Management of Duodenal Adenomas Involving the Ampulla of Vater – A Warning against Limited Resection

Jeremy Rossaak Philip Bagshaw Saxon Connor

Department of General Surgery, Christchurch Hospital, Christchurch, New Zealand

Key Words

Ampullary adenoma/carcinoma · Duodenal adenoma/carcinoma · High-grade dysplasia · Management · Surgery

Abstract

Duodenal adenomas are uncommon, however, when present a proportion have dysplasia associated with the adenoma and therefore require treatment. The options range from less invasive endoscopic treatments to a pancreaticoduodenectomy. This case report describes two patients with adenomas involving the ampulla of Vater. One patient had familial adenomatous polyposis, the other was a renal transplant patient with a large adenoma. Both patients' adenomas contained high-grade dysplasia. Both patients underwent a pancreaticoduodenectomy. Histology of both specimens demonstrated that the adenoma had migrated up the bile duct for at least 7 mm, and the pancreatic duct for 8 mm in one patient. Limited resection of ampullary adenomas may leave residual adenomatous tissue in the bile duct with the risk of recurrent adenomatous disease and malignant transformation.

Introduction

Polyyps in the second part of the duodenum are uncommon, however, up to 22% are adenomas [1]. A proportion of these have high-grade dysplasia and need some form of intervention to prevent the progression to carcinoma. There are a range of options to manage the adenomas, from less invasive endoscopic resections [2–4] to surgical resection [5] and pancreaticoduodenectomy [6, 7]. This paper describes the management and outcome of two patients who presented with high-grade villous adenomas of the ampulla. The aim is to highlight the potential pitfalls of a minimally invasive approach to resecting adenomas with high-grade dysplasia involving the ampulla of Vater.

Patient 1

A 66-year-old woman with familial adenomatous polyposis (FAP) had a colectomy and ileorectal anastomosis in 1961, followed by a completion proctectomy in 2004. As part of the follow-up, a gastroscopy was performed and demonstrated multiple adenomas carpeting the duodenum with high-grade dysplasia on biopsy. A computed tomography scan confirmed that the disease was localised but was associated with mild bile duct dilatation. Her other medical illness included hypertension and gastro-oesophageal reflux disease. Previous history included cholecystectomy and a hysterectomy.

Because of the number and the extent of the lesions, it was elected to perform a pancreaticoduodenectomy. The patient made an uneventful post-operative recovery and was discharged 14 days after surgery.

On histology multiple discrete and merging polypoid lesions were seen to stud the duodenal mucosa, ranging in size from 3 mm to $22 \times 15 \times 5$ mm, including a 10 mm polyp in the periampullary region. Histologically these demonstrated multiple tubular and villous adenomas with focally high-grade glandular dysplasia ([fig. 1](#)). Both high- and low-grade dysplasia extended down the common bile duct at the ampulla of Vater for a distance of 12 mm and down the separate pancreatic duct for a distance of 8 mm ([fig. 2](#), [fig. 3](#)). No invasion was seen. Low-grade dysplasia involved the jejunal margin but not the pancreatic or bile duct margins. All 11 lymph nodes in the pancreaticoduodenectomy specimen were negative.

Patient 2

A 61-year-old woman was investigated for iron deficiency anaemia in August 2004. Gastroscopy revealed a villous adenoma of the second part of the duodenum and histology confirmed a villous adenoma with low-grade dysplasia. An ERCP in October 2004 showed progression of the adenoma, then being 10 cm in length, almost circumferential ([fig. 4](#)). Histology now demonstrated high-grade dysplasia. A computed tomography scan indicated that the disease was localised.

The patient had significant co-morbidities, including ischaemic heart disease and a coronary artery bypass graft (2003), chronic renal failure and a renal transplant (2000), hypercholesterolaemia, hypertension, gastro-oesophageal reflux disease and recurrent urinary tract infections. Despite this, she elected to undergo a pancreaticoduodenectomy. The post-operative course was prolonged and complicated by a pancreatic anastomotic leak, resulting in a 4-week hospital stay.

On histology, a periampullary villous adenoma was present (macroscopically $90 \times 60 \times 17$ mm) featuring high- and low-grade glandular dysplasia. Both high- and low-grade dysplasia extend down the common bile duct at the ampulla for a distance of 7 mm, and there was colonisation of periampullary glands by dysplastic epithelium without invasion ([fig. 5](#)). Local excision was complete. All 4 lymph nodes were negative.

Discussion

The diagnosis of duodenal polyps is likely to increase in frequency as their association with hereditary neoplastic syndromes, such as hereditary non-polyposis colorectal cancer and FAP, becomes more recognised. An appreciation of the behaviour of duodenal polyps is important for their optimal management. Research into the natural history of duodenal polyps is inconclusive, with some authors indicating rapid progression of polyps to carcinoma [8], and a high incidence of carcinoma at diagnosis [9], whilst others describe a benign course [10]. Obviously this has implications for the management of these polyps.

Pancreaticoduodenectomy is the procedure of choice for malignant lesions of the duodenum, ampulla of Vater or head of the pancreas. Because of the considerable morbidity and mortality, there has been increased interest in less extensive operations for the management of benign disease. Alternate strategies include endoscopic resection, piecemeal [2], snaring [2, 4], or fulguration, argon plasma coagulation [3], trans-duodenal

resection [5], and pancreas-preserving duodenectomy [6]. These procedures limit the complications of surgery or post-operative morbidity, whilst attempting to maintain adequate disease control.

In all of these situations, except pancreas-preserving duodenectomy, where a biopsy of the bile duct is obtained at the time of surgery, dysplastic tissue may inadvertently be missed. The role of endoscopic ultrasound is not well established but may add information regarding invasion and/or ductal involvement. The two cases described here demonstrate one of the pitfalls of treating patients with dysplastic villous adenomas of the second part of the duodenum, involving the ampulla of Vater, with a limited resection as the dysplasia extended up the bile duct in both these cases. This would potentially have been missed in a trans-duodenal, endoscopic and pancreas-preserving resection of the duodenum.

The first patient presented with multiple duodenal polyps secondary to FAP. This is a common situation in which duodenal polyps are identified. The migration of dysplastic adenomatous tissue into the ampulla of Vater may explain some of the high recurrence rate of these adenomas after local excision [7]. The second patient had just one large villous adenoma, which was not related to a known hereditary polyposis syndrome. In this situation, dysplastic adenomatous tissue had again migrated up the ampulla of Vater. Perhaps the immunosuppression from the renal transplant could explain the aggressiveness of the adenoma.

In conclusion we present two cases with villous adenomas of the duodenum, both of which displayed migration of the dysplastic adenoma up the bile duct. This would indicate that duodenal adenomas involving the ampulla of Vater, with features of dysplasia, should be treated with definitive surgery.

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Fig. 1. Haematoxylin and eosin (H+E) stained specimen showing the duodenal villous adenoma.

Fig. 2. H+E specimen demonstrating migration of the adenoma (dark) up the common bile duct and a transition from normal to adenomatous tissue (arrow).

Fig. 3. H+E section of the adenoma in the pancreatic duct sectioned longitudinally. The adenomatous tissue appears darker (arrow).

Fig. 4. Gastrosocopy demonstrated large villous adenoma of the 2nd part of the duodenum (black arrow); normal mucosa (white arrow).

Fig. 5. Histology (H+E) demonstrating migration of the dysplastic epithelium into the common bile duct (arrow).

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