Even though there is no evidence to support the assumption that depletion of glutathione due to fasting will influence DILI in paracetamol overdose [5], there are several variables such as chronic ethanol misuse, use of enzyme-inducing drugs, prolonged fasting and dehydration that may have a causative role in hepatic injury. These “risk factors” are thought to increase the metabolism of paracetamol to the toxic metabolite NAPQI via induction of mixed function oxidases, or decrease hepatocellular glutathione stores, or both [5]. Hepatotoxicity with certain medications such as nitrofurantoin, chlorpromazine, tetracycline, halothane, and diclofenac has been reported more frequently in women [7]. Female sex along with hepatocellular liver damage on admission is suggested to be a risk factor for development of fulminant liver failure [6,7]. The fact that our patient is a female may have made her more vulnerable to DILI.

This case report illustrates that hepatocellular damage could occur even with a ‘sub-toxic’ dosage of paracetamol in vulnerable patients. It is likely that prolonged fasting has made the patient vulnerable in this instance. If more patients with hepatocellular damage following ‘sub-toxic’ doses of paracetamol overdose in the backdrop of prolonged fasting are reported in the future, we may have to consider repositioning the ‘risk line’ at a lower level in the Rumack/Matthew Paracetamol Nomogram, under certain circumstances.

Acknowledgements

We acknowledge the help of Professor Andrew Dawson, Programme Director of the SACTRC for logistical support and Ms. Fathima Shihana Salahudeen, postgraduate research student of the SACTRC for estimating the paracetamol level.

References


A case of peritoneal encapsulation syndrome

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Introduction

Peritoneal encapsulation [PE] is a rare condition characterised by a peritoneal sac covering the small intestine. This was first described in 1868 by Cleland [1]. Most patients with PE develop acute or sub acute small intestine obstruction. We report a patient with asymptomatic PE.

Case report

A 44-year old woman presented with rapid weight gain, generalised weakness and nocturia of 6 months. On examination she had trunkal obesity, a buffalo hump, and hypertension. Abdominal examination was unremarkable except for striae. A clinical diagnosis of Cushing’s syndrome made and was confirmed by abnormally high serum cortisol. A CT scan showed a right adrenal tumour.

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At operation the small intestine was covered in a sac made up of thin peritoneal membrane from duodenum to terminal ileum. The membrane was found inferior to the omentum and transverse colon. It was attached laterally to the parietal peritoneum, medially to the ascending and descending colon, superiorly to the transverse mesocolon and caudally to the posterior parietal peritoneum. The small intestine was visible through the membrane and appeared normal. The right adrenal tumour which was macroscopically malignant was excised. Peritoneal covering of the small intestine was left undisturbed. She made an uneventful recovery. Histology of the tumour revealed a well differentiated adreno-cortical carcinoma.

Unlike this congenital form, the acquired disease (also called abdominal cocoon, or sclerosing encapsulated peritonitis) will have a thick greyish white membrane [2]. It usually presents with intestinal obstruction and has a mortality of 50% [2,4]. This condition is described in association with chronic ambulatory peritoneal dialysis, patients on long term practolol (beta blocker), tuberculous peritonitis, sarcoidosis, ventriculo-peritoneal and peritoneo-venous shunts and retrograde menstruation [2,4,5].

References

Discussion
A membranous covering over the small intestine is a rare condition presenting almost always as intestinal obstruction. This condition is interchangeably described as peritoneal encapsulation, abdominal cocoon or sclerosing encapsulated peritonitis [2,3].

It is a congenital abnormality characterised by the small intestine lying behind an accessory peritoneal membrane. It is believed to be due to abnormal return of the mid gut loop to the abdominal cavity in the early stages of development. The small intestine is thus covered by a peritoneal sac derived from the original dorsal mesentery or from the yolk sac. Patients are usually asymptomatic but may present as acute intestinal obstruction. Rarely, like in our patient, the condition may be detected as an incidental finding. Patients with PE may not show characteristic radiological features if the membrane is very thin as in this patient. We did not disturb the peritoneal covering during surgery because of the risk of secondary adhesion formation and poor prognosis of the large malignant adrenal tumour.