Non Ampullary Primary Duodenal Tumor (Villious Tumor of Duodenum) A Rare Case Report

Jigar V Shah[1], Ronak D Vyas[2]

ABSTRACT

Introduction: Solitary duodenal tumors are very rare. Majority of them (75%) are periampullary, sized less than 1 cm, 10 % are in the non periampullary region in 2nd part, 10 % in the 3rd part and only 5% are in the first part of duodenum. Majority of patients are asymptomatic or the complaints are non specific.

Case report: We present a case of 46 year old male, without any co-morbidity who on evaluation found to have a large ulcerated mass at d1/d2 junction.

Conclusion: Surgical management of VTD is selective, based on clinical presentation, information from pre-op diagnostic evaluation, presence of polyposis syndrome and intra op findings.

Keywords: Villous tumors of duodenum, duodenal tumors, local resection, pancreatico duodenectomy.

INTRODUCTION

Small bowel tumors are rare and account for less than 10% of the GI neoplasm.(1-3) Most of the time they are identified incidentally while upper GI endoscopy is done for some other purpose. Majority of patients present with small bowel obstruction, bleeding, or anemia. Small intestine makes about 75 % of the length and 90% of the surface area of the GI tract, but it is unique in being highly resistant to tumor formation. Bacteriological factors and the rapid passage of its liquid contents are the most obvious reasons.

With the use of upper GI endoscopy Villous Tumors of Duodenum (VTD) are being recognized with increasing frequency. Association of colonic polyps with duodenal tumors has increased awareness. 20 % have an associated polyposis Syndrome.

CASE REPORT

The patient was a 46 year old male without any co-morbidity, presented in October 2015, with complains of pain abdomen and vomiting for 6 months, decrease appetite, and weight loss for 4-5 months. Clinical examination was within normal limit and further evaluation with upper GI endoscopy revealed a large ulcerated mass about 6x4x3 cms at d1/d2 junction and biopsy revealed tubulovillous adenoma (Figure-1).

Blood investigations: HB – 7.4, TLC – 8100, Platelets - 413000, INR - 1.10, Bilirubin - 0.58, SGOT - 19, SGPT - 12, SAP - 71, TP - 6.61, Albumin - 3.65, Cr - 0.91, Na - 139, K - 4.11

Chest X-Ray: No Abnormality Detected

SPECIAL INVESTIGATIONS

CT Abdomen: Suggestive of large, relatively ill defined heterogeneously enhancing, hypo dense mass lesion within the lumen of 2nd and 3rd part of duodenum causing luminal distention. The mass showed ill defined planes with the distal common bile duct, head and uncinate process of pancreas which appeared bulky and heterogeneous in attenuation – signs of invasion into the surrounding structures, with few sub centimeter size loco-regional lymph nodes which suggested possibility of mitotic etiology likely from duodenum with extension (Figures-2,3).

CT – Angiography: Enhanced mass lesion was seen in d2 and d3.

CancerAntigen: 19-9 – 2.09, Carcino Emroyonic Antigen – 0.75.

After preoperative clearance he was taken up for surgery with a intent to do frozen section biopsy and proceed. He was operated for Vertical duodenal enterotomy, cholecystectomy, cannulation of lower bile duct and ampulla of vater by placing infant feeding tube through cystic duct stump, mass excision and frozen section biopsy, billroth 2 distal gastrectomy with duodenal enterotomy closure, with loop gastrojejunostomy with feeding jejunostomy (Figure-4).

Operative findings

1. Large pedunculated mass arising from the mesentric border of d1 and d2 junction around 6x4x3 cms. On cannulation of the ampulla of vater through cystic duct stump, it was found 2 to 3 cm proximal to it.2.Intraoperative frozen section: tubullovillous adenoma.3.Liver and other viscera normal.

Hospital Course: Postoperatively patient was kept in ICU for observation for one day. Post op day 2, Ryle's tube was removed and patient was started with oral sips. He was started with FJ trial feed on post op day 3 with oral liquid diet which was gradually increased and was discharged in a stable condition on post op day 6.

No. of complications: No complications.

Claviens grade: 0.

Charlson's Co-morbidity index score: 1.

