• 58-year-old female came for routine CT surveillance for primary site recurrence of RCC and potential renal transplant.

• **PMH:**
  - RCC resected 10 years ago.
  - DM on insulin.
  - Renal failure on peritoneal dialysis.

*Case submitted by Akeel Al Ali, PGY2*
• What are the imaging findings?

• What is the differential diagnosis? Diagnosis?
POST CONTRAST
Metastasis ?
Liver infarction ?
Steatosis ?
CT FINDINGS

- The renal bed was clear with no evidence of recurrence.

- The liver is hugely enlarged up to 23 cm in size. An enhanced CT shows multiple geographic sub-capsular low attenuation fatty lesions.

- There is no significant enhancement on the arterial nor the venous phases.
Diffuse increased echogenicity in keeping with fatty infiltration.
The overall findings are likely in keeping with subcapsular steatosis.

Metastasis is less likely given the anatomical distribution, fatty density and the absence of enhancement.

Infarction is less likely given the patency of the hepatic vasculature and the vessels were seen crossing the lesions.
Hepatic subcapsular steatosis is a rare and specific form of fatty change in the liver. It is a unique finding in diabetic patients receiving continuous ambulatory peritoneal dialysis (CAPD) and intraperitoneal insulin treatment.

Continuous ambulatory peritoneal dialysis is a well-established method of dialysis in patients with end-stage renal disease.
For patients with renal failure and insulin-dependent diabetes, insulin can be delivered in the peritoneal dialysate rather than by the usual subcutaneous route.

The absorption of insulin by the visceral peritoneum and subsequent transfer into the portal venous system results in a more physiologic delivery of insulin.
**DISCUSSION**

- Disadvantages to intraperitoneal insulin delivery include an increased rate of peritonitis and a worsening serum cholesterol profile. In addition, this route exposes the subcapsular hepatocytes to a higher concentration of insulin than the remainder of the liver.

- Insulin blocks the usual oxidation of free fatty acids in the hepatocytes, leading to preferential esterification into triglycerides, which then accumulate in the cell. The result is a unique pattern of fatty infiltration in a subcapsular location known as hepatic subcapsular steatosis.
REFERENCES

