic IgG4-related sclerosing disease. The focus at the left upper anterior chest wall represents an artifact (b). S: salivary glands; T: thyroid gland; H: bilateral hilar nodes; L: right middle pulmonary lobe; K: upper pole of the left kidney; P: pancreas; PR: prostate.

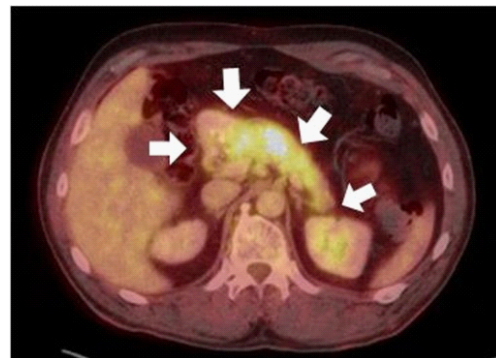


Figure 3: Fused axial PET/CT image shows diffusely hypermetabolic lesion of autoimmune pancreatitis (arrows).

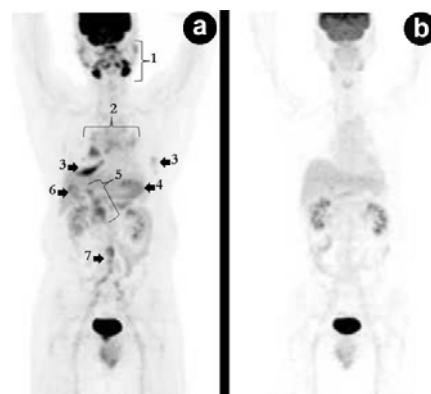


Figure 4: A 52-year-old Chinese male is suspected to have pancreatic cancer. However, PET coronal Maximum Intensity Projection (MIP) shows pattern of systemic IgG4-related sclerosing disease (a). CT-guided biopsy of the left submandibular gland was performed and immunochemical staining was consistent with IgG4-related sclerosing disease. The patient's symptoms resolved with steroid and the scheduled pancreaticoduodenectomy was canceled. One month post-corticotherapy with resolution of all hypermetabolic IgG4-related lesions (b).

1: salivary glands; 2: bilateral peri-hilar regions; 3: bilateral lower lungs; 4: pancreas; 5: porta-hepatitis/common bile duct; 6: gallbladder; 7: abdominal aorta.

***Corresponding author:** Vien X Nguyen, Department of Gastroenterology and Hepatology, Mayo Clinic, 13400 East Shea Blvd, Scottsdale, AZ 85259, USA, Tel: 1-480-301-6990; Fax: 1-480-301-6737; E-mail: vnguy01@gmail.com

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a precise localization of lesions seen on PET imaging. As a result, the first integrated PET/CT scanner was introduced in 1998. FDG is not a tumor-specific tracer; it can accumulate in the inflammatory cells due to their increased glycolytic metabolism. Hence, PET/CT can sometimes distinguish benign conditions (i.e. AP) from malignancies (i.e. pancreatic cancer) by their unique FDG tracer uptake patterns (Figure 2a). Being able to distinguish between the two disorders preoperatively is crucial because invasive surgery can be avoided in patients with AP since corticosteroid is the treatment of choice for patients with AP.

Several small case series have documented the utility of PET/CT in diagnosing and monitoring therapy of AP and its extrapancreatic lesions (Figures 2 and 3). Our group has also demonstrated the clinical utility of integrated PET/CT in selecting extrapancreatic sites for target biopsy in figure 4 [4].

In conclusion, AP is a new disorder which is characterized

by infiltration of pancreas and extrapancreatic sites with IgG4-positive plasma cells. Correct diagnosis of this condition is crucial because corticotherapy is effective. PET/CT may have an impact on differentiating pancreatic cancer from AP. Further research is warranted.

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