Villous Tumors of the Duodenum

CHARLES W. CHAPPUIS, M.D., FRANK C. DIVINCENTI, M.D., and ISIDORE COHN, JR., M.D.

Department of Surgery, Louisiana State University School of Medicine, New Orleans, Louisiana

Five cases of villous tumors of the duodenum are reported, all of which involve the ampulla of Vater. Three of the five lesions contained either infiltrating carcinoma or carcinoma *in situ*. Although preoperative endoscopic biopsy was performed on all tumors, no malignancy was identified. Frozen sections done at the time of operation on the three patients with carcinoma also failed to identify malignancy. One patient underwent pancreaticoduodenectomy and four patients had local excision of the tumor. Three of the patients treated with local excision developed recurrence and two subsequently had pancreaticoduodenectomy. Because of the difficulty in making an accurate diagnosis and the chance of recurrence when local excision is employed, strong consideration should be given to pancreaticoduodenectomy as the initial form of treatment of these lesions.

VILLOUS TUMOR OF THE DUODENUM was first described by Perry in 1893 as a broad-based cauliflower-like mass that he referred to as a duodenal papilloma.[1] These lesions are relatively uncommon, with only 73 cases reported by Komorowski and Cohen in a 1981 review.[2] Pathologic descriptions have included the terms villous adenoma, villous papilloma, papillary adenoma, tubulovillous adenoma, and villoglandular polyp. Most lesions are found in the second portion of the duodenum with presenting symptoms usually related to biliary tract obstruction, occult bleeding, or obstruction of the duodenal lumen.

The high incidence of malignancy in villous lesions of the duodenum as well as the frequent involvement of the ampulla of Vater has resulted in considerable controversy regarding proper management. Pancreaticoduodenectomy remains the procedure of choice with invasive cancer. The appropriate treatment for those lesions that are benign or contain carcinoma *in situ* remains controversial.

In this report, five new cases are described and the literature is reviewed with emphasis on the incidence of malignancy, problems in achieving accurate preoperative diagnosis, and the choice of surgical therapy for ampullary villous tumors.

Materials and Methods

Pathology records at Charity Hospital were reviewed in order to identify all patients with the diagnosis of villous tumor of the duodenum. In addition, three cases were contributed from the private practice of two of the authors. Information on age, symptoms, diagnostic studies, operative procedure, pathologic findings, and follow-up was recorded. Invasive carcinoma was defined as invasion of the muscularis mucosae or of deeper structures. The greatest diameter measured in centimeters was used as the size of the lesion.

Results

There were three women and two men with an average age of 61 years (range, 34 to 84 years; Table 1). The tumor was located at the ampulla of Vater in all patients. In addition, one patient with Gardner's syndrome had multiple villous tumors of the duodenum. Three of the five patients presented with abdominal pain as their chief complaint. Two patients were jaundiced at the time of presentation. The patient with Gardner's syndrome was

Presented at the 100th Annual Meeting of The Southern Surgical Association, Boca Raton, Forida, December 5–7, 1988.

Correspondence and reprint requests: Charles W. Chappuis, M.D., Department of Surgery, Louisiana State University School of Medicine, 1542 Tulane Avenue, New Orleans, Louisiana 70112.

TABLE 1. *Summary of Clinical Data*

Patient	Age	Sex	Symptoms	Diagnostic Studies	Procedure	Pathology	Follow-Up
1 (1979)	60	M	Abdominal pain	US; dilated ducts UGI; normal Endo; mass at Ampulla	Local excision, sphincteroplasty	Benign	Recurrence at 4 years; Whipple; Well at 5 years
2 (1985)	84	M	Abdominal pain	Obstruction of CBD on T-tube cholangiogram Endo; mass at Ampulla	Local excision, sphincteroplasty	INV	Recurrence; Died at 2 years
3 (1985)	34	F	Asymptomatic	UGI; multiple polyps in the duodenum Endo; multiple polyps	Local excision, sphincteroplasty	Benign	Recurrence at 1 year; Whipple; Well at 2 years
4 (1986)	61	F	Jaundice	US; Dilated ducts and Ampullary mass Endo; mass at Ampulla	Whipple	INV	Well at 2.5 years
5 (1986)	66	F	Abdominal pain, jaundice	US; Dilated ducts and Ampullary mass Endo; mass at Ampulla	Local excision, sphincteroplasty	CIS	Well at 1.5 years

CBD, common bile duct; CIS, carcinoma in situ; Endo, esophagogastroduodenoscopy; INV, invasive carcinoma; UGI, upper gastrointestinal series; US, ultrasound.

