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Cutaneous T-cell lymphoma with adrenal insufficiency

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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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anaplastic large-cell carcinoma, one of the main differential diagnoses, was ruled out since the lymphoma was ALK negative and negative for the t(2;5) translocation in the TCR receptor, and no tumour cells were found in the vascular endothelium.

The importance of CD30 for prognosis has only lately been realized.⁶ It is a type 1 membrane glycoprotein and belongs to the tumour necrosis factor receptor family. CD30+ve CTCL is indolent whereas CD30-ve CTCL is aggressive. This is reflected in their 5-year survival rates of 90% and 15%, respectively.

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Lifelong migraine aura without headache: change of pattern with upper respiratory infection

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Changes in the features of migraine during a lifetime may offer clues to the pathogenesis.

CASE HISTORY

A retired professor of psychiatry (SIC) experienced migraine aura without headache from the age of 20, about once in 2 years. Without prodromal symptoms he suddenly

became aware of not being able to see properly in the left visual field and had difficulty with reading. Within two minutes a bright light appeared, enlarged into three-quarters of a circle enclosed by zigzag scintillating lines coloured black with a golden edge surrounding a scotoma (Figure 1). The visual symptoms lasted about twenty minutes then gradually disappeared; he never experienced headache or gastrointestinal, visual or mental symptoms after his aura. He was unable to identify any precipitants for the visual disturbance, which could occur during reading or other activities.

At the age of 76 there was an episode with different features. A typical attack occurred but as the day progressed he experienced several more. On the next day an aura lasted 2 hours. The following day two more attacks occurred, each of ten minutes' duration. Throughout the subsequent day similar episodes recurred, about twelve in all. All auras were in the left visual field. Later that day he had a severe, steady pain behind the right eye and began to feel cold although the day was hot; his temperature was 37.6°C. He assumed that the pyrexia was due to sinusitis, as he had developed a yellow nasal discharge in the previous few days. With amoxicillin the temperature became normal within a day and the nasal discharge cleared. The last visual episode, still on the left, occurred on the subsequent day, lasting twenty minutes, but was joined by a right-sided headache starting ten minutes later; the aura and headache persisted together for another thirty minutes, when the aura disappeared, although the headache continued for a further hour. He then noticed a faint glow surrounding bright white objects—for example, around the edges of the kitchen dishwasher—which lasted over the next few hours. There were no further attacks in the subsequent nine months.

Figure 1 Scintillations surrounding scotoma drawn up to full development, at which they persisted for a further 10 minutes before gradually disappearing

The previous history is relevant. A year earlier, mild effort intolerance had been treated with a coronary artery stent. At that time a raised blood cholesterol was found and was restored to normal with atorvastatin. He noticed an increased nasal discharge and a hoarse voice—a rare side effect. A month before the above episode, his general practitioner therefore changed the statin to simvastatin, which both increased and thickened the discharge which

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