Histopathology: Section from the mass showed a polypoidal tumor composed of tubules which were closely packed and irregular. The cells lining them showed focal stratification,

[1]Associate Professor, Department of Surgery SBKSMIRC, Sumandeep University, [2]Assistant Professor, Department of Surgery MP Shah Medical College Jamnagar, India

Corresponding author: Dr Jigar V Shah, 4, Keya Duplex, Near Radhika Society, Behind Devnagar, Makrand Desai Road, Vadodara, 390007, India

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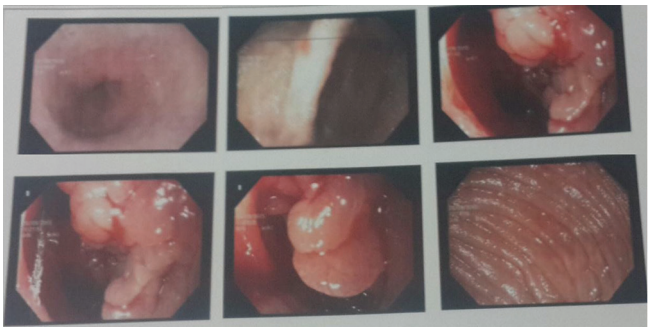

Figure-1 Endoscopy pictures showing tumour in the second part of duodenum.