TABLE 2. *Literature Review: Villous Tumors of the Ampulla of Vater*

Author	Age	Sex	Procedure	Pathology	Results
Cattell[5]	64	M	Ex	Benign	Alive and well at 5 years
Cattell[5]	76	M	Ex	Benign	Alive and well at 3 years
Cattell[5]	53	F	Ex	Benign	Alive and well at 12 years
Cattell[5]	43	F	Ex	Benign	Alive and well at 7 years
Catetll[5]	55	M	Ex	Benign	Alive and well at 3 years
Cattell[5]	76	F	Ex	Benign	Alive and well at 0.5 years
Cattell[5]	33	F	Ex	Benign	Alive and well at 1 year
Cattell[5]	64	M	Ex	Benign	Alive and well at 1 year
Cattell[5]	39	M	Ex	Benign	Alive and well at 1 year
Cattell[5]	64	M	PD	Carcinoma-Inv	Dead; 4 years-Recurrence
Cattell[5]	70	F	PD	Carcinoma-Inv	Dead; 2 years-Recurrence
OH[6]	71	F	Ex	Benign	Alive and well
OH[6]	75	F	Ex	Benign	Alive and well
Meltzer[7]	69	M	PD	Carcinoma-Inv	Alive and well
Meltzer[7]	60	M	Ex	Carcinoma-IM	Alive and well at 5 years
Deucher[8]	58	M	PD	Carcinoma-Inv	Alive and well at 10 years
Deucher[8]	63	F	PD	Carcinoma-Inv	Alive and well at 14 years
Griffin[9]	55	M	Ex	Benign	Alive and well
Dayal[10]	56	F	PD	Carcinoma-IM	Alive and well
Faust[11]	74	M	Ex	Carcinoma-Inv	Liver metastasis at operation
Mir-Madjlessi[12]	59	F	PD	Benign	Alive and well
Thomas[13]	69	F	PD	Benign	Alive and well at 2.5 years
Spira[14]	59	M	Ex	Carcinoma-Inv	Alive and well
Moore[15]	57	F	PD	Carcinoma-Inv	Alive and well at 5 months
Sobol[16]	47	F	Ex	Benign	Alive and well
Taxier[17]	77	F	Ex	Carcinoma-Inv	Alive and well at 1 year
Hasleton[18]	39	M	PD	Carcinoma-Inv	Alive and well
Hinder[19]	11	M	Ex/PD	Carcinoma-Inv	Dead at 13 months
Herbsman[3]	65	F	PD	Benign	No follow-up
Everett[20]	52	M	Ex	Benign	Alive and well at 10 months
Perzin[21]	56	M	Ex	Benign	Alive and well at 3 months
Perzin[21]	52	F	PD	Benign	Dead, postoperative complications
Perzin[21]	58	M	PD	Carcinoma-IM	Alive and well at 10 years
Perzin[21]	63	M	BP	Carcinoma-Inv	Dead at 9 days; postoperative complications
Perzin[21]	61	M	PD	Carcinoma-Inv	Dead at 16 years; cirrhosis
Perzin[21]	51	F	PD	Carcinoma-Inv	Alive and well at 2 years
Perzin[21]	75	M	PD	Carcinoma-Inv	No follow-up
Perzin[21]	69	M	BP	Carcinoma-IM	No follow-up
Perzin[21]	68	M	Ex	Carcinoma-IM	No follow-up
Perzin[21]	75	F	BP	Carcinoma-IM	Dead at 3 weeks postoperative
Perzin[21]	65	M	PD	Carcinoma-Inv	Dead at 1 month postoperative

TABLE 2. (*Continued*)

