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Case Report

Sporadic non-ampullary duodenal adenoma with high grade dysplasia: A rare case report

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ABSTRACT

Duodenal adenomatous polyps are usually classified according to the mucin phenotype into intestinal (89.1%) and gastric type (10.9%). The intestinal-type polyps are morphologically divided into tubular and tubulo-villous adenomas, whereas the gastric-type into pyloric gland adenomas and foveolar adenomas. The duodenal adenomas are also clinically categorised into sporadic duodenal adenomas and adenomas associated with genetic syndromes for instance familial adenomatous polyposis (FAP), or MUTYH associated adenomatous polyposis (MAP). Sporadic duodenal adenomas are less common than FAP related adenomas and are usually recognized in elderly men in their 6^{th} to 8^{th} decade of life. The large non-ampullary solitary duodenal adenomas ≥ 20 mm in diameter with high grade dysplasia show a significantly high risk of progression to adenocarcinoma and therefore, must be treated immediately. Our case, is a middle-aged female who presented with symptoms and had an exophytic duodenal mass of about 60mm in diameter, microscopically showing tubulo-villous intestinal pattern with high grade dysplasia and no breach in muscularis mucosae, thereby making it a case with distinct presentation and characteristics.

1. Introduction

Duodenal adenomas usually occur in the setting of familial polyposis where there are found in multiple numbers that often carpet the entire duodenal surface or they can occur sporadically which are now being increasingly identified by upper endoscopies performed for other reasons. These adenomas can be located in the duodenal bulb, ampullary/peri-ampullary region or distal duodenum.[1] Sporadic duodenal adenomas are uncommon and present in approximately 40% of the patients, whereas the remaining 60% patients present with FAP.[2]

Here, we report a rare case of Tubulo-villous non-ampullary adenoma with high grade dysplasia in a middle-aged female.

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2. Case Report

A 48-year female came to the OPD with the complain of pain in abdomen, yellowish discolouration of eyes and loss of appetite from last twenty days. Her hemogram revealed Hb: 10.6g/dl, TLC: 11,500 cells/cumm, platelets: 310,000 cells/cumm with a microcytic hypochromic blood picture. Her LFTs were as follows; AST: 22, ALT: 23, ALP: 175, Gamma glutamyl transferase: 123, total bilirubin: 0.53g/dl (direct: 0.21, indirect: 0.32). Kidney function tests and other tests were normal. Her whole abdomen ultrasound revealed an overdistended gall bladder with dilated common bile duct and pancreatic duct. No mass was identified. Her upper GI endoscopy revealed an exophytic growth in the second part of duodenum and malignancy was suspected. After an informed consent, she was taken for a Whipple's procedure and we received a 10% formalin preserved gastro-

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pancreatico-duodenectomy specimen along with a gastric-duodenal lymph node. On gross examination, an exophytic mass was identified in second part of duodenum measuring about 6.5X5X0.5 cm (Figure 1). Microscopy examination from the growth revealed a tubule-villous architecture of the polyp. The lining cells were pseudostratified, tall columnar with elongated, hyperchromatic nuclei. Occasional atypical mitosis was also noted. Nuclei at places were round, vesicular with prominent nucleoli. The lamina propria revealed intense chronic inflammation and oedema. There was no breach of muscularis mucosae seen. No desmoplasia was seen (Figure 2). The resected margins of stomach, gall bladder, CBD, pancreas and lymph node were negative for malignancy. The peri-ampullary region was negative for malignancy.

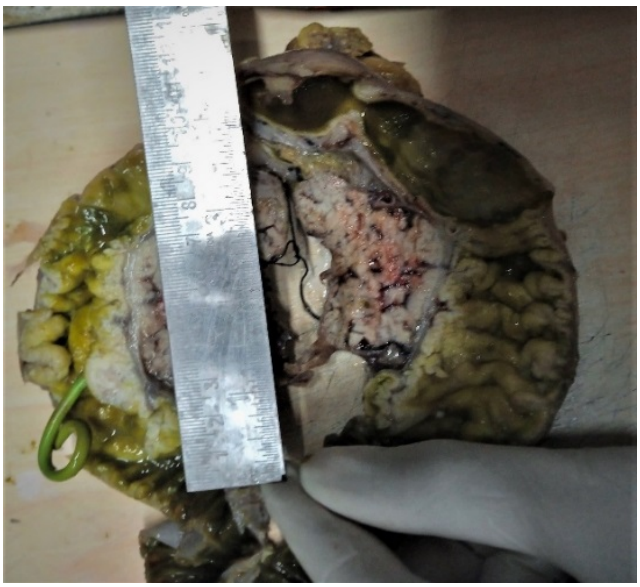

Figure 1: Gross specimen showing theexophytic growth in the second part of duodenum.