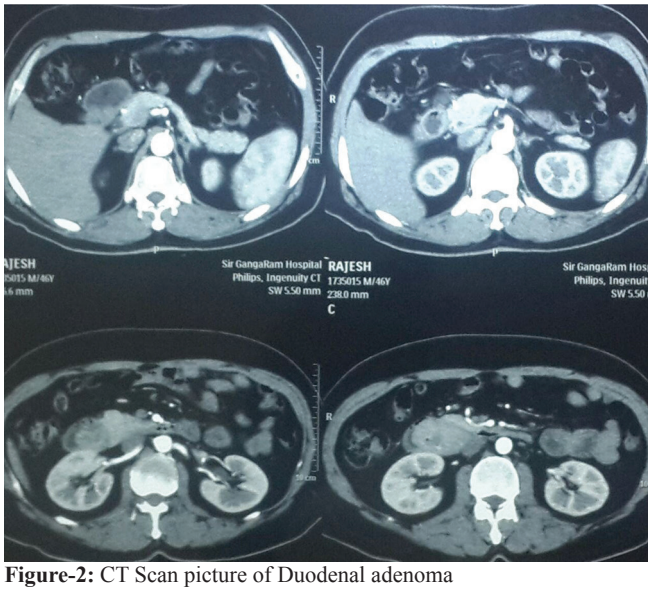

Figure-2: CT Scan picture of Duodenal adenoma

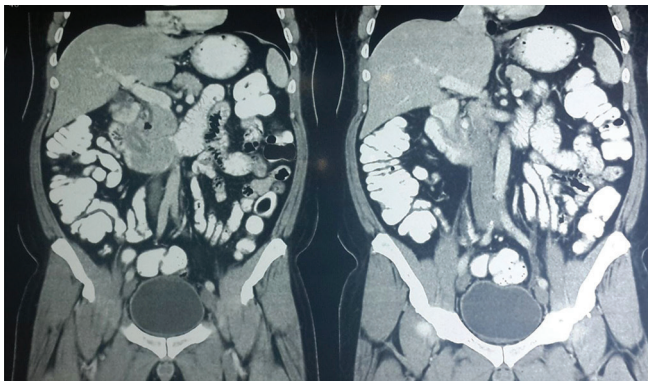

Figure-3: CT Scan picture of Duodenal adenoma

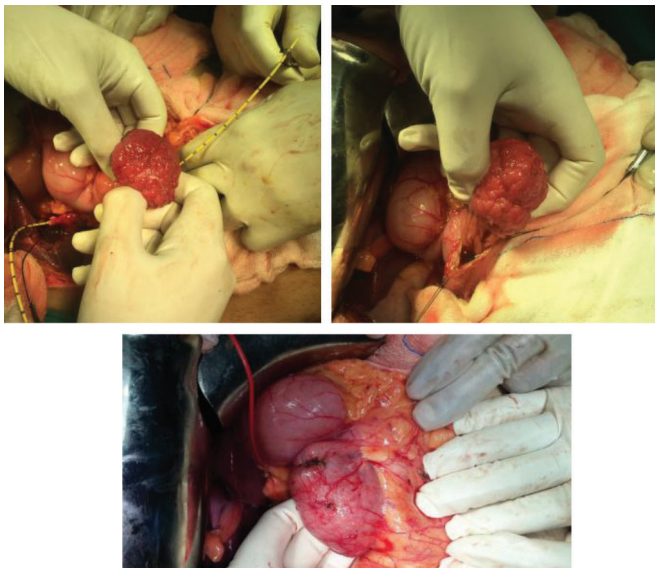

Figure-4: Intra-op: showing duodenal adenoma and cannulation of cystic duct

nuclear hyperchromasia and a few mitosis, but there was no loss of polarity and no invasion into the underlying muscularis mucosa. Mild chronic inflammatory infiltrate was present in the stroma. The stalk and resected base were free of tumor. Section from the base of the stalk showed patchy ulceration. No residual tumor was seen. The duodenal mucosa on either side showed brunner gland hyperplasia. The proximal margin showed normal gastric mucosa.

Final Impression: Tubular adenoma.

DISCUSSION

Cruveilhier described the 1st case of benign duodenal Brunner's gland adenoma in 1835.[2] In 1928, golden published the first definitive report for the treatment of duodenum adenoma. Duodenum is the common site for villous tumors of duodenum, and account for less than 1% of the total gastrointestinal tumors and only 16% of all benign tumors of intestine.[3,4]

Villous tumors of duodenum can behave in a manner similar to adenomas of the colon and rectum.[5] About 25% harbors invasive malignancy at the time of surgical excision. Size ranged from 0.5 to 9 cms. 75% are benign, 90% are solitary while 10% are multiple.[6]

Most common symptoms are abdominal pain, nausea and symptoms of pancreatitis.[6] They also present as anemia, obstructive jaundice and weight loss in patients containing invasive malignancy. VTD are asymptomatic in one third of patients.[6] They may cause GI bleed and small intestinal obstruction.[7-10] Malignant changes occur in 30% to 60% of duodenal villous adenomas and much less in tubullovillous and tubular adenomas.[11] Most of the cases of VTD are sporadic. They are also found to be associated with Familial Adenomatous polyposis, Gardener's syndrome, and Peutz Jegher syndrome.[6,12] Patients with sporadic duodenal adenomas may be associated with colonic neoplasia and should be offered colonoscopy.[13] Extended Fiber-optic endoscopy with full visualization of duodenum is the most useful and accurate tool for VTD. Endoscopy allows both visualization and biopsy. ALL lesions at ampulla are within reach of side viewing duodenescope.[6]

There are controversies regarding the most appropriate treatment of VTD. Treatment depends on their location, size, and degree of dysplasia. Rapid growth, polyp induration, severe dysplasia or villous change makes intervention necessary.[14] Various treatment options are available as endoscopic snare removal or ablation, endoscopic mucosal resection, endoscopic submucosal resection, pancreas sparing duodenectomy and pancreaticoduodenectomy.[6,12] Many a times it is not possible to reach a accurate pre-op diagnosis because sample taken by endoscopic forceps are very small. Frozen section is sometimes associated with false negative results. There have been reports of recurrence after local resection.

For benign tumors which are less than 1 cm in size, endoscopic resection by an experienced endsocpist is considered

appropriate,[6] however endoscopic removal of lesions more than 1 cm has also been reported. For lesions larger than 2 cms, piecemeal resection may be required.[13] The lesion should be assessed carefully to determine size, involvement of mucosal fold, proportion of the circumference involved and the relationship with the ampulla of vater. Assessment focuses on endoscopic respectability of the lesion and detection of any feature predicting sub mucosal invasion.[13] Endoscopic treatment includes endoscopic mucosal resection (EMR) and endoscopic sub mucosal dissection (ESD). The procedural risks of EMR increases with the size of the lesion. Prior submucosal injection reduces the risk of duodenal perforation. Most common in use are 0.9% saline. ESD involves a sub mucosal injection to lift the lesion in similar fashion to EMR. Submucosal plane meticulously dissected to remove the lesion en bloc. The rates of enbloc resection are high but at the risk of perforation and increase duration of procedure.[14] It is recommended that all the patients who have undergone endoscopic resection should be considered for surveillance endoscopy for the detection and treatment of recurrence.[15]

Surgical Treatment

For benign lesions more than 1 cm, transduodenal local excision versus PD pancreaticoduodenectomy. Whipples in proved cases of carcinoma, advanced disaease and carcinoma in situ. It is also an option for benign VTD in selected fit patient, especially for large or multicentric VTD to avoid recurrence. Factors like hard areas on palpation, an ulcerated tumour, dilatation or obstruction of common bile duct or pancreatic duct, pre-op biopsy showing severe dysplasia or villous lesions extending into bile or pancreatic duct should be considered as malignant lesions. In presence of these factors PD pancreaticoduodenctomy should be strongly recommended. In absence of these factors local resection is advised.[5]

