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Case Report

Radiology Section

Pancreatic Neuroendocrine Tumour Co-existing with Crohn's Disease: A Case Report

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ABSTRACT

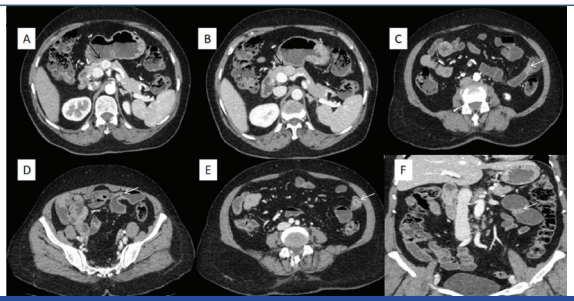
Pancreatic Neuroendocrine Tumours (PNETs) are rare pancreatic neoplasms, while Crohn's disease is a chronic inflammatory bowel disorder characterised by transmural inflammation and skip lesions. The co-existence of these two conditions is exceedingly uncommon. We report the case of a 62-year-old female presenting with non-specific abdominal symptoms, in whom Contrast-Enhanced Computed Tomography (CECT) revealed a hypervascular lesion in the neck of the pancreas with arterial enhancement and venous washout, consistent with a PNET, along with discontinuous small-bowel wall thickening suggestive of Crohn's disease. Histopathological examination of resected ileocolonic specimens confirmed Crohn's disease by demonstrating transmural inflammation and non-caseating granulomas. This case highlights the diagnostic value of contrast-enhanced imaging in detecting concurrent pancreatic and bowel pathology and underscores the importance of histopathology in confirming inflammatory bowel disease.

Keywords: Arterial enhancement, Contrast-enhanced computed tomography (CECT), Insulinoma, Multidisciplinary management, Venous washout

CASE REPORT

A 62-year-old female presented with nonspecific abdominal symptoms along with giddiness for two weeks. The patient had a history of hypoglycaemia for two years. Baseline laboratory investigations revealed haemoglobin 12.6 g/dL, total leukocyte count 7.8×10^9/L, platelet count 265×10^9/L, fasting blood glucose 48 mg/dL, serum creatinine 0.8 mg/dL, blood urea nitrogen 18 mg/dL, serum sodium 138 mmol/L, serum potassium 4.2 mmol/L, total bilirubin 0.7 mg/dL, Aspartate Aminotransferase (AST) 28 U/L, Alanine Aminotransferase (ALT) 25 U/L, alkaline phosphatase 92 U/L, and serum amylase and lipase within normal limits.

Arterial-phase CECT revealed a well-circumscribed, intensely enhancing lesion measuring approximately 2.5 cm in the neck of the pancreas [Table/Fig-1a]. On portal venous phase imaging, the lesion demonstrated relative contrast washout [Table/Fig-1b], consistent with a hypervascular pancreatic mass suggestive of a PNET. There was no associated pancreatic ductal dilatation or peripancreatic inflammatory change. Additionally, CECT demonstrated multiple discontinuous segments of small-bowel wall thickening with prominent mucosal hyperenhancement, forming skip lesions across different portions of the small intestine [Table/Fig-1c-f]. No radiologic evidence of bowel obstruction, abscess, fistula, or perforation was identified.



[Table/Fig-1]: Contrast-Enhanced Computed Tomography (CECT) findings: (a) Axial arterial-phase CECT shows a well-defined, intensely enhancing lesion in the neck of the pancreas (black arrow); (b) Axial portal venous-phase CECT demonstrates relative contrast washout of the pancreatic lesion (black arrow), consistent with a hypervascular tumour; (c-e) Axial CECT images show multiple discontinuous segments of small-bowel wall thickening with mucosal hyperenhancement (white arrows), forming skip lesions; (f) Coronal CECT image further delineates segmental small-bowel involvement with mucosal hyperenhancement (white arrow), suggestive of active Crohn's disease.

Based on the characteristic imaging findings, a provisional diagnosis of pancreatic neuroendocrine tumour with concomitant inflammatory bowel disease was made preoperatively. Crohn's disease was suspected radiologically because of the presence of skip lesions and segmental bowel wall thickening.

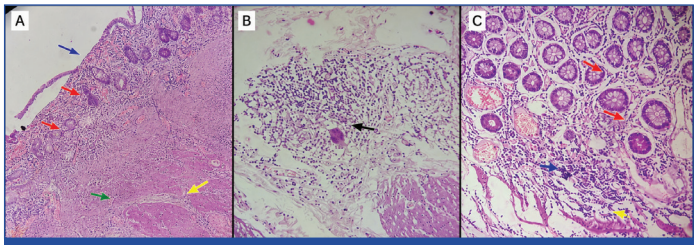
Given the history of recurrent hypoglycaemic episodes for two years, biochemical evaluation was performed. During symptomatic hypoglycaemia, plasma glucose was 38 mg/dL, serum insulin was 18.6 µIU/mL, and C-peptide was 3.8 ng/mL, findings consistent with endogenous hyperinsulinaemic hypoglycaemia. A fasting test demonstrated persistent insulin secretion despite hypoglycaemia, with plasma glucose falling to 42 mg/dL while serum insulin remained elevated at 12.4 µIU/mL, supporting the diagnosis of insulinoma.

