and judicious fluid therapy are essential in managing patients with hypovolemic hyponatremia.

We report a case who developed severe hypovolemic hyponatremia following the insertion of a percutaneous transhepatic biliary drain for pancreatic head cancer.

CASE
A 66-year-old female with type-2 diabetes mellitus and hypertension was diagnosed with carcinoma of the head of the pancreas. The patient underwent percutaneous transhepatic biliary drainage (PTBD), which involved inserting a pigtail to facilitate biliary drainage. She presented with a 2-day history of altered behaviour and lethargy. Her Glasgow Coma Scale (GCS) was E4V3M5 and serum sodium level was 112 mmol/L. Prior to PTBD insertion, the patient had a baseline sodium level of 133 mmol/L. The patient's PTBD output ranged from 300 mL to 1400 mL daily. Furthermore, the patient's serum osmolarity was 252 mOsm/kg, her urine osmolarity was 331 mOsm/kg, and her urine sodium was 21 mmol/L, which indicated hypovolaemic hyponatremia. She was initially managed with isotonic fluid (NaCl 0.9%) replacement, and her serum sodium improved to 130 mmol/L. However, the patient's IVD regime was not adjusted to account for PTBD drain output, resulting in fluctuating serum sodium levels ranging from 107 to 119 mmol/L. The patient's bile fluid was sent for analysis, which revealed a sodium level of 119 mmol/L. She was restarted on IVD (NaCl 0.9%) for 4 pints to normalise her serum sodium level while waiting for surgery and oncology therapy.

CONCLUSION
Biliary tract loss is a rare cause of hypovolemic hyponatremia. It is important to recognize it in order to plan for appropriate fluid replacement.

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POSTPRANDIAL HYPOGLYCEMIA: A RARE PRESENTATION OF AN INSULINOMA

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Nicholas Ken Yoong Hee, Quan Hziung Lim, Ying Guat Ooi, Tharsini Sarvanandan, Jeyakantha Ratnasingam, Sharmila Paramasivam, Lee Ling Lim, Shireene Ratna Vethakkan
Endocrinology Unit, Department of Medicine, Universiti Malaya Medical Centre, Kuala Lumpur, Malaysia

INTRODUCTION/BACKGROUND
Insulinomas typically present with fasting hypoglycemia, owing to the autonomous secretion of insulin from this neuroendocrine tumor. A presenting complaint of postprandial hypoglycemia in a patient with insulinoma is rare, with a slight male predominance.

CASE
A 66-year-old male with chronic kidney disease (stage G4A2) and interstitial lung disease presented to our centre with two episodes of post-prandial hypoglycaemia, resulting in loss of consciousness. The first occurred 2 hours after breakfast; the second, a month later, occurred 3 hours after breaking fast following a 14-hour fast during Ramadan. During the episode of postprandial hypoglycaemia, he fulfilled Whipple's triad with venous blood glucose 1.0 mmol/L, insulin 42.9 m IU/L and C-peptide 21.1 ng/mL. A 72-hour prolonged fast conducted and terminated at 42 hours revealed a nadir glucose of 2.5 mmol/L but low insulin of 2.0 m IU/L and elevated C-peptide 2.2 ng/mL. Peak ketone was 2.4 mmol/L, consistent with endogenous hyperinsulinism. A mixed meal test triggered hypoglycaemia of 2.3 mmol/L at 210 minutes, with insulin 25.5 m IU/L and C-peptide 17.2 ng/mL. Sulphonylurea screen and insulin autoantibodies were negative.

Both transabdominal and endoscopic ultrasounds revealed a 3.3 x 3.3 cm hyperechoic head of pancreas mass. A fine needle biopsy during EUS was done and immunohistochemistry of the specimen was positive for insulin and synaptophysin, consistent with insulinoma. The patient was started on diazoxide and put on a continuous glucose monitoring system. This also showed low glucose readings predominantly after meals. Unfortunately, he developed severe pneumonia and passed away prior to functional imaging and definitive surgery.

CONCLUSION
Kidney disease does elevate C-peptide levels, which may confound the evaluation of hypoglycemia.2 Clinicians should have a high index of suspicion for insulinoma, even in patients with a history of predominantly postprandial hypoglycaemia when confronted with high C-peptide levels in both fasting and postprandial states.