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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J R Soc Med 2003;**96**:501–503

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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revascularized his left internal iliac artery by use of a jump graft from the right limb of the aorto-bifurcation graft. Electromyographic studies indicated axonal degeneration of peripheral motor and sensory nerves supplying the lower limbs, with the sciatic nerve more affected than the tibial nerves. These findings suggested an acute compressive axonotmetic lesion affecting the lumbosacral plexus. A subsequent MRI scan ruled out any compressive lesion in the pelvis. With intense physiotherapy the patient made slow but steady progress, becoming able to walk with a stick and recovering continence.

COMMENT

Understanding of the pathophysiology of this complication demands a brief account of the blood supply of the spinal cord (Figure 1). Three spinal arteries, one anterior and a pair of posterior spinal arteries from the vertebral arteries, supply the cord. The anterior spinal artery is the principal artery of the three, supplying the anterior two-thirds of the cord, including the critical motor area.³ Segmental arteries from subclavian, intercostal, upper lumbar and branches from the internal iliac and middle sacral arteries regularly feed the anterior spinal artery. The largest of these segmental arteries is the great radicular artery of Adamkiewicz or *arteria radicularis magna*, which originates

Figure 1 Arterial supply of the spinal cord [Reproduced from Fitzgerald MJT, J Folan Curran. *Clinical Neuroanatomy and Related Neuroscience*. New York: Saunders, 2002]

Table 1 Classification of ischaemic injuries to the spinal cord and lumbosacral roots or plexus

Classification	Site of ischaemia	Neurological deficit
I	Distal thoracolumbar cord	Bilateral flaccid paraplegia and sensory loss. Bowel and bladder dysfunction
II	Anterior two-thirds of the spinal cord	Bilateral flaccid paraplegia and loss of pain and temperature sensation; proprioception and vibratory sensation maintained
III	Lumbosacral roots with/without patchy infarcts of cord	Bilateral asymptomatic paraparesis with or without bowel and bladder incontinence
IV	Lumbosacral plexus	Bilateral asymmetric paraparesis with/without bowel and bladder incontinence. Preservation of paraspinal muscle innervation
V	Segmental infarction of spinal cord	Bilateral spastic paraplasia with sensory loss
VI	Posterior third of spinal cord	Loss of proprioception and vibratory sensation

as a branch from a left intercostal artery between T9 and T12 in 75% of patients, T5 and T8 in 15%, and L1 and L2 in 10%.⁴ Operative interference with this artery is implicated as one of the causes of paraplegia in thoraco-abdominal repair. The inferior mesenteric artery and branches of the profunda femoris join the hypogastric artery and its branches to form the pelvic circulation. The pelvic circulation supplies some of the distal spinal cord. At present there are no angiographic techniques to display the entire cord supply in an individual patient; this limits our ability to avoid neurological complications completely during aortic operations. Gloviczki *et al.*² classified ischaemic injuries to the spinal cord and lumbosacral roots or plexus according to their location and extent (Table 1).

Interference with pelvic blood supply, prolonged aortic cross-clamping, prolonged suprarenal clamping, intraoperative hypotension, thromboembolic phenomena and interference with a low-origin *arteria radicularis magna* have all been suggested as possible causes of spinal cord ischaemia.⁵ MRI should be done to rule out a compressive lesion, since this is potentially curable. Electromyographic studies, for indicating the exact site of the lesion, will help in prognosis: patients with an ischaemic injury to the lumbosacral roots or plexus recover best. In the present case the injury to the lumbosacral plexus was probably due to interruption of the collateral flow via the branches of the internal iliac arteries.

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Cutaneous T-cell lymphoma with adrenal insufficiency

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J R Soc Med 2003;**96**:503–504

Lymphomas commonly metastasize to the adrenal glands,¹ but they seldom cause adrenal insufficiency.²

CASE HISTORY

A woman of 74 was admitted for investigation of nausea, dizziness exacerbated by head movements, anorexia and progressive weight loss. Eight months earlier, cutaneous T cell lymphoma (CTCL) had been diagnosed, without extracutaneous spread as judged by CT scans of chest and abdomen. After radiotherapy, the disease had remitted. She now had postural hypotension (blood pressure 139/73 mmHg lying, 120/88 mmHg standing) and cutaneous nodules were noted over both upper limbs consistent with recurrence of CTCL. There was slight hyponatraemia (Na^+ 134 mmol/L) but potassium was normal (4.3 mmol/L). All other electrolytes, liver function tests, and the full blood count were normal. However, as her symptoms rapidly got worse, the serum sodium fell to 119 mmol/L and the serum potassium rose to 5.0 mmol/L. The 9 am cortisol was low at 121 nmol/L and a short Synacthen (tetracosactide) test showed adrenal insufficiency (cortisol 117 nmol/L 30 minutes after 250 mg tetracosactide). Hydrocortisone 50 mg three times daily was prescribed and over the next five days the patient made a remarkable recovery, with the sodium rising to 136 mmol/L and the potassium falling to

4.1 mmol/L. An MRI scan of the brain was normal but a CT scan of the abdomen showed bilateral suprarenal masses (Figure 1). Eight days later the patient suddenly lost consciousness. An Addisonian crisis was suspected but there was no response to intravenous hydrocortisone. A CT scan of the brain then revealed a frontal haemorrhage, from which she died five days later. At necropsy the tumour was seen to have infiltrated both kidneys and both adrenal glands. Deposits were also seen in the liver and in one para-aortic lymph node. The pituitary gland was macroscopically normal. On histological examination the cutaneous plaques were positive for T cell markers (CD3; CD43) and negative for B cell markers (CD20; CD79a). Staining for CD30 and ALK protein was also negative. The histology of the tumour infiltrating the adrenal glands matched that of the cutaneous lymphoma.

COMMENT

For any tumour to cause adrenal insufficiency it has to destroy over 90% of the glandular tissue in both adrenal glands.³ This is a rare event with lymphomas: at the Mayo Clinic only 5 such cases were seen in 18 years.² However, lymphomas do commonly metastasize to the adrenals.³ The lymphoma in this case was classified as a CD30-ve CTCL (EORTC⁴) or peripheral T cell lymphoma, type unspecified (WHO⁵). Although one subtype of CTCL, mycosis fungoides, has been associated with adrenal insufficiency we have found no previous cases with CD30-ve CTCL. None of the histological sections taken at necropsy showed the tumour invading the vessel walls, making an alternative diagnosis of lymphomatous vasculitis unlikely. Systemic

Figure 1 CT scan of abdomen showing enlargement of both adrenal glands

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