Author	Age	Sex	Procedure	Pathology	Results
Perzin[21]	47	F	PD	Carcinoma-Inv	Dead at 2 months postoperative
Perzin[21]	72	F	PD	Carcinoma-Inv	No follow-up
Perzin[21]	61	M	PD	Carcinoma-Inv	Dead at 2 months postoperative
Perzin[21]	68	F	PD	Benign	Alive and well at 1 year
Hsueh[22]	33	M	PD	Carcinoma-Inv	Alive and well at 10 months
Geir[23]	80	F	Ex	Benign	Alive and well at 3 years
Haglund[24]	45	F	Ex	Benign	Alive and well at 4 years
Haglund[24]	44	F	PD	Carcinoma-Inv	Alive and well at 2 years
Lewis[25]	53	M	PD	Benign	Alive and well at 1 year
Rosenberg[26]	62	F	Ex	Benign	Dead at 5 years
Rosenberg[26]	81	F	Ex	Benign	Alive and well at 3 years
Rosenberg[26]	80	F	Ex	Benign	Dead at 2.5 years; No recurrence
Rosenberg[26]	56	F	Ex	Benign	Alive and well at 8 years
Rosenberg[26]	68	M	PD	Benign	Dead at 2 years
Rosenberg[26]	62	M	Ex	Benign	Alive and well at 13 years
Ryan[27]	53	F	Ex/PD	Benign	Alive and well at 8 months
Ryan[27]	68	M	PD	Benign	Dead at 17 months; complications
Ryan[27]	65	F	Ex	Benign	Dead at 4 years; No recurrence
Ryan[27]	70	F	Ex	Carcinoma-Inv	Dead at 1 year; liver metastasis
Ryan[27]	56	F	PD	Carcinoma-Inv	Dead at 6 months; liver metastasis
Ryan[27]	78	M	BP	Carcinoma-Inv	Dead at 5 years
Ryan[27]	76	F	BP	Carcinoma-IM	Alive and well at 18 months
Ryan[27]	41	M	BP	Carcinoma-Inv	Alive and well at 2 months; liver metastasis
Ryan[27]	42	M	PD	Carcinoma-IM	Alive and well at 1 year
Ryan[27]	53	M	PD	Carcinoma-Inv	Alive and well at 6 months
Ryan[27]	43	F	PD	Carcinoma-Inv	Alive and well at 6 months
Ryan[27]	71	F	PD	Carcinoma-Inv	Alive and well at 3 months
Ryan[27]	79	F	BP	Benign	Alive and well at 18 months
Ryan[27]	76	M	Biopsy	Benign	Dead, preoperative arrhythmia
Ryan[27]	70	F	Ex	Benign	Alive and well at 4.5 years
Ryan[27]	63	F	ER	Carcinoma-IM	Alive and well at 2 years
Present series	60	M	Ex/PD	Benign	Alive and well at 5 years
Present series	84	M	Ex	Carcinoma-Inv	Dead at 2 years; recurrence
Present series	34	F	Ex/PD	Benign	Alive and well at 2 years
Present series	61	F	PD	Carcinoma-Inv	Alive and well at 2.5 years
Present series	66	F	Ex	Carcinoma-IM	Alive and well at 1.5 years

BP, Bypass; Ex, Local Excision; ER, Endoscopic Resection; IM, Intramucosal; INV, Invasive; PD, Pancreaticoduodenectomy.

asymptomatic and underwent an upper gastrointestinal contrast study (UGI) as part of her routine follow-up. Both patients presenting with jaundice were found to have malignant lesions. Follow-up ranged from 1.5 to 9 years.

Barium contrast studies (UGI) were done in two of the five patients. One was read as normal and one revealed multiple filling defects in the duodenum. Ultrasonography was done in 3 patients, revealing dilated bile ducts in all 3 and an ampullary mass in 2 patients. Esophagogastroduodenoscopy was performed in all five patients, identifying the ampullary lesion in each. Endoscopic biopsies were done in each patient and all were interpreted as benign. All lesions were sessile with an average size of 3.3 centimeters (range, 2 cm to 6 cm).

