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RESEARCH ARTICLE

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COLLISION TUMOUR OF PANCREAS: A CASE REPORT WITH REVIEW OF LITERATURE

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ABSTRACT

Collision tumours are rare tumours; defined as tumours composed of two histologically distinct neoplasms located in the same organ or anatomical site. According to the World Health Organisation (WHO) histological classification, collision tumours include at least two different malignant components, without mixed or transitional area. Collision tumours are different from the mixed tumours formed by more than one type of neoplastic tissue, but, having the same histological origin, as shown by the transitional area. Pathogenesis of this rare entity remains unclear, although there are several proposed hypotheses. Preoperative diagnosis of collision tumours is difficult because of the lack of specific clinical symptoms and radiological features. Histological diagnosis also poses a significant problem and Immunohistochemistry needs to be done in all the cases to reach the conclusion. We, hereby, report a case of 60 year male having Pancreatic head Neuroendocrine carcinoma and Ductal Adenocarcinoma collision tumour. Based on our case report and review of literature, we conclude that collision pancreatic cancer is a very uncommon tumour which poses a significant challenge in diagnosis and treatment.

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INTRODUCTION

Collision tumours are rare clinical entity. According to WHO histological classification, collision tumours include at least two histologically distinct tumour types occurring at the same anatomic site without mixed or transitional area.⁽¹⁾ Collision tumours are described in various locations – gastric cardia, cervix, appendix, urinary bladder, liver, lung, oral cavity, thyroid, ovary, bile duct. Usually a collision is seen between carcinoma and sarcoma/lymphoma. It is rarely between two types of carcinoma.⁽²⁾ Diagnosing collision tumours is challenging and to differentiate them from the more common mixed tumours is difficult.

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This case report emphasizes the importance of clinical history, radiological findings, histopathological and immunohistochemical analyses in diagnosing a tumour composed of two/multiple malignant morphologies.

CASE REPORT

A 60 year male patient presented with weight loss, epigastric pain and fatigue in surgical gastroenterology OPD. He had no past history of pancreatic disorders. He was subjected to investigations: pancreatic amylase – 265IU/l (normal range, 40-129IU/l); Blood sugar – 150mg/dl (70-109 mg/dl); HbA1c, 7.2% (4.6-6.2%); S.CEA – 4.4ng/ml (<5.0ng/ml); CA 19-9 levels - 139.9U/mL (<37U/mL). CT abdomen showed dilated main pancreatic duct with a large solid lesion in the pancreatic head measuring 4.5cm in diameter. EUS guided FNA from the lesion was performed which showed adenocarcinoma. Under the diagnosis of pancreatic head adenocarcinoma, pancreaticoduodenectomy with regional lymph node dissection was performed with curative intent.

Gross examination of the specimen revealed a solid tumour in the head of pancreas measuring 6 x 5 x 3.5 cm. The tumour was involving the duodenal wall and CBD. Multiple nodes were resected from the peripancreatic tissue. Microscopic examination showed a tumour composed of two cell population (figure 1,2). 10% of the tumour was composed of well formed glands lined by malignant cells (figure 3) whereas 90% of the tumour revealed solid nests and cords of tumour cells with round nuclei, clumped chromatin, prominent nucleoli, moderate eosinophilic cytoplasm and brisk mitotic activity (figure 4) involving the head of pancreas, infiltrating the CBD and duodenal wall.

There was a clear demarcation between the two types and no intermixed/transition zone was identified. Lymphovascular and perineural invasion were identified. Out of the total 18 lymph nodes resected from the peripancreatic soft tissue, 6 were positive for tumour metastasis with extranodal extension. On Immunohistochemistry, 90% tumour component was CD 56 positive, Synaptophysin positive and

