Case Report

Hepatic abscess formation following embolisation of a carcinoid metastasis

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Accepted 6 September 1995

Carcinoid tumours are rare with an annual incidence of 1.3 per 100,000 in the N. Ireland population.¹ Carcinoid tumours are unusual in that hepatic metastases are compatible with a 20 to 40% five year survival.² Nevertheless, most patients become symptomatic when hepatic metastasis occurs. The resulting local symptoms and those of the carcinoid syndrome (where hepatic metastasis can be found in more than 95% of cases) greatly reduce the quality of the patient's life.^{3,4} It is therefore usual to treat such cases aggressively with somatostatin analogues and other anti-serotonin agents, chemotherapy, surgical debulking and hepatic artery occlusion (by ligation, temporary occlusion or embolisation).⁵ The latter is effective as hepatic neoplasms are almost solely supplied by the hepatic artery.⁶ The resultant selective tumour necrosis produces effective palliation of symptoms, which may be prolonged due to the slow growing nature of the carcinoid tumour.⁷

The following case report presents an unusual post-embolisation complication (hepatic abscess formation). The overall management of carcinoid hepatic metastases is briefly discussed.

CASE REPORT

A 61 year old man presented in 1987 with haemoptysis. A right lower lobe bronchial carcinoid tumour was discovered and subsequently resected. Over the next few months he developed diarrhoea, nocturnal wheeze and flushing. In 1989, investigation disclosed deranged liver function tests (LFTs), raised urinary 5-hydroxyindole acetic acid (5-HIAA) and raised plasma pancreatic polypeptide (PPP). An alcohol provocation test induced marked flushing which was greatly reduced by injection of a somatostatin analogue. There was no evidence of secretion of 5-hydroxytryptamine, prostaglandins, insulin, gastrin, vasoactive intestinal peptide, ACTH, parathormone or other active peptides, which may occur with carcinoid tumours of foregut embryonic origin. Four hepatic metastases were revealed on abdominal ultrasound (three in the right lobe and one in the left lobe). Intraheptic arterial chemotherapy combined with somatostatin was used at this time with good symptomatic control.

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Fig 1. Chest X-ray showing hepatic abscess as gas-fluid level under right hemidiaphragm.



Fig 2. CT scan showing hepatic abscess with gas in liver substance.

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Fig 3. Post-operative CT scan after resection and drainage showing fluid-filled cavity in right hepatic lobe.

In early 1992 there was symptomatic deterioration combined with biochemical and radiological evidence of tumour recurrence. In October 1992 he was admitted to hospital with pain in the right upper quadrant (RUQ) of the abdomen and evidence of a right basal pneumonia. Following treatment of the pneumonia, hepatic artery embolisation using particulate polyvinylchloride (PVC) was carried out on a large right lobar metastasis. He subsequently developed a short lived episode of vomiting and abdominal pain which settled with symptomatic treatment. An abdominal ultrasound scan prior to discharge at ten days post-embolisation revealed an eight centimetre embolised lesion with no evidence of complications.

Fifteen days later he was re-admitted with malaise, RUQ pain and tenderness, and constipation. Chest X-ray (Figure 1) revealed an air-fluid level under the right hemidiaphragm and abdominal films showed hepatic mottling with gas shadows. These findings were consistent with the development of an aseptic abscess in the liver with gas formation. This was confirmed on computerised tomography (CT) which revealed that the embolised lesion was now liquid in attenuation values with areas of gas giving rise to a "soapbubble" appearance (Figure 2).

Based on worsening clinical and radiological findings, rupture of the abscess was thought to be imminent with the risk of carcinoid crisis or peritonitis. A formal drainage procedure was therefore carried out with resection of the necrotic right lobar metastasis under somatostatin and antibiotic cover. Post-operative recovery in the intensive care unit was uneventful. A post-operative CT scan demonstrated multiple small hepatic metastases and a significantly improved fluid filled cavity in the right lobe of the liver (Figure 3). He was discharged on maintenance somatostatin and has remained well since, with flushing as his only complaint.

DISCUSSION

In Northern Ireland there is a central diagnostic laboratory and register collecting data on neuroendocrine tumours. There were 368 such tumours in the province between 1970 and 1985, of which 318 were carcinoids and 28 (9%) arose in the lung.¹ Carcinoid tumours are slow growing neuroendocrine tumours.⁸ They occasionally produce local symptoms (e.g. haemoptysis with a pulmonary carcinoid, obstruction with an appendiceal carcinoid) but usually produce symptoms secondary to hormone secretion.⁹ Eighty percent of carcinoid tumours are "midgut" in origin and secreted hormones are thus subject to hepatic first pass metabolism.¹⁰ Such tumours are therefore usually asymptomatic until hepatic metastases have occurred when hormones can be secreted into the systemic circulation and produce the carcinoid syndrome. It is at this time that most cases are diagnosed.