Surgical intervention was undertaken primarily for management of the suspected insulinoma causing recurrent hypoglycaemia. The patient subsequently underwent surgical intervention, during which enucleation of an insulinoma was performed. Intraoperatively, a 1.5×1.5 cm hard tumour was identified in the body of the pancreas, reflecting differences between radiological and intraoperative assessment, since imaging showed an approximate 2.5 cm lesion. The apparent discrepancy in lesion location reflects differences in anatomical description between radiological and intraoperative assessments. As the lesion was located at the neck–body junction of the pancreas, both descriptions refer to the same lesion.

Following enucleation, the pancreatic duct was intact, and no pancreatic juice leak was identified; the remaining pancreas appeared soft. Additionally, the terminal ileum was noted to contain multiple palpable strictures with mesenteric fat wrapping, suggestive of Crohn's disease.

In addition to pancreatic tumour enucleation, the patient underwent ileocolic resection involving approximately 15 cm of terminal ileum and the adjacent caecum and ascending colon because of multiple strictures and gross inflammatory changes. The resected specimen was subsequently submitted for histopathological examination.

Microscopic examination revealed chronic active inflammatory bowel disease with marked crypt architectural distortion, dense lymphoplasmacytic infiltration of the lamina propria, focal cryptitis, and occasional crypt abscesses [Table/Fig-2c]. Inflammation



[Table/Fig-2]: Histopathological features of Crohn's disease: (a) Section showing transmural inflammation with extension beyond the mucosa into the submucosa (red arrows), accompanied by fibrosis (green arrow) and vascular congestion (yellow arrow). The mucosal surface demonstrates inflammatory involvement (blue arrow); (b) Well-formed non-caseating epithelioid cell granuloma within the lamina propria (black arrow), composed of epithelioid histiocytes without central necrosis; (c) Distorted colonic crypt architecture (red arrows) with increased lamina propria cellularity (blue arrow) and focal cryptitis (yellow arrow), consistent with chronic active colitis.

extended into the submucosa, indicating transmural involvement [Table/Fig-2a]. Well-formed non-caseating epithelioid granulomas were identified within the lamina propria, confirming the diagnosis of Crohn's disease [Table/Fig-2b]. Histopathological examination revealed uniform round-to-oval cells arranged in nests and trabeculae separated by delicate fibrovascular stroma. The tumour cells exhibited finely granular eosinophilic cytoplasm and characteristic salt-and-pepper chromatin, with minimal nuclear atypia. The mitotic index was low (<2 mitoses/2 mm^2, approximately 10 high-power fields), consistent with a low-grade pancreatic neuroendocrine tumour. Gross examination demonstrated a well-circumscribed tumour measuring 1.5 × 1.5 cm.

The postoperative course was uneventful. The patient experienced complete resolution of hypoglycaemic symptoms following insulinoma enucleation. No pancreatic fistula or other major postoperative complications were observed. At six months follow-up, the patient remained symptomatically improved, with no radiological evidence of tumour recurrence.

DISCUSSION

The PNETs are rare neoplasms arising from the endocrine cells of the pancreas, comprising approximately 2% of all pancreatic tumours [1]. These tumours exhibit a wide range of clinical behaviours, from indolent to highly aggressive, and can be either functional (secreting hormones causing distinct syndromes) or non-functional [1]. In contrast, Crohn's disease is a chronic inflammatory bowel disease characterised by transmural inflammation, often presenting with skip lesions throughout the gastrointestinal tract [2].

Although neuroendocrine tumours, particularly ileal NETs, have been reported in association with inflammatory bowel disease, the co-existence of PNETs with Crohn's disease remains poorly documented and not well established [3,4].

One proposed mechanism is that chronic inflammation in Crohn's disease may promote tumourigenesis through sustained cytokine release, oxidative stress, immune dysregulation, and mitochondrial dysfunction, thereby creating a microenvironment favourable for neoplasm development, including neuroendocrine tumours [5]. Alternatively, PNETs may influence gastrointestinal inflammatory pathways, or both conditions may develop independently due to shared genetic or environmental factors. To date, however, there is no direct evidence supporting a causal relationship.