Four of the five patients underwent local excision as an initial procedure and one underwent pancreaticoduodenectomy. Frozen section evaluation was done at the time of operation in four patients and all were interpreted as benign. Two were subsequently read as invasive carcinoma and one as carcinoma *in situ* with foci of superficial microinvasion.

Both patients with benign lesions underwent local excision as an initial procedure. One experienced recurrence at 1 year and the other at 4 years. Each underwent pancreaticoduodenectomy following discovery of the recurrence and is alive and well without evidence of recurrent disease at 2 and 5 years, respectively. One of the three patients with carcinoma developed a recurrence and died at 2 years. The other two patients are alive and well without evidence of recurrence at 2.5 and 1.5 years, respectively.

Discussion

Although villous tumors of the small intestine occur most frequently in the duodenum, they account for only 1% of all duodenal tumors.[3] Since Golden published the first definitive case of villous tumor of the duodenum in 1928,[4] 161 cases have been described, including the five cases reported here. Seventy-seven (47.8%) involved the ampulla of Vater and form the basis of this review (Table 2).

TABLE 3. *Presenting Symptoms*

Symptoms	# Patients	%
Jaundice	53	69
Abdominal Pain	31	40
Anemia/Melena/Guaiac + stool	15	19
Weight loss	14	18
Fever	14	18

TABLE 4. *Treatment of Ampullary Villous Tumors*

	Benign	Intramucosal Carcinoma	Invasive Carcinoma
Pancreaticoduodenectomy	8	3	21
Local excision	27	3	6
Bypass	1	3	3
Biopsy only	1	0	0
Endoscopic resection	0	1	0
Total	37	10	30

The average age of patients with ampullary villous tumors was 60.6 years with a range of 11 to 84 years. Of the 77 cases reviewed, 41 (53%) were women and 36 (47%) were men. The presenting symptoms listed in Table 3 occurred most frequently. Other symptoms included diarrhea, cholangitis, anorexia, nausea and vomiting, and pancreatitis. Thirty-three (82.5%) of the patients with malignant lesions presented with jaundice. Twenty-five had invasive carcinoma and eight had intramucosal carcinoma. Twenty (54%) of the patients with benign lesions were jaundiced. The association of mucorrhea and electrolyte loss, often described with villous lesions of the colon and rectum, has been reported only once with duodenal villous tumors.[28]

The average size for tumors located at the ampulla of Vater was 3.67 cm with a range of 1 cm to 11 cm. Kutin et al. previously reported that no duodenal villous tumor under 4 cm in size showed evidence of invasive malignancy.[29] Size was recorded for 37 of the patients with ampullary tumors. Of the 22 lesions less than 4 cm in diameter, 14 had evidence of malignancy, 10 of which were invasive (Fig. 1). Previous reports by Schulten et al. and Galandiuk et al. have found size to be a poor predictor of malignancy.[30,31] Our data support this view.

Recent reports have indicated an overall incidence of malignancy ranging from 35% to 63%.[27,30] For purposes of this review, malignant lesions were classified as either invasive or intramucosal based on whether there was penetration of the muscularis mucosae by the tumor. Forty of the 77 lesions located at the ampulla of Vater were malignant (52%). Thirty-nine percent had invasive carcinoma and 13% contained intramucosal carcinoma. In comparison, villous tumors of the colon and rectum have an overall incidence of malignancy of 23.4% (range, 21% to 29%).[32-34]

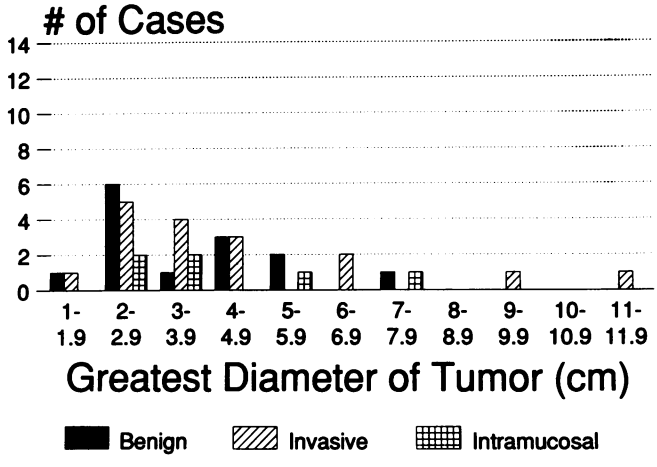

FIG. 1. The greatest diameter in centimeters of 37 reported ampullary villous tumors. The cross-hatched areas represent invasive and intramucosal carcinoma as indicated.

