Laparoscopic-assisted oesophagectomy for adenocarcinoma in an AIDS patient

Introduction

Since the advent of highly active antiretroviral therapy (HAART), life expectancy for human immunodeficiency virus (HIV) patients has significantly improved. It is unknown as to whether there is a link between HIV and oesophageal carcinoma and as such, HIV patients on HAART may be treated surgically for oesophageal malignancies to improve life expectancy and quality of life.

Discussion

Some physicians consider HIV to be a manageable chronic disease with the use of HAART. Despite this, HIV is associated with an increased risk of particular malignancies.

Acquired immunodeficiency syndrome (AIDS)-defining malignancies include Kaposi's sarcoma, non-Hodgkin's lymphoma and cervical cancer. These may be related to human herpes virus 8 (HHV 8), Epstein–Barr virus and human papilloma virus respectively, in an immunocompromised individual. Malignancies that occur more frequently in HIV patients but are not AIDS-defining illnesses (as they are not related to immunodeficiency) include Hodgkin's disease, squamous cell carcinoma of the eye, multiple myeloma, leiomyoscarcoma, testicular, lung and anal cancer.

Oesophageal disease presenting with dysphagia occurs less commonly since the use of HAART. Dysphagia is usually attributable to oesophageal candidiasis. Since HIV patients are susceptible to opportunistic infection other causes of dysphagia such as cytomegalovirus oesophageal ulceration should be considered (Conolly et al, 1989).

Cytomegalovirus ulceration is usually associated with odynophagia and is the most common viral cause of oesophageal disease. Interestingly herpes simplex virus oesophagitis is not particularly common; this is in contrast to individuals that are not immunosuppressed as a result of HIV infection.

In HIV patients presenting with dysphagia and/or odynophagia it is reasonable to commence a trial of antifungal therapy since candidiasis is the most likely cause. If no response is found within 2 weeks, endoscopy should be performed and biopsies may be taken. Barium swallow may be helpful. Indeed oesophageal candidiasis may be visualized as multiple plaque-like lesions. However, viral oesophagitis may produce a picture of single or multiple discrete ulcerations. Oesophageal ulceration may be found in patients with seroconversion to HIV, biopsy specimens of such ulcers have demonstrated the presence of retroviral virions (Wilcox, 1992).

In AIDS patients Kaposi's sarcoma is the most common neoplasm of the oesophagus. This may be asymptomatic and may not necessarily be present with cutaneous disease. This case raises the issue as to whether there is a link between HIV-associated immunosuppression and oesophageal adenocarcinoma; further study is needed.

A link between HIV and oesophageal carcinoma has not been established as there are few case reports in the literature of oesophageal malignancies in HIV patients and of those, histological subtype has not always been reported.

A single case of an AIDS patient diagnosed with squamous cell carcinoma that received preoperative radiation and chemotherapy followed by transhiatal oesophagectomy was found. This patient survived for over 1 year (Issa et al, 2004).

One case of an AIDS patient who underwent oesophagogastrectomy has been described (Issa et al, 2004). This patient had adenocarcinoma in association with Barrett's oesophagus. While the patient presented underwent chemotherapy, significant side effects were experienced. Thus neoadjuvant chemotherapy may reduce tumour size and ultimately improve survival in some individuals, but raises the issue of administering chemotherapeutic agents to patients already with a degree of immunosuppression (Chalasani et al, 1997).

Conclusions

The role of chemotherapy and surgical intervention in HIV patients with malignancy is an important consideration (Klugman and Schaffner, 1994). This case report describes the first case of a laparoscopic-assisted oesophagectomy in an AIDS patient. A laparoscopic approach is less invasive than an open procedure thus postoperative recovery is expedited (Diettrich and Kaplan, 1991). Fewer wound complications arise, postoperative pain is reduced and pulmonary function is optimized. **BJHM**

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Case Report

A 44-year-old man with known AIDS (acquired immunodeficiency syndrome) presented with an 18-month history of progressive dysphagia, occasional reflux, reduced appetite and significant weight loss. There was no odynophagia, haematemesis or haemoptysis. Upper gastrointestinal endoscopy and endoscopic ultrasound demonstrated a tumour 41–44 cm from the incisors. Two 6 mm regional paraoesophageal lymph nodes were seen at 34 cm and there were two 5 mm splenic artery nodes. Endoscopic staging was of a T3 N1 tumour at the gastro-oesophageal junction and biopsy findings revealed poorly differentiated adenocarcinoma. Computed tomography confirmed endoscopic findings and showed no evidence of metastatic disease.

