Resolution of hepatic subcapsular steatosis after discontinuation of CAPD

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Introduction

Fatty change of the liver is an extremely common lesion, generally found as a diffuse process involving the entire organ. Focal fatty infiltration appears to be rather rare and can be found in many different patterns [1]. Delivery of insulin in the peritoneal dialysate, instead of the usual subcutaneous route, is a well-established method for diabetic patients on continuous ambulatory peritoneal dialysis (CAPD). It has been suggested that intraperitoneal insulin induces better metabolic control than subcutaneous insulin. However, this means of delivery can increase the risk of peritonitis, deteriorate serum cholesterol profile and expose the subcapsular hepatocytes to a higher concentration of insulin resulting in a unique pattern of subcapsular fatty infiltration [2,3]. Although presence of this unique fatty infiltration does not have any known clinical adverse effect, it is important to differentiate it from other low attenuated lesions including metastatic cancer, primary infiltrative neoplasms, hamartomatous lesions, haematomas and abscess of the liver [1].

The reversibility of fatty infiltration after caloric restriction in obese patients and alcohol withdrawn in patients with alcohol-induced fatty liver have been reported; however, no report exists demonstrating the complete resolution of subcapsular fatty infiltration in patients on CAPD. This report represents the case of a unique subcapsular steatosis induced with intraperitoneal insulin administration, that disappeared completely after discontinuation of peritoneal dialysis.

Case report

A 32-year-old woman had been on a CAPD programme since 1999. She had chronic renal insufficiency, dorsal pancreatic agenesis and type II diabetes mellitus. Insulin was delivered in her peritoneal dialysate between 2002 and 2003. A computed tomography (CT) examination of the abdomen during the period when she was on CAPD revealed characteristic subcapsular fatty infiltration (Figure 1). Because of recurrent pancreatitis attacks, peritoneal dialysis was discontinued and an arteriovenous fistula was created for haemodialysis. The patient had another CT examination of the abdomen a year after she had discontinued CAPD, which revealed complete disappearance of the characteristic fatty infiltration of the liver (Figure 2). At this time, there was no difference with the patient's other risk factors that can supplement the resolution of fatty infiltration, such as diet, physical activity or drugs.

Discussion

This report shows that hepatic subcapsular steatosis (HSS) is a totally reversible process upon withdrawal of the inciting event, which is intraperitoneal insulin administration. This not only helps us correctly diagnose the reversibility of the condition after withdrawal of the intraperitoneal insulin administration but also prevents unnecessary diagnostic work-up. This also may help understand the reversibility of hepatic steatosis due to other causes.

Hepatic subcapsular steatosis is a rare form of fat accumulation in the liver, first described by Wanless and coworkers, in 11 of 12 autopsied diabetic CAPD patients treated with intraperitoneal insulin [4]. High concentrations of glucose and insulin in the peritoneal cavity have been suggested as causal factors in the pathogenesis of HSS. The prevalence of HSS was found to be 62% in diabetic CAPD patients receiving intraperitoneal insulin [5]. The same study revealed that none of the diabetics receiving subcutaneous
insulin and none of the non-diabetic patients exhibited HSS. They also suggested that insulin dosage plays a more important role in the pathogenesis of HSS than does dialysate glucose load [5]. It has been shown that glucose as well as insulin diffuses through the serosal lining of the abdominal cavity—including the liver surface—from dialysis solutions [6]. As with the glucose, the insulin concentration is the highest in the subcapsular hepatocytes and decreases as it is diluted by blood from terminal portal tracts. Insulin facilitates glycogen synthesis. The high glucose load in conjunction with the insulin may result in glycogen accumulation prior to the development of steatosis.

CT is a valuable means for imaging the liver, and diagnosing fatty liver is well established. CT attenuation in the hepatic parenchyma is strongly correlated with the amount of triglyceride in the liver biopsy specimens [1]. CT can also be used to monitor the degree of fatty infiltration during treatment. Fatty infiltration of the liver is reversible and repeated CT scans may be useful in monitoring the disappearance of fat from the liver during recovery. Hepatic subcapsular steatosis has a unique appearance on imaging that makes its recognition relatively easy but it is sometimes difficult to distinguish fatty infiltration of liver from other serious conditions using ultrasonography or CT [7]. Characteristic subcapsular steatosis is described in two different patterns: (i) discrete, nodular, subcapsular low-attenuation lesions, (ii) thin, confluent, subcapsular rings of low attenuation. The degree of hepatic involvement is variable.

No evidence appears in the literature to suggest that HSS is clinically significant. Therefore, the importance of recognizing hepatic subcapsular steatosis is to avoid misinterpreting the findings as resulting from more sinister entities, such as metastatic disease or liver infarction. A correct diagnosis will prevent additional unnecessary investigations and further anxiety for the patient [2]. If deemed necessary, CT or ultrasound-guided needle biopsy can be carried out to confirm the diagnosis.

Regression of subcapsular hepatic steatosis with discontinuation of intraperitoneal insulin has been previously reported in a few cases. In Khalili’s study, two patients with multiple follow-up CT scans and one patient with follow-up sonograms were reported. The decrease suggests that the changes are partially reversible once the subcapsular hepatocytes are no longer exposed to high insulin concentrations. But total clearance of this type of steatosis has not been reported [3].

In our study, a case of subcapsular steatosis is reported, which was proven to recover totally 1 year after the patient started haemodialysis and gave up intraperitoneal insulin. To our knowledge, this is the first report showing that HSS, due to intraperitoneal insulin administration, is totally reversible. This finding supports the fact that fatty infiltration of the liver can resolve completely when the inciting event disappears in certain situations.

Conflict of interest statement. None declared.

References
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