Endoscopic examination has all but replaced upper gastrointestinal series as the diagnostic procedure of choice in identifying the majority of duodenal villous tumors. All lesions located at the ampulla of Vater should be within reach of the side-viewing duodenoscope. Hypotonic duodenography may offer some advantage over the standard barium contrast study, especially in identification of lesions in the distal second, third, and fourth portions of the duodenum.[35] Ultrasound has occasionally been useful in identifying duodenal villous lesions. Bluth and Merritt have described a "pseudokidney" pattern of echos that is characteristic of a bowel lesion.[36] Ultrasound identified an ampullary mass in two of the three cases in which it was used in our series.

Accurate preoperative histologic diagnosis has remained a problem in evaluation of duodenal villous tumors. Because of the frequently large lesion size and the small sample taken *via* the endoscopic biopsy forceps, inaccurate diagnoses are common. False-negative endoscopic biopsy ranges from 25% to 56% in series reported by Galandiuk et al. and Ryan et al., respectively.[27,31] Fifteen patients underwent preoperative endoscopic biopsy in this review and six were found to be falsely negative for carcinoma (40%). Histologic evaluation at the time of frozen section was also associated with a high false-negative rate (33%).

The normal villous architecture of the small intestinal mucosa often makes classification of adenomas of the small bowel difficult.[21] Malignant transformation is also difficult to identify histologically. Geir et al. indicate that changes such as loss of cellular polarity and goblet cells, increased nuclear size, hyperchromatism, and high mitotic index are not universally accepted as criteria of malignancy.[23] They have suggested that more reliable evidence of malignant transformation may include piling up of the

epithelium to form multilayered masses, secondary gland formation in the intramucosal region, and desmoplastic reaction in the stroma.

Twenty-seven of the 37 patients with benign lesions underwent local excision of the tumor as the initial procedure. Eight patients had pancreaticoduodenectomy, one had bypass, and one had biopsy alone and died prior to any further therapy (Table 4). Twenty-four of the 40 patients with malignant lesions underwent pancreaticoduodenectomy as the initial procedure. Nine patients had local excision, six had palliative bypass, and one patient underwent endoscopic resection. One patient who underwent local excision had a pancreaticoduodenectomy 3 days later when the final pathology report indicated invasive adenocarcinoma.

Several cases of endoscopic excision of villous duodenal tumors have been reported.[31,37,38,39] Attempts at endoscopic removal are often difficult because of the size of the lesion as well as the sessile nature at the ampulla. In addition, these tumors are frequently friable and may require piece-meal excision. Retrieval of the resected lesion can be difficult, often rendering histologic examination impossible.[37] Biliary stents may be necessary in order to avoid ductal injury.[31] For these reasons, endoscopic resection of ampullary villous tumors should be used only in patients who are unfit for operative excision.

Treatment of ampullary villous tumors can be divided into two procedures: local (submucosal) excision or pancreaticoduodenectomy. Justification for local excision of villous tumors of the duodenum has been based on the presumed similarities to colorectal villous tumors. Such treatment has been based on the observation that colonic carcinoma does not metastasize to lymph nodes until there is invasion of the muscularis mucosae.[40] In the colon there are lymphatics associated with the muscularis mucosae but there are no lymphatics above this level. Similar findings have been noted in the stomach where Lehnert et al. describe the almost-complete absence of lymphatics from the mucosa.[41] This may account in part for the excellent prognosis of lesions described as early gastric cancer confined to the mucosa.[42]

In comparison, the small bowel mucosa contains lymphatics that course through the normal villi extending near the luminal surface.[40,43] Intramucosal carcinoma arising in a villous duodenal tumor may theoretically metastasize before invading the muscularis mucosae. The data to support this remain inconclusive.