Diagnostic imaging, particularly CECT, plays a pivotal role in identifying both conditions. PNETs characteristically demonstrate intense arterial-phase enhancement with subsequent portal venous washout, reflecting their hypervascular nature [6]. In Crohn's disease, CECT effectively depicts skip lesions, bowel wall thickening, and mucosal hyperenhancement, consistent with active inflammation [3].

Although the imaging findings strongly suggested Crohn's disease, differential diagnoses for segmental bowel wall thickening and skip lesions include intestinal tuberculosis, ischemic enteritis, NSAID-induced enteropathy, infectious enterocolitis, and lymphoma [7]. In the present case, the presence of transmural inflammation together with well-formed non-caseating granulomas and characteristic architectural distortion favoured Crohn's disease over these alternative diagnoses.

Nuclear imaging plays a key role in the management of PNETs by enabling functional localisation and staging through somatostatin receptor imaging (e.g., Ga-68 DOTATATE PET/CT). It also helps assess tumour burden, detect metastases, and determine suitability for Peptide Receptor Radionuclide Therapy (PRRT) [8].

A study by Bassoff L et al., 2025, highlighted that non-functioning PNETs often present silently and are frequently detected incidentally on imaging due to the absence of hormonal symptoms. The study emphasised the pivotal role of radiological modalities in diagnosis and noted that surgical resection remains the mainstay of definitive management, with careful follow-up required due to variable clinical behaviour [9].

Management of patients with co-existing PNET and Crohn's disease requires a multidisciplinary approach. Surgical resection is the primary treatment for PNETs, while Crohn's disease is managed medically using anti-inflammatory agents, immunomodulators, and biologic therapies [1,10]. Given the rarity of this association, individualised treatment strategies are essential to optimise patient outcomes.

CONCLUSION(S)

This case illustrates the rare coexistence of a PNET and Crohn's disease. CECT enabled early identification of the hypervascular pancreatic lesion and inflammatory bowel changes, while histopathology confirmed Crohn's disease through non-caseating granulomas and transmural inflammation.

REFERENCES

[1] Sulciner ML, Clancy TE. Surgical management of pancreatic neuroendocrine tumours. Cancers (Basel). 2023;15(7):2006. Doi: 10.3390/cancers15072006. PMID: 37046665; PMCID: PMC10093271.
[2] Parray FQ, Wani ML, Bijli AH, Thakur N, Irshad I, Nayeem-ul-Hassan. Crohn's disease: A surgeon's perspective. Saudi J Gastroenterol. 2011;17(1):6-15. Doi: 10.4103/1319-3767.74430. PMID: 21196646; PMCID: PMC3099084.
[3] Abuassi M, Peles S, Alfar B, Obed A. Neuroendocrine tumour of the terminal ileum presenting as refractory Crohn's disease: A diagnostic challenge. ACG Case Rep J. 2025;12(10):e01861. Doi: 10.14309/crj.0000000000001861. PMID: 41112852; PMCID: PMC12528589.
[4] Azzam N. A rare case of rectal adenocarcinoma and small-bowel neuroendocrine Tumour in a young patient with long-standing Crohn's disease: A case report. Journal of Nature and Science of Medicine. 2021;4(2):209-11. Available from: https://doi.org/10.4103/JNSM.JNSM_114_20.
[5] Haque PS, Kapur N, Barrett TA, Theiss AL. Mitochondrial function and gastrointestinal diseases. Nat Rev Gastroenterol Hepatol. 2024;21:537-55. https://doi.org/10.1038/s41575-024-00931-2.
[6] Battistella A, Partelli S, Andreasi V, Marinoni I, Palumbo D, Tacelli M, et al. Preoperative assessment of microvessel density in nonfunctioning pancreatic neuroendocrine tumours (NF-PanNETs). Surgery. 2022;172:1236-44. Available from: https://doi.org/10.1016/J.SURG.2022.06.017.
[7] Bakkaloğlu OK, Çelik AF. Differential diagnosis of inflammatory bowel disease. J Enterocolitis 2025;4:20-27. https://doi.org/10.14744/Jenterocolitis.2025. 82883.
[8] Prosperi D, Gentiloni Silveri G, Panzuto F, Faggiano A, Russo V, Caruso D, et al. Nuclear medicine and radiological imaging of pancreatic neuroendocrine neoplasms: A multidisciplinary update. J Clin Med. 2022;11:6836-42. Available from: https://doi.org/10.3390/jcm11226836.
[9] Bassoff L, Kersey K, Debus I, Glotzer D. The silent presentation of a non-functioning pancreatic neuroendocrine tumour: A case report. Cureus. 2025;17:e89256-61.
[10] Cushing K, Higgins PDR. Management of Crohn disease: A review. JAMA. 2021;325:69. Available from: https://doi.org/10.1001/JAMA.2020.18936.

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