For benign VTD, without the above mentioned factors in the lateral wall of 2nd part, 1st part, distal 3rd or 4th part segmental resections are reasonable options. For VTD, harboring invasive malignancy in 1st, 2nd, 3rd part, PD is the treatment of choice. Pancreatic fistula, anastomotic leakage and pancreatitis are serious complications after PD. Although the morbidity and mortality rates following PD have decreased drastically now, the procedure still results in considerable operative stress. For lesions in the distal 3rd or 4th portion, pancreas sparing duodenectomy with extended resection, because PD does not remove primary LN basins of these distal duodenal cancers. Previous studies had shown a recurrence rate of 32 % at 5 yrs after transduodenal local excision and 24% recurrences were cancer. Transduodenal local excision, wedge resection, segmental partial, full thickness and pancreas sparing duodenectomy are suitable for patients with premalignant or early malignant duodenal lesions. For early non ampullary duodenal cancer located in the distal third and 4th part of duodenum segmental resection is associated with reduced morbidity and mortality, while allowing for satisfactory clear margins and adequate lymph node desection.

CONCLUSION

Surgical management of VTD is selective, based on clinical presentation, information from pre-op diagnostic evaluation, presence of polyposis syndrome and intra op findings. For VTD treated by local excision and endoscopic method, regular, frequent, and longterm endoscopic surveillance is mandatory. In our patient, the tumor was at D1/D2 junction, away from ampula of vater with benign histology, so one time surgical procedure was curative.

REFERENCES

1. Cancer of the small bowel: laura doyon,Alexander greenstein, and Adrian Greenstein. Abeloff's clinic al oncology. 76,1271-1277.
2. J. Anatomy of the Human Body. New York, NY: Harper and Bros 1844.
3. Van de Walle P, Dillemans B, Vandellanotte M, Proot L. The laparoscopic resection of benign stromal tumor od duodenum. Acta Chir Belg. 1997;97:127-9.
4. Ohba R, Otaka M, Jin M, Odashima M, Matsuhashi T, Horikawa Y, Hatakeyama N, Mimori N, Kinoshita N, Koizumi S, Takahashi T, Watanbe S. Large Brunner's gland hyperplasia treated with modified endoscopic submucosal dissection. Dig Dis Sci. 2007;52:170-2.
5. Sakorafas GH, Friess H, Dervenis CG. Villous tumors of the duodenum: biological characters and clinical implications. Scan J Gasteroenterol. 2000;35:337-344.
6. Mayo clinic of gastrointestinal surgery. 2004. Mayo foundation of medical education and research.
7. Isolated duodenal adenoma presenting as GI bleed JCDR: 2014 june 8 (6) NDO1-NDO2.
8. Schulten MF, jr, Oyasu R, Beal JM. Villous adenoma od duodenum. A case report and review of literature.Am J Surg. 1976;132:90-96.
9. Bremer EH, Battaile W, Bulle PH. Villous tumors of the upper gastrointestinal tract. Am J Gasteroenterol. 1968;50:135-143.
10. Cohen A, Mcneil D, Terz JJ, et al. Neoplasms of small intestine. Am J Dig Dis. 1971;16:815-824.
11. Adedeji OA, Trescoli –Serrano C, Garcia – Zarco M. Primary duodenal carcinoma. Postgraduate Med J. 1995;71;354-358.
12. Aslan S, Cetin B, Markoc F, et al. A duodenal villous adenoma associated with in situ carcinoma: a case report. Turk J Cancer. 2001;31:162–167.
13. Basford PJ, Bhandari P. Endoscopic management of nonampullary duodenal polyps. Therap Adv Gastroenterol. 2012;5:127–138.
14. HF Vasen, S Bulow, T Myrhoj, L Mathus-Vliegen, G Griffioen, E Buskens, et al. Decision analysis in the management of duodenal adenomatosis in familial adenomatous polyposis. Gut. 1997;40:716–9.
15. D Apel, R Jakobs, A Spiethoff, JF Riemann. Follow-up after endoscopic snare resection of duodenal adenomas. Endoscopy. 2005;37:444–8.

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