Galandiuk et al. reported a recurrence rate of 46% in patients undergoing local excision.[31] This high rate of local recurrence was attributed to the narrow margins obtained because of the proximity to bile and pancreatic ducts and the difficulty in obtaining clear margins because of the soft, friable nature of these lesions. Three patients in this series with benign lesions treated by local excision had recurrence at 1, 3.5, and 4 years, respectively. Each of these patients underwent pancreaticoduodenectomy and is alive and well as of this writing.

Based on the high incidence of malignancy in ampullary villous tumors, the difficulty in achieving an accurate preoperative diagnosis and the frequency of local recurrence following submucosal excision, strong consideration should be given to pancreaticoduodectomy as the initial form of treatment. The operative mortality from pancreaticoduodenectomy should be less than 5% today.[44] Local submucosal excision and endoscopic resection should be reserved for those patients whose overall health does not permit a more extensive procedure.

Acknowledgment

The authors wish to thank Ms. Lynn Baker for her assistance with the preparation of the manuscript.

References

1. Perry EC. Papilloma of the duodenum. Trans Pathol Soc (London) 1893; 44:84.
2. Komorowski RA, Cohen EB. Villous tumors of the duodenum: a clinicopathologic study. Cancer 1981; 47:1377–1386.
3. Herbsman H, Wetstein L, Rosen Y, et al. Tumors of the small intestine. Curr Probl Surg 1980; 17:121–182.
4. Golden R. Non-malignant tumors of the duodenum. Am J Roent and Rad Ther 1928; 20:405–413.
5. Cattell RB, Braasch JW, Kahn F. Polypoid epithelial tumors of the bile ducts. N Engl J Med 1962; 266:57–61.
6. OH C, Jemerin EE. Benign adenomatous polyps of the papilla of Vater. Surgery 1965; 57:495–503.
7. Meltzer AD, Ostrum BJ, Isard HJ. Villous tumors of the stomach and duodenum. Report of three cases. Radiology 1966; 87:511–513.
8. Deucher F, Villiger KJ. Zwei maligne degenerierte duodenalpapillome; 10-und 14-Jahed-Heilung nach duodenopankreatektomie. Schweiz Med Wochenschr Nr. 46 1968;Bd. 98, 1822–1825.
9. Griffin WO Jr, Schaefer JW, Schindler S, et al. Ampullary obstruction by benign duodenal polyps. Arch Surg 1968; 97:444–449.
10. Dayal Y, Bass AG, Kraft AR, et al. Villous adenoma of the duodenum. Am J Surg 1972; 124:394–398.
11. Faust H, Hartweg H, Eugenidis N. Zur differentialdiagnose neoplastischer prozesse im duodenum. Fortschr Roentgenstr 1972; 116:499–508.
12. Mir-Madjlessi SH, Farmer RG, Hawk WA. Villous tumors of the duodenum and jejunum. Report of four cases and review of the literature. Dig Dis 1973; 18:467–476.
13. Thomas JM, Deshmukh N. Villous adenoma of the duodenum. Guthrie Bull 1975; 44:163–166.
14. Spira IA, Wolff WI. Villous tumors of the duodenum. Am J Gastroenterol 1977; 67:63–68.
15. Moore EL, Potts DW. Villous tumors of the duodenum. Oklahoma State Med Assoc J 1977; 70:481–488.
16. Sobol S, Cooperman AM. Villous adenoma of the ampulla of Vater. An unusual cause of biliary colic and obstructive jaundice. Gastroenterology 1978; 75:107–109.
17. Taxier M, Sivak MV Jr, Cooperman A. Villous adenoma of the ampulla of Vater. Gastrointest Endosc 1979; 25:155–156.
18. Hasleton PS, Shah S, Buckley CH, Tweedle DEF. Ampullary carcinoma associated with multiple duodenal villous adenomas. Am J Gastroenterology 1980; 73:418–422.
19. Hinder RA, Levy JI, Hamilton DG. A villous tumour of the duodenum with malignant change in a child. A case report. SA Med J 1980; 58:85–88.
20. Everett GD, Shirazi SS, Mitros FA. Villous tumors of the duodenum. Am J Gastroenterology 1981; 75:376–379.