The patient had previously been treated for oesophageal candidiasis, an AIDS-defining illness. He was diagnosed with human immunodeficiency virus (HIV) 4 years ago and was currently taking zidovudine, lamivudine and efavirenz or highly active anti-retroviral therapy (HAART). At presentation his viral load was less than 50 copies (Roche polymerase chain reaction assay, Amplicor HIV-1 Monitor) and his T-cell count was approximately 200/mm^3.

The patient received three cycles of neoadjuvant chemotherapy for tumour regression. This was complicated by the development of peripheral neuropathy. Despite ongoing nausea the patient was able to continue with HAART.

After completing the course of chemotherapy, the patient underwent a laparoscopic-assisted Ivor Lewis oesophagectomy. This is a two-stage procedure where a laparotomy is performed followed by thoracotomy and an anastomosis of the stomach to the upper oesophagus in the chest. He had a brief stay on the high dependency unit as a result of shortness of breath. Chest X-ray showed bilateral infiltrates suggestive of acute lung injury. *Pneumocystis carinii* pneumonia was deemed unlikely as a result of septrin prophylaxis. Nutritional status improved and he was discharged on day 16. Peripheral neuropathy is an ongoing issue.

Postoperative histology demonstrated a poorly differentiated adenocarcinoma of the gastro-oesophageal junction that had penetrated through gastric serosa. The pathological stage was pT3 N2. Ten out of 26 lymph nodes were found to be positive for adenocarcinoma.

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Malignant causes of back pain

Between 0.5 and 1% of patients presenting with back pain will have a neoplasm. Of these, 4% will present with compression fractures and 1–3% with a prolapsed disc. Clinical warning signs ('red flags') which should raise the suspicion of malignancy as a cause of back pain include non-mechanical pain (unrelenting, unaffected by position, not relieved with rest, nocturnal pain), age of onset <20 years or >55 years, thoracic pain, unexplained weight loss, history of malignancy, fever, no improvement despite 4–6 weeks of treatment and progressive neurological impairment.

The spine is usually affected by metastatic tumours (most commonly from breast, lung, prostate, thyroid, kidney and bowel) and occasionally by primary neoplasms. These may develop within the spinal vertebrae and spread into the spinal cord, or arise within the spinal canal or spinal medulla directly. In adults, 75% of tumours are extradural (mainly metastases, myeloma, or lymphoma); 20% are intradural (meningioma or schwannoma) and 5% are intramedullary (astrocytoma or ependymoma). The thoracic vertebrae are most affected by metastasis although it may occur at any site.

Neurological symptoms follow as collapse of the vertebral body allows neoplastic tissue and softened bone to extrude into the extradural space and compress the spinal cord or cauda equina. In general, the more severe the neurological deficit and the longer the duration of symptoms, the less likely the chances for recovery. Plain X-ray may reveal osteolytic lesions or vertebral collapse (*Figure 1*). Magnetic resonance imaging (MRI) may demonstrate extradural compression (*Figure 2*) or the presence of multiple lesions.

Radiotherapy is the current standard treatment for patients with metastatic spinal cord compression. Surgical intervention is required in cases of spinal instability with or without neural compression or in patients who fail to respond to radiation therapy (i.e. progressive neurological deficit or persistent debilitating pain). **BJHM**

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Figure 1. Plain X-ray of the lumbar spine showing an anterior wedge fracture of L3 in a previously healthy 49-year-old woman with a 2-month history of non-mechanical back pain and progressing weakness in the right lower limb.

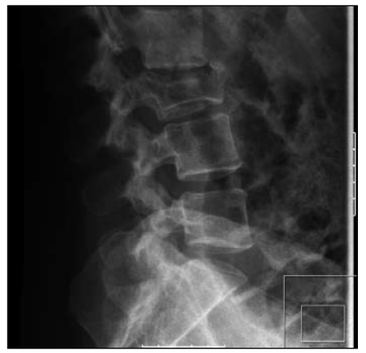

Figure 2. Magnetic resonance imaging of the lumbar spine in the same patient showing destruction of the L3 vertebral body with extension of the neoplastic tissue and softened bone into the extradural space causing spinal cord compression. Bone biopsy confirmed the lesion to be myeloma.