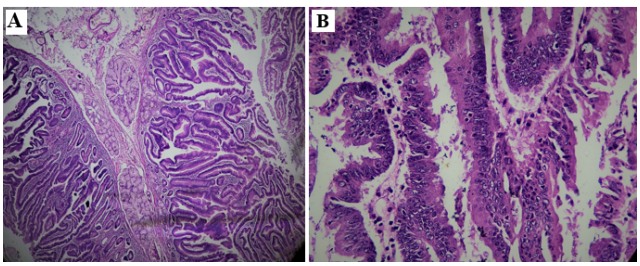

Figure 2: A- Histologic section showing adenomatous glands intubulo-villous architecture with high grade dysplasia, intact muscularis mucosae and benign Brunner's glands. H&E 200X. B- Tall columnar intestinal cells showing nuclear crowding, overlapping, loss of polarity, prominent nucleoli and mitotic figures consistent with high grade dysplasia. H&E 400X.

3. Discussion

Duodenal adenomatous polyps are usually classified according to the mucin phenotype into intestinal (89.1%) and gastric type (10.9%).[3]The intestinal-type polyps are morphologically divided into tubular and tubulo-villous adenomas, whereas the gastric-type into pyloric gland adenomas and foveolar adenomas.[4] The duodenal adenomas are also clinically categorised into sporadic duodenal adenomas and adenomas associated with genetic syndromes for instance familial adenomatous polyposis (FAP), or MUTYH associated adenomatous polyposis (MAP).[1] Sporadic duodenal adenomas are less common than FAP related adenomas and are usually recognized in elderly men in their 6^{th} to 8^{th} decade of life. Most are asymptomatic, being identified endoscopically as sessile polyps that are usually arising in the second part of the duodenum.[5,6]FAP related duodenal adenomas generally are multiple, sessile, and largely located in the descending duodenum.[7]Histologically, low grade adenomas comprise of crowded cells with elongated, hyperchromatic nuclei involving the entire thickness of the mucosa in a tubular or tubular-villous architecture. High grade dysplastic lesions, display distorted, back-to-back or cribriform glands, cells showing marked nuclear stratification, severe atypia with enlarged hyperchromatic nuclei, prominent nucleoli and loss of nuclear polarity.[6]Immunophenotypically, intestinal adenomas are defined by the expression of CD10, CDX2 and/or MUC2 proteins. MUC5AC and MUC6 which are typically expressed by gastric type of adenomas, are negative in intestinal adenomas. However, a focal gastric differentiation, may be present. Both sporadic duodenal adenomas and FAP-related adenomas demonstrate molecular alterations similar to those observed in colorectal adenomas that is characterized by genetic alterations, involving the APC and KRAS genes whereas, DNA mismatch repair abnormalities and TP53 mutations are only rarely identified. BRAF mutations have not been acknowledged yet.[8]Okada et al[9] in their multivariate analysis of duodenal adenomas concluded that lesions with low-grade dysplasia and < 20 mm have only 4.7% risk of progression to adenocarcinoma, whereas adenomas that are > 20 mm, or have high grade dysplasia, have approximately 54.5% rate of progression to adenocarcinoma. They emphasized that large non-ampullary solitary duodenal adenomas ≥ 20 mm in diameter with high grade dysplasia show a significantly high risk of progression to adenocarcinoma and therefore, must be treated immediately.[10]Similar results were published by Kim HK et al who concluded that HGD lesions and non-ampullary SDA >40mm in diameter have high risk of progression to adenocarcinoma and therefore should be treated by snare polypectomy, advanced endoscopic techniques, or surgery.[11]Our case, is a middle-aged female who presented with symptoms and had an

exophytic duodenal mass of about 60mm in diameter, microscopically showing tubulo-villous intestinal pattern with high grade dysplasia and no breach in muscularis mucosae, thereby making it a case with distinct presentation and characteristics.

4. Conclusion

Solitary non-ampullary duodenal adenomas with high grade dysplasia are uncommon and are associated with a high risk of developing duodenal adenocarcinoma thereby, prompting appropriate and timely surgical intervention and management.

5. Source of Funding

None.

6. Conflict of Interest

None.

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