21. Perzin KH, Bridge MF. Adenomas of the small intestine: a clinicopathologic review of 51 cases and a study of their relationship to carcinoma. Cancer 1981; 48:799–819.
22. Hsueh YS, Huang TY, Milos RJ. Primary adenocarcinoma of the duodenum arising from villous adenoma. J Indiana St Med Assoc 1983; 76:770–772.
23. Geier GE, Gashti EN, Houin HP, et al. Villous adenoma of the duodenum. A clinicopatholgic study of five cases. Am Surgeon 1984; 50:617–622.
24. Haglund U, Fork FT, Genell S, Rehnberg O. Villous adenomas in the duodenum. Br J Surg 1985; 72:26–27.
25. Lewis JH, Shorb PE, Nochomovitz LE. Benign duodenal villous adenoma obstructing the ampulla of Vater: a surgical dilemma. South Med J 1985; 78:1507–1514.
26. Rosenberg J, Welch JP, Pyrtek LJ, et al. Benign villous adenomas of the ampulla of Vater. Cancer 1986; 58:1563–1568.
27. Ryan DP, Schapiro RH, Warshaw AL. Villous tumors of the duodenum. Ann Surg 1986; 203:301–306.
28. Weiss JB, Semerdjian GG. Recurrent villous adenoma of the duodenum. Gastroenterology 1986; 90:440–442.
29. Kutin ND, Ranson JHC, Gouge TH, Localio SA. Villous tumors of the duodenum. Ann Surg 1975; 181:164–168.
30. Schulten MF Jr, Oyasu R, Beal JM. Villous adenoma of the duodenum. A case report and review of the literature. Am J Surg 1976; 132:90–96.
31. Galandiuk S, Hermann RE, Jagelman DG, Fazio VW, Sivak MV. Villous tumors of the duodenum. Ann Surg 1988; 207:234–239.
32. Shinya H, Wolff WI. Morphology, anatomic distribution and cancer potential of colonic polyps. An analysis of 7,000 polyps endoscopically removed. Ann Surg 1979; 190:679–683.
33. Galandiuk S, Fazio VW, Jagelman DG, et al. Villous and tubulovillous adenomas of the colon and rectum. A retrospective review, 1964–1985. Am J Surg 1987; 153:41–47.
34. Welch JP, Welch CE. Villous adenomas of the colorectum. Am J Surg 1976; 131:185–191.
35. Ring EJ, Ferrucci JT, Jr, Eaton SB, Clements JL. Villous adenomas of the duodenum. Radiology 1972; 104:45–48.
36. Bluth EI, Merritt CRB, Sullivan MA. Ultrasonic evaluation of the stomach, small bowel, and colon. Radiology 1979; 133:677–680.
37. Haubrich WS, Johnson RB, Foroozan P. Endoscopic removal of a duodenal adenoma. Gastrointest Endosc 1973; 19:201.
38. Siegel JH, Yatto RP. Endoscopic evaluation and therapy of periampullary adenoma. Am J Gastroenterol 1983; 78:225–226.
39. Pollak EW, Crow J, Jacobs WH, Choctaw WT. Villous duodenal adenoma. A less aggressive approach. J Kansas Med Soc 1981; 82:231–232.
40. Fenoglio CM, Kaye GI, Lane N. Distribution of human colonic lymphatics in normal hyperplastic, and adenomatous tissue. Its relationship to metastasis from small carcinomas in pedunculated adenomas, with two case reports. Gastroenterology 1973; 64:51–66.
41. Lehnert T, Erlandson RA, Decosse JJ. Lymph and blood capillaries of the human gastric mucosa. Gastroenterology 1985; 89:939–950.
42. Bringaze WL, Chappuis CW, Cohn I, Jr, Correa P. Early gastric cancer. 21-year experience. Ann Surg 1986; 204:103–107.
43. Morson BC, Dawson IMP. Gastrointestinal Pathology. 2nd ed. Oxford: Blackwell Scientific Publications, 1979.
44. Braasch JW. Pancreaticoduodenal resection. Curr Probl Surg 1988; 25:323–363.