Treatment firstly requires anatomical and functional characterisation.⁹ Small primary tumours can be locally resected whereas larger ones may require formal "enbloc" resection or palliative debulking. Such operations can be very successful e.g. resection of bronchial primary carcinoids results in a 70% five year survival.

Since hepatic metastases produce the main symptoms but are not invariably fatal, they are treated aggressively with the following procedures:

1. Pharmacological hormonal manipulation. Somatostatin decreases both the synthesis and release of hormones by the tumour, as well as increasing the synthesis of inhibitory proteins. It may also prove to be anti-neoplastic and to slow tumour progression.

2. Chemotherapy with streptozotocin.

3. Surgical debulking – this is of particular use with unilobar hepatic metastases where a hepatic lobectomy can be performed.

4. Hepatic artery occlusion.^{5,7,11,12,13} This can be achieved by ligation or embolisation. Formal ligation requires a laparotomy and is associated with an operative mortality and rapid collateral formation. It produces symptomatic relief lasting a mean of five months. Temporary occlusion can be carried out by vessel layer formation or by implanting a tourniquet. These options reduce perioperative mortality by separating tumour necrosis from the laparotomy. They can also be repeated, and reduce collateral formation as the hepatic artery occlusion is only temporary.

Hepatic artery embolisation is a palliative procedure used to control pain, haemorrhage, hormone secretion and tumour size. Any accessible tumour can be embolised but it is most effective when used for metastatic neuroendocrine tumours. The procedure is covered with antibiotics and somatostatin to reduce the risk of infection and a carcinoid crisis respectively.¹⁴ Percutaneous selective angiography is used to identify the feeding vessels of the tumour. A shower of small particles embolises the smaller peripheral vessels, thus reducing collateral formation (analogous to surgical isolation), and is followed by embolisation of the main feeding vessels.^{7,11} A choice of embolising materials can be used including iodized oil, Gelfoam, Oxycel, PVC (available in graded sizes) and coils (which exclude reembolisation as well as concurrent intra-arterial chemotherapy). Re-embolisation can be carried out for tumour regrowth or to occlude subsequent collateral arteries. Re-embolisation was only required in under 10% of cases within 5 years in a Swedish trial.¹⁵

Palliation of symptoms occurs in almost every case and has a duration of 2-18 months (mean 7 months) and a further 3-18 months (mean 11 months) following reembolisation.¹⁴ Hepatic artery embolisation was initially used for bilobar hepatic metastases, where resection is impossible, and for patients unfit for surgery. It is now more widely used as it has a lower morbidity and negligible mortality compared to the alternatives, and requires less than one week of hospitalisation. Earlier and repeated embolisation may also improve patient survival.⁹ It also enables concurrent intra-arterial chemotherapy if a degradeable material is used. Such a combined chemoembolisation procedure improves tumour uptake of the chemotherapeutic agent while protecting the normal liver. This allows control of carcinoid symptoms as well as regression or stabilisation of the liver tumours in 80% of patients.¹⁶

As all embolised tumours undergo selective aseptic necrosis, the postembolisation syndrome (RUQ pain, nausea, pyrexia, leucocytosis and derangement of LFTs) is ubiquitous to a greater or lesser extent. Symptomatic treatment in the early stages with analgesia and antiemetics is sufficient.¹⁷ Prolonged symptoms lasting more than five days should be investigated to exclude rare complications. This would include abscesses which can be drained percutaneously or drained at laparotomy.^{5,12} Other complications of embolisation include septicaemia, carcinoid crisis, haemorrhage, gall-bladder ischaemia, pancreatic pseudocyst, hepatic artery aneurysm formation, and embolisation of unrelated arteries by release of the material into the general circulation.^{3, 9, 13}

Embolisation is becoming increasingly popular since it produces effective clinical and biochemical suppression of carcinoid metastases; it is both safe and inexpensive. However, it remains a purely palliative procedure. The only real hope for cure lies with orthoptic liver transplantation (OLT). In a series of 1000 OLTs over 6 years in Pittsburgh, two cases were for carcinoid metastases.¹⁹ Both patients died from causes unrelated to tumour recurrence and had no evidence of this at postmortem. This hopeful pointer to curative management should be examined prospectively in a trial of OLT against standard treatment. Early and repeated embolisation appears to be the current safest mode of management for patients with hepatic carcinoid metastases.

ACKNOWLEDGEMENT

We wish to thank Professor K D Buchanan for his help with this patient's management, and for permission to report this case.

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