idiopathic pancreatitis. We have found only three reports^{1–3} on pancreatitis associated with a gastric polyp and none with repeated episodes as in the present case.

Gastroduodenal intussusception is most commonly due to a benign tumour,¹ and Gardner's syndrome (familial adenomatous polyposis)² is one condition in which it has caused pancreatitis.²

A cystic lesion in the vicinity of the pancreas on CT scan, in a patient with a history of acute pancreatitis, is usually interpreted as pseudocyst. In the patient reported here this misinterpretation led to a delay in diagnosis and definitive treatment.

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Disseminated herpes simplex after total skin electron beam radiotherapy for mycosis fungoides

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In mycosis fungoides, CD4⁺ T-cells localize to the skin, resulting in patches, plaques, tumours, and erythroderma. Superficial radiotherapy delivered with electrons to all skin surfaces is an important component of treatment in this disease. A theoretical risk of total skin radiotherapy is cutaneous immunosuppression.

CASE HISTORY

At age 76, a woman with a history of chronic lymphocytic leukaemia, hypothyroidism, recurrent herpes zoster, and cutaneous basal and squamous cell carcinomas developed

mycosis fungoides involving the trunk and right shoulder. Despite treatment with multiple systemic and topical agents, the disease progressed and 4 years later she was referred to a radiation oncologist. At that time she had circular, deep pink, 1–6 cm patches and plaques on more than 10% of her body surface area localized primarily to the trunk, arms, and legs and with ulceration in the gluteal cleft and perineal region. There was no obvious adenopathy or hepatosplenomegaly.

Total skin radiotherapy was initiated with 6 mega-electronvolt electrons produced by a linear accelerator mounted on a dual-angle gantry. The patient was treated with 100 centigray (cGy) per day, 4 days per week, by a standard six-field technique.¹ After receiving 500 cGy she became severely fatigued and sought medical attention. On examination she had developed numerous 2–3 mm vesicles, pustules, yellow-crusts erosions, and haemorrhagic erosions, all of which were localized to the previously described plaques (Figure 1). A Tzanck smear gave an equivocal result but a direct immunofluorescence test for herpes simplex virus was positive. She was admitted to hospital and treated with intravenous aciclovir, 10 mg/kg every 8 hours for 24 hours then 5 mg/kg. By hospital day five, all of her lesions had encrusted. She was discharged on oral valaciclovir 1 g twice daily and has not resumed radiotherapy.

COMMENT

This patient developed Kaposi's varicelliform eruption (KVE), a disseminated vesicular eruption localized to plaques from a pre-existing lesion. KVE most frequently occurs in patients with atopic dermatitis, but has been reported in other skin diseases including mycosis fungoides.^{2–4} Herpes simplex virus is the primary cause, but individuals with atopic dermatitis who receive the smallpox (variola) vaccine may develop an identical clinical syndrome.

Figure 1 A cluster of vesicles and erosions arising on a pre-existing cutaneous plaque

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Risk factors for development of KVE remain unclear. A report of two cases in patients with atopic dermatitis treated with topical tacrolimus suggests that downregulation of cutaneous cell-mediated immunity can increase the risk in susceptible individuals.⁵ A case report of KVE after sun exposure points to non-ionizing radiation as another possible trigger.⁶ In the present case, the direct cause of the disseminated herpes simplex eruption is uncertain. Clearly, the patient was already immunosuppressed by her chronic lymphocytic leukaemia and by the various immunomodulatory treatments for mycosis fungoides; therefore, it is quite possible that the temporal relation between initiation of total skin radiotherapy and occurrence of KVE arose solely by chance. Alternatively, the radiotherapy may have further suppressed cutaneous cell-mediated immunity and thus put the patient at risk of herpes infection.

Only one other group has documented KVE after total skin electron therapy. In this report, a woman of 39 with plaque stage mycosis fungoides developed KVE after receiving 4600 cGy to all skin surfaces.³ From serial samples of peripheral blood, the investigators detected a transient depression of natural killer cell activity that coincided with the start of radiotherapy and may have contributed to the development of KVE.

Our case report illustrates that KVE can occur even after small doses of external radiation. Radiation oncologists, dermatologists, and general practitioners should be aware that a vesicular eruption during the course of total skin radiotherapy may signify a life-threatening condition that requires immediate diagnosis and intravenous antiviral therapy. In view of the rarity of this complication, we do not advise routine antiviral prophylaxis during total skin radiotherapy.

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Paraplegia after elective repair of an infrarenal aortic aneurysm

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Paraplegia is a rare but devastating complication of thoracic/thoraco-abdominal aortic aneurysm repair. The incidence after repair of infrarenal aneurysms is lower, at 1.4–2.0% for emergency cases and 0.1–0.2% for elective cases.^{1,2}

CASE HISTORY

A man of 68 was seen in the outpatient department with a symptomatic abdominal aortic aneurysm. Ultrasound scanning showed a 6.9 cm infrarenal aneurysm with involvement of the right external iliac artery together with bilateral femoral aneurysms. He was fit and well with stable cardiorespiratory status. Elective aneurysmal repair was conducted under general anaesthesia. Aorta and groin vessels were dissected and controlled and heparin (5000 U) was infused intravenously. An aorto-bifemoral graft was sutured in place. The distal end of the aorta was oversewn and the two limbs of the graft were joined, end to end, to the distal common femoral arteries after resection of the femoral aneurysms. The operating time was 3 hours 15 minutes and the aorta cross-clamping time was 1 hour and 45 minutes. There was no significant hypotension during the operative procedure. The patient had an epidural catheter for analgesia.

On the first postoperative day the patient developed flaccid paraplegia and became incontinent of urine and faeces. The paralysis affected distal muscles more than proximal muscles. Deep tendon reflexes were absent and plantars were equivocal. Sensation was diminished from L4 to S1. The findings on distal vascular examination had not changed, with palpable femoral, dorsalis pedis and posterior tibial arteries. Initially we suspected that the paresis was due to the effects of epidural analgesia, which we discontinued. An MRI scan ruled out compression or infarction of the spinal cord. Next day there was no improvement, so we

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