Discussion

DR. KENNETH W. WARREN (Boston, Massachusetts): Dr. Berry, Dr. Jones, Members, and Guests: I have had the privilege of reading this manuscript. It is well documented and is an excellent review of the clinical aspects and especially the pathologic aspects of an unusual problem.

This represents a very small segment of the pathologic study of the ampulla of Vater and the periampullary area, but it is extremely important for two reasons: it illustrates the difficulty in establishing a precise diagnosis and, as a consequence, the difficulty in selecting an appropriate surgical procedure.

It has always been my attitude that local resection should be avoided and yet, as the literature shows, many of these patients are treated by local excision.

In our experience when I was at the Lahey Clinic and subsequently, we have seen about 12 of these. One malignant one was locally excised by one of my colleagues and the patient did very well.

I think the technical aspects have been touched on more precisely in the manuscript, but the difficulty is very obvious. On the side of the bile duct, one has reasonable leeway. One can do a papillotomy extending 1.5 cm or 2 cm, but when you approach the pancreatic duct you have very limited freedom. When I have done a local excision, I put a sound, a Bake's dilator, in each duct to minimize the prospect of ductal injury.

The best description of the pitfalls of doing local resection was described by Lord Smith. He said, "You start out. It's a small tumor. It is a small cancer." You do a sphincterotomy, you do a local excision. You decide that you haven't done enough or maybe the pathologist tells you that you haven't done enough, so then you do a wider excision. The first thing you know you may have dissociated the common-bile or the pancreatic duct, or both from the duodenum.

Then you have to reconstruct the ducts. You have done a complicated operation, ultimately, an operation more difficult, more dangerous than a Whipple, and an operation that is associated with a high rate of recurrence. I would advocate doing a Whipple in most instances with this type of lesion.

On the other hand, if you are going to do a Whipple, you are obligated to warn the patient and the family before the operation. The Whipple procedure, even for cancer of the head of the pancreas, today can be done with a mortality rate of less than 5%, and if it is done for a benign or malignant villous polyp, the mortality rate should be even less.

DR. JAMES T. EVANS (Macon, Georgia): Dr. Berry, Dr. Jones, Fellows, and Guests: I wish to compliment Drs. Di Vincenti, Cohn, and Chappuis on an excellent review, and I wish to thank them for the opportunity to discuss their paper and review the manuscript in detail.

They have, indeed, drawn our focus to the preoperative difficulty and frozen section difficulty in defining the malignant subset of villous tumors of the duodenum.

There have been several reports recently in the literature concerning computed tomography to help define a wall abnormality in malignant duodenal tumors including villous adenomas, and I would ask the authors to contribute their assessment of these preliminary reports of CT.

Second, in the area that they highlighted between the differential comparing villous duodenal malignancies and colorectal lesions, I would wonder if they would comment on the experimental animal data in which certain rodent models using carcinogens have revealed a very similar biphasic pattern of malignant duodenal and malignant colon tumors.

DR. FRANCIS C. NANCE (Livingston, New Jersey): The comment that I have relates to the conclusion that pancreaticoduodenectomy should be done as a primary procedure in this series of patients.

It seems to me that you did very well with the way that you managed these patients, which was by local excision for some of the patients and subsequent resection for recurrences in other patients. I gather that the follow-up on those patients has been satisfactory.

Did, in fact, anybody lose the opportunity to be cured by the way you managed those patients? I would be interested to know that.

Second, as a speculative question, it has been noted that most of these tumors are downstream from the biliary orifice at the ampulla, and there has been speculation by Watne and others that there is something in the bile that predisposes to the development of these tumors.

The question I have is whether diversion of the biliary tract with a Roux-en-Y type of anastomosis would, in fact, have altered the recurrence rate in this group of tumors.

It is a very interesting area of research, and I think it is something that might play a role in our understanding of the etiology of the disease.