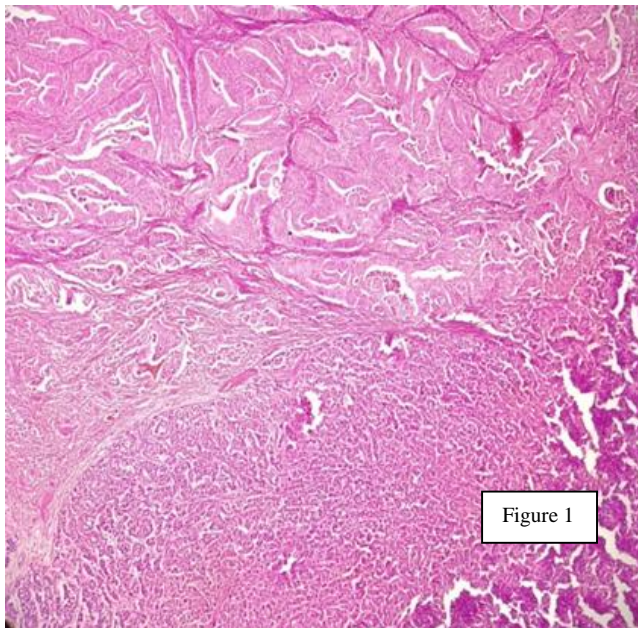


Figure 1

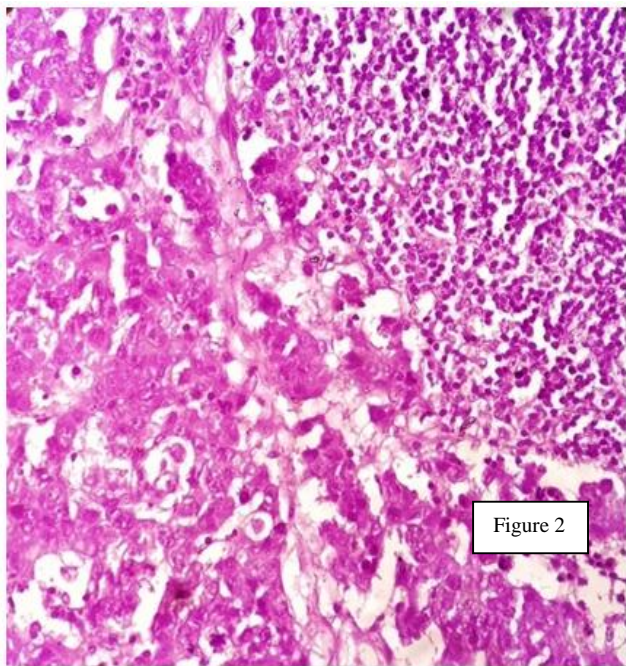


Figure 2

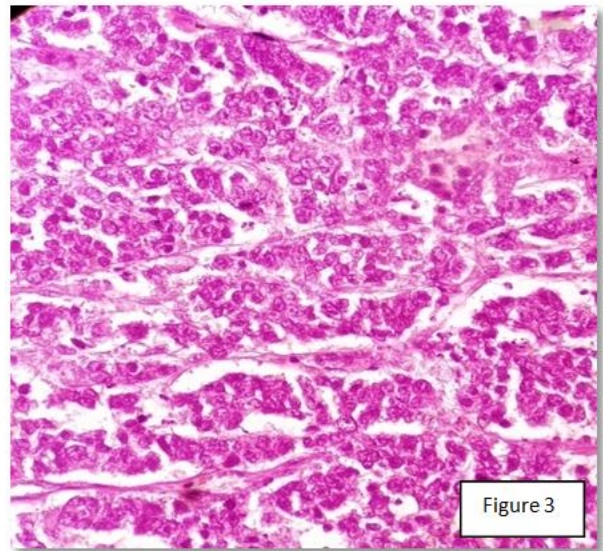


Figure 3

Figure 3: H&E stained section shows solid nests and cords of tumour cells with round nuclei, clumped chromatin, prominent nucleoli, moderate eosinophilic cytoplasm and brisk mitotic activity (x400)

