

Pancreatic hyperechogenicity on endoscopic ultrasound examination

Yucel Ustundag, Guray Ceylan, Koray Hekimoglu

Yucel Ustundag, Guray Ceylan, Zonguldak Karaelmas University School of Medicine, Department of Internal Medicine, Gastroenterology Clinics, Zonguldak 67100, Turkey
Koray Hekimoglu, Baskent University School of Medicine, Department of Radiology, Ankara 06100, Turkey
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Correspondence to: Guray Ceylan, MD, Zonguldak Karaelmas University School of Medicine, Department of Internal Medicine, Gastroenterology Clinics, Zonguldak 67100, Turkey. gurayceylan@yahoo.com
Telephone: +90-537-2616655 Fax: +90-372-2610155
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Abstract

There is an ongoing discussion on how to diagnose a hyperechogenic pancreas and what is the clinical significance of diffusely hyperechogenic pancreas. Computerized tomography and magnetic resonance imaging are the more appropriate methods to diagnose pancreatic hyperechogenicity when compared with transcutaneous or endoscopic ultrasound examination. More importantly, pancreatic hyperechogenicity may not be a certain indicator of pancreatic fat infiltration. Even if it is true, we do not know the clinical significances of pancreatic fat accumulation. Some suggested that excess fat in the pancreas is associated with chronic pancreatitis. However, several histological studies on human alcoholic chronic pancreatitis did not prove the presence of fatty pancreas in such cases. Thus, except for aging, it is very rare to have truly steatotic pancreas in the absence of certain human diseases.

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Peer reviewers: Alexander S Rosemurgy, MD, FACS, Professor, Department of Surgery, Department of Medicine, University of South Florida, Tampa General Hospital, PO Box 1289, Room F145, Tampa, Florida, FL 33601, United States; Dr. Thiruvengadam Muniraj, MBBS, MD, PhD, MRCP (UK), University of Pittsburgh Medical Center, 100 Chatham Park Drive, Apt 511, Pittsburgh, 15220, United States

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TO THE EDITOR

We read with interest the article by Choi *et al*^[1] entitled "Associated factors for a hyperechogenic pancreas (HP) on endoscopic ultrasound examination". The authors investigated the risk factors for hyperechogenic pancreas on endoscopic ultrasound (EUS). Their study group included 53 cases of HP and the control group consisted of 79 cases having various indications for endosonographic examination with normal pancreas echogenicity on EUS. They noted that HP was significantly associated with fatty liver, male gender, age older than 60 years, hypertension and visceral adipose tissue area (cm²).

Pancreatic fat, readily observed on EUS, is only suspected when an overt HP is noted. However, as the authors in their study noted, mild hyperechogenic pancreas with respect to liver is a normal finding on ultrasound examination. Since the quantitative analysis of pancreatic parenchymal echogenicity was not conducted in their study, how could the authors be sure that pancreatic echogenicity they saw can be "hyper"? The authors also indicated the limitations of their study as the absence of direct determination of the pancreatic fat and visceral fat in pancreatic tissue. It would be unethical to get pancreatic biopsy samples. However, they could estimate the pres-

ence of pancreatic steatosis with the help of computerized tomography (CT) imaging which was already done to estimate the visceral adipose tissue area in all cases in their study. CT can be very helpful for the diagnosis and quantification of the existence of pancreatic steatosis^[2].

We also do not know the clinical consequences of pancreatic steatosis as yet. Some epidemiologic data suggest that obesity is a risk factor for pancreatic cancer development^[3] and that obese patients develop more severe pancreatitis than lean individuals^[4]. Furthermore, postoperative fistula develops more commonly in obese subjects than in lean individuals^[5]. However, in the absence of regular alcohol consumption, the obese patients with increased visceral adiposity are not accepted as having increased risk for chronic pancreatitis. We know that diffusely increased parenchymal echogenicity has not been suggested to be a EUS finding associated either with early or with late stage chronic pancreatic inflammation. Unlike the current evidence for the association between fatty liver and steatohepatitis, there is no similar evidence as yet to suggest that steatotic pancreas progresses to pancreatohepatitis and then to chronic pancreatitis. Indeed, pancreatic hyperechogenicity may not be a certain indicator of pancreatic fat infiltration. The belief that hyperechogenicity of the pancreas indicates the presence of fat in this organ has now been largely abandoned^[6]. Moreover, several histological studies on human alcoholic chronic pancreatitis did not support the presence of fatty pancreas^[7-9].

Thus, it would not be appropriate to diagnose diffuse HP solely on EUS, but CT or Magnetic resonance imaging would be more reliable for such a diagnosis. More importantly, we need to clarify what is the clinical importance of HP on EUS. We are even not sure that HP

represents pancreatic steatosis. Even if it does, we do not know the clinical consequences of pancreatic steatosis.

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