Serous Microcystic Adenoma to Adenocarcinoma of Pancreas—A Case Report

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Introduction

Cystic neoplasms account for approximately 10 to 15 % of all cystic lesions of the pancreas and malignant cystic lesions form less than 1 % [1–3]. Early identification of asymptomatic cases is now possible due to easy availability of better imaging techniques. These neoplasms are divided into two major groups: serous cystadenomas and mucinous cystic neoplasms [2–5]. Unlike mucinous cystadenomas which are known to be precancerous, malignant transformation of serous cystadenomas is not common. Only 25–30 cases have been reported so far in the literature [3–7]. We report a case of 57-year-old female who progressed from benign pancreatic serous microcystic adenoma (SMA) to pancreatic adenocarcinoma in a follow-up span of 9 months.

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Case Report

A 57-year-old female, who was previously healthy, presented with upper abdominal dull aching and non-radiating pain since 2 months. There was no associated history of fever or jaundice. General physical examination showed mild pallor and per abdominal examination revealed nodular mass in right lumbar and hypochondriac region measuring 8×6 cms, which was mobile on respiration.

Ultrasound abdomen showed mixed echogenic lesion in the head of pancreas extending from umbilicus to the right hypochondrium with gall bladder infiltration and dilated intra hepatic biliary radicles. Computed tomography (CT) of abdomen showed inhomogenous enhancing lesion in uncinate process/head of pancreas measuring 6x5cm with dilated intra hepatic biliary radicles and widened C loop of duodenum. [Fig. 1] A radiological differential diagnosis of uncinate process malignancy and/or malignant gastro intestinal stromal tumor was offered.

Aspiration cytology from the lesion yielded sparse material. Hence, ultrasound guided core biopsy was performed which showed lesion with multiple cystic spaces lined by cuboidal cells with abundant eosinophilic secretion. [Fig. 2] Cells were uniform. [Fig. 3] There was no evidence of malignancy in the core biopsy. Hence the diagnosis of serous microcystic adenoma-pancreas was made and surgical excision was advised, which the patient refused. Patient was symptomatically treated and regularly followed up for 9 months. Repeat CT scan showed progressive growth with gall bladder invasion and multiple liver lesions. [Fig. 4] Follow up core biopsy appeared similar to previous biopsy but with few atypical glands with hyperchromatic nuclei within the desmoplastic stroma. [Fig. 5 and 6] Periodic acid Schiff (PAS) showed focal cytoplasmic positivity.

Fig. 1 Axial section of the CT abdomen showing inhomogeneously enhancing lesion (★) in the uncinate process and head of pancreas. [Initial presentation]

[Fig. 7] Subsequently patient developed enlarged supra clavicular lymph nodes. Lesional and liver aspirate yielded adenocarcinoma with metastatic deposits respectively. [Fig. 8] The patient was started on therapy and palliative analgesia and remains under regular follow up.

Discussion

Pancreatic tumours generally have a poor prognosis; the majority of these being ductal adenocarcinomas [1, 3, 4]. The pancreatic cystic neoplasms are much less common, comprising only 1–2 % of all pancreatic exocrine tumors [2, 5–7]. SMA is the most common encountered tumour amongst serous cystic neoplasms. They occur more commonly in women and manifest with non-specific symptoms. Up to one third of the patients are asymptomatic and they are diagnosed incidentally during abdominal imaging performed for unrelated reasons [3, 5, 7–9]. SMA may occur in any part of the pancreas, but more commonly in the head and tail [2, 3]. The prevalence

Fig. 2 Core biopsy of the lesion showing multiple cystic spaces lined by cuboidal cells with abundant eosinophilic secretion (H&E, X100)

Fig. 3 Core biopsy showing multiple cystic spaces lined by cells with uniform nuclei. (H&E, X400)

of cancer among serous cystadenomas is reported to be 3 % since the first case reported in the literature in 1989 [7–10].

SMA is composed of multiple small cysts separated by thin septa and often manifest a honeycomb appearance on imaging [2, 3]. The pre-operative differentiation between

Fig. 4 Oblique reformatted view of the CT images showing pancreatic mass (★) with gall bladder infiltration (➡). [On follow up CT after 9 months of initial presentation]

Fig. 5 Repeat core biopsy showing atypical glands with hyperchromatic nuclei (H&E, X400)

benign SMA and malignant serous cystadenocarcinomas cannot be made and has not been done in any of the previous 25–30 reported cases. Histology is similar in both with the main difference being that of invasion (gross or microscopic) to differentiate between benign and malignant counterpart [3–8].

None of the methods available for diagnosis including clinical assessment, imaging, fluid analysis, cytology or histopathology can differentiate between the various lesions of the pancreas [2–4]. Studies have shown that centres experienced in pancreatic imaging are more accurate in differentiating various lesions. Although histology can differentiate between various forms, there is overlapping of features between benign and malignant tumors, which makes it difficult. Biochemical analysis of cyst fluid may include amylase and some tumor markers (eg: carcino embryonic antigen, NB/70 K, CA 72–4, CA 125, CA 15–3 tissue polypeptide antigen and PS 2 protein). Amylase content of fluid can differentiate between serous and mucinous cystic tumors. Cytological analysis is diagnostic in about 50 to 60 % of cases [3–9].

Serous cystadenomas almost invariably have a benign course. Therefore, some authors suggest that SMA may be conservatively managed and closely followed-up in asymptomatic patients without ductal [6–10]. However, conservative

Fig. 6 Core biopsy showing atypical glands with hyperchromatic nuclei within the desmoplastic stroma (H&E, X400)

Fig. 7 Histopathology showing lesion with strong PAS positivity (H&E, X400)

management has the risk of progressive growth of the tumour and development of complications such as haemorrhage, erosion into adjacent structures and gastrointestinal or biliary obstruction [6–10].

Clinicians should be aware of the possibility of malignant transformation in serous cystic neoplasms and should maintain a high index of suspicion. The worrying features include rapid enlargement of the mass, the onset of new symptoms or worsening of existing symptoms. In these cases, resection may be indicated, despite the lack of objective evidence for malignancy obtained from preoperative imaging, endoscopy, and biopsies [8, 9]. Symptoms of the patient, the accuracy of the preoperative diagnosis, safety of the resection, and risks of conservative management play a role in the decision of the treatment of pancreatic tumors [6, 7]. Surgically resected serous cystic neoplasms of the pancreas are typically seen in asymptomatic women and are predominantly benign. Most are resected via either a left- or right-sided pancreatectomy with low mortality risk, but with notable major or minor

Fig. 8 FNAC supraclavicular lymph node showing metastatic adenocarcinoma (Pap, X400)

morbidity [6–9]. Resection is carried out usually for symptoms, large size, or the inability to distinguish a serous cystic neoplasm from a mucinous lesion, which has greater malignant potential [5–7]. In our patient, a rapid transformation of SMA was noted to serous cystadenocarcinoma (invasion and metastasis) in a matter of 9 months.

In conclusion, although prognosis for benign SMA is excellent, there is a small but definite risk of malignant change in serous cystadenoma as illustrated by our case. Regular follow-up is essential particularly when excision is not performed.

Conflict of interest None.

Source of support None.

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