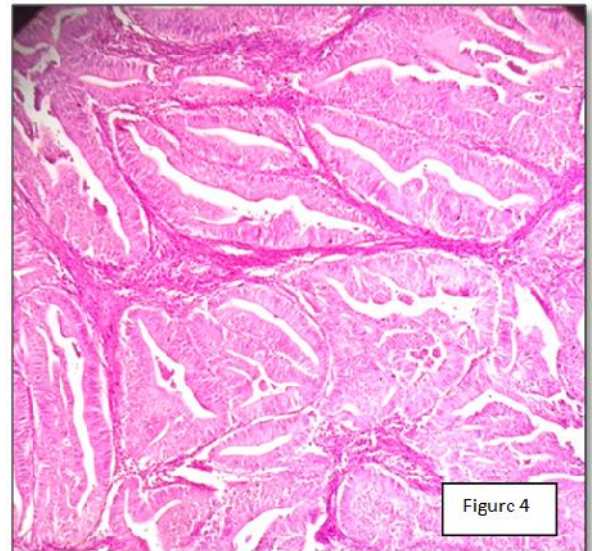


Figure 4

Figure 4. H&E stained section shows well formed glands lined by malignant cells (x400)

Chromogranin positive, Ki 67 was 70-80%, PAS negative, CK, CLK, CK 20, CK19, CA19.9 and CEA were faint positive. Glandular 10% component showed positivity for CK, CLK, CK 20, CK19, CA19.9 and CEA. Ki67 was 30-40%. CD 56, Synaptophysin and Chromogranin were negative. Final histopathological diagnosis of Collision tumour with Neuroendocrine carcinoma and Ductal Adenocarcinoma – Head of Pancreas with invasion into ampulla and duodenum was made. Adenocarcinoma component was conventional type, well differentiated and involved the adjoining duodenal wall. No mixed or transitional area between the two tumour types was identified. All lymph nodes showed metastasis from Neuroendocrine Carcinoma. The tumour was categorized as pT3N1. Patient had uneventful postoperative period and was discharged on 6th post op day. Keeping neuroendocrine carcinoma as more aggressive component, the patient was advised chemotherapy with Cisplatin and Etoposide. However, he refused to continue the chemotherapy. 4 months later he came with weight loss and abdominal distention. USG scan showed multiple liver SOLs. USG guided FNA from liver SOLs revealed Metastatic deposits from Neuroendocrine Carcinoma. A week later patient expired.

Figure 1&2: H&E stained sections show an interface between two Histologically different tumours with no intermixed area (x100) (x400)

DISCUSSION

The pathogenesis of collision tumours remains controversial. Various hypothesis have been proposed – (1) Incidental occurrence, especially in tumours originating from neighbouring tissues or chance opposition of two unrelated tumours, (2) Simultaneous proliferation of two different cell lines, (3) Common origin from pluripotent precursor stem cell that differentiates into two components, (4) A carcinogenic agent may interact with different tissues, inducing different tumours, (5) Growth of second primary tumour at the site of metastasis by oncogenic growth factors produced by metastatic tumour, (6) Alteration in microenvironment, such as angiogenesis and inflammation, by primary tumour could facilitate the growth of metastases from a second primary tumour from another organ.⁽²⁻⁷⁾

Collision tumours should be differentiated from composed or mixed tumours like adenosquamous carcinoma of esophagus or cervix. Mixed tumours are formed by more than one type of neoplastic tissue with histological characteristics of different primary tumours. However, they have the same histological origin, as shown by transition areas.⁽⁸⁾ Collision tumours of pancreas are rare; sporadic cases have been reported in the literature.⁽⁹⁾ Preop diagnosis is difficult because of the lack of specific symptoms and radiological features. Radiological examination is useful in typing certain types of tumours.

Patterson et al, retrospectively analysed radiological findings in histologically proven cases of collision tumours that might help in their preoperative diagnosis.⁽¹⁰⁾ Neuroendocrine tumours are generally well circumscribed lesions that appear hyperenhancing on CT. Adenocarcinomas are typically hypovascular lesions on CT. CT findings depend on the ratio of the different histological types. However, diagnosing Collision tumours only by means of radiological examination is difficult. EUS-FNA, CT guided FNA, USG guided FNA are useful techniques. FNA has limitations for making a definite diagnosis, as FNA cytology cannot cover the entire tumour. The confirmed diagnosis mainly depends on the histopathological and immunohistochemical analyses from a surgically resected specimen. Histopathologist, surgeons, and oncologists need to be aware of existence of such rare collision tumours. They will dictate appropriate treatment strategies dependent on individual biological aggressiveness of each of the tumour components. The types of components, most aggressive component and the stage of tumour determine the prognosis.

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