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HEPATIC GLYCOGENOSIS IN TYPE 1 DIABETES MELLITUS; MORPHOLOGIC DIAGNOSIS

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Hepatic glycogenosis is a rare unrecognised complication of poorly controlled diabetes mellitus type 1. Hepatomegaly with or without abnormal liver function tests, are the common features of both paediatric and adult patients with diabetes mellitus. We report a 25-years old female patient who has a background of diabetes mellitus type 1 and Grave's disease and presented with increased liver enzymes. Her liver biopsy reveals a macrovesicular steatosis in about 30% of the hepatocytes. The hepatocytes are diffusely swollen and contain abundant glycogen which stains deep magenta with PAS and disappear after digestion with diastase. Nuclear pseudoinclusions are present in a number of hepatocytes which also stain positive for glycogen. Megamitochondria-like rounded eosinophilic bodies are identified in some hepatocytes. Focal lobular inflammation is present associated with spotty necrosis in one focus. These findings occur due to excessive reversible accumulation of glycogen in the hepatocytes where it is trapped as a result of a combination of both hyperglycaemia and insulin treatment with consequent liver damage. These changes are reversible if patient's glucose is controlled. Hepatic glycogenosis can be part of Mauriac's syndrome in which the patient has hepatomegaly, abnormal liver enzymes, growth retardation and/or dwarfism, delayed puberty, cushingoid features and hypercholesterolaemia.

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MIXED NEUROENDOCRINE-NON NEUROENDOCRINE NEOPLASM OF THE COLON

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Mixed neuroendocrine non-neuroendocrine neoplasm (MiNEN) is a rare diagnosis in which neuroendocrine component is combined with a non-neuroendocrine component, each of which is morphologically and immunohistochemically recognisable as a discrete component and constitute $\geq 30\%$ of the neoplasm. This case study reports two elderly patients with MiNEN in their

colon, the first presented with caecal tumour on colonoscopy and the second had an obstructing mass in hepatic flexure region. They underwent hemicolectomy which revealed similar morphological findings which showed high grade adenocarcinoma in the superficial part and poorly differentiated components with signet rings and extracellular mucin in the deeper aspects. Areas of rhabdoid differentiation is also present in the first patient. Grade 3 neuroendocrine component is well defined within the deep aspect and constitute about 30% of the tumour. These components are positive for synaptophysin and focally for chromogranin with high Ki67. Perineural and lymphovascular invasion is identified in both cases along with metastasis to regional lymph nodes. MiNEN has an aggressive biological behaviour, mostly driven by its (often high-grade) neuroendocrine component, and a dismal prognosis. In summary, we are discussing two cases of MiNEN with poorly differentiated mucin secreting adenocarcinoma and grade 3 neuroendocrine tumour.

References

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IDENTIFICATION OF SPECIFIC MARKERS TO DIFFERENTIATE PRIMARY PULMONARY CARCINOMA FROM METASTATIC GASTROINTESTINAL CARCINOMA TO THE LUNGS

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Differentiating primary from secondary lung tumours is difficult in poorly differentiated samples. In such cases, the tissue biomarkers CK7, Napsin-A, TTF1, CK20, CDX2, and SATB2 can offer insight into a tumour's origin.^{1–3} Our study aimed to identify the smallest panel that is most sensitive and specific at differentiating primary lung tumours from metastatic GIT tumours to the lungs for the purposes of informing current practices of more time and cost efficient methods. A total of 170 samples were collected, including 140 primary and 30 non-primary lung tumours and staining for CK7, Napsin-A, TTF1, CK20, CDX2, and SATB2 was performed via tissue microarray. The data were then analysed using univariate regression models and a combination of multivariate regression models and receiver operating characteristic (ROC) curves. The results confirmed the ability of individual markers to predict the correct