haematoma at the level of the right basal ganglia with collapse of the lateral ventricle. Serum calcium measurements ranged between 3.07–3.6 mmol/l. Serum phosphorus was 0.74 mmol/l. Parathyroid hormone concentration, determined by immunoassay, was over 2700 pg/ml. Urinary cAMP excretion was 0.9 mmol/l. Serum creatinine was 141 μmol/l. Urinalysis was normal. Abdominal ultrasound showed increased echogenicity in both kidneys. No phaeochromocytoma was detected. Intravenous urography was normal. Bone X-rays revealed subcortical resorption. Cerebral angiography performed 3 months after the acute episode was considered normal. A 3 × 1.5 × 1 cm parathyroid adenoma was excised from the lower left parathyroid gland. Metabolic anomalies reverted to normal. Propanolol 120 mg/day was instated to maintain blood pressure within normal limits but a severe residual hemiplegia persisted.

Young adults with non-traumatic intracerebral haemorrhage are a heterogeneous group. A cause can be established in most patients, but in our patient no arteriovenous malformation, ruptured saccular aneurysm or sympathomimetic drug abuse could be demonstrated. Arterial hypertension undoubtedly played a determinant role. Arterial hypertension was notable in 6 of the 343 cases of hyperparathyroidism studied by Cope. The metabolic anomalies induced in vascular walls by excess parathyroid hormone or vitamin D\textsuperscript{3} can contribute to this grave complication of hyperparathyroidism.

E. Garcia-Albea
J. Medina
A. Ucles

Neurology Service
Hospital de Alcalá de Henares
Alcalá de Henares
28800 Madrid, Spain

we report a case of bilateral acoustic neuropathy attributable to vincristine.

A 77 year old woman with 3 weeks' history of weight loss, pruritis and generalized lymphadenopathy, was admitted to the hospital with right lower lobe pneumonia. Lymph node biopsy confirmed the diagnosis of low grade malignancy non-Hodgkin’s lymphoma (centrocytic centroblastic in type).

After she had recovered from pneumonia, a regime of cytotoxic therapy (vincristine, chlorambucil and prednisolone in the conventional doses) was commenced. A few days later she developed rhinorrhea, dry cough, and impaired hearing in both ears. She was not able to hear whispered words; however, conversational voice was heard if it was loud. Examination showed congested throat and nasal passages with normal external auditory canals and drums. Hearing returned to normal as the upper respiratory tract infection resolved in a few days.

However, soon after the second course of chemotherapy was given, she became profoundly deaf in both ears. Conversational voice was not heard, no matter how loud it was. Auroscopic examination was normal. A tuning fork test (512 Hz) showed that although both air and bone conduction were considerably impaired, the former was less severely affected, indicating a sensineural type of deafness. Vincristine was suspected as the cause of her hearing loss and it was withdrawn from the regime. Apart from sluggish ankle jerks she did not have other signs of vincristine toxicity. Hearing returned to near pre-treatment level in the ensuing few weeks.

The anti-tumour properties of vinca alkaloids probably stem from their capacity to bind cellular tubulin. This results in inhibition of microtubule formation, an essential step in cellular division, causing the arrest of the dividing cells at the metaphase. Neutrotubules seem to be vulnerable to some of the actions of vinca alkaloids. Their disruption is thought to be responsible for impairment of the axoplasmic transport mechanism. Hence vinca alkaloids are thought to cause axonal rather than demyelinating type of neuropathy.\textsuperscript{9,10}

Soon after their introduction in 1963 these agents were reported to cause peripheral neuropathy.\textsuperscript{11,12} This has consistently manifested itself as impairment of the tenoachilles reflex.\textsuperscript{1,2} Casey et al. reported that all patients who received vincristine developed evidence of peripheral neuropathy clinically or on electrophysiological testing. Nevertheless the neuropathy was largely reversible if the dose of vincristine was reduced or it was discontinued.\textsuperscript{1} Cranial neuropathy is far less frequent (around 10%), and involvement of the VIII nerve is extremely rare.\textsuperscript{4} There has been only one previous case report (to our knowledge) of vincristine-induced acoustic neuropathy.\textsuperscript{13} Interestingly, that case was not dissimilar from ours. The patient, like ours, was an elderly lady with non-Hodgkin’s lymphoma. Deafness after the first course of chemotherapy was attributed partly to acute otitis media. However, the suspected diagnosis of vincristine neuropathy became clear after the second course was initiated. This supports the view that hyperaemia of the inner ear, due to the accompanying infection, has probably exposed the sensitive neurmechanisms to high doses of vincristine, and might have precipitated the neuropathy.

Awareness of this serious complication of vincristine requires close monitoring of the auditory function in the

References


Partially reversible nerve deafness due to vincristine

Sir,

Sensory-motor peripheral neuropathy is a frequent complication of vincristine therapy.\textsuperscript{1,3} Although involvement of various cranial nerves has also been reported, for some reason the eighth cranial nerve is usually spared.\textsuperscript{4,6} Here

Downloaded from http://pmj.bmj.com/ on April 19, 2017 - Published by group.bmj.com
elderly patient, or those with established hearing impairment. The appearance of hearing loss or deterioration of auditory function would require discontinuation of treatment. Concomitant upper respiratory tract infection or ear infection should perhaps indicate deferring the commencement of treatment with vincristine until the episode is over.

H. Yousif (Al-Najjar)
S.G.N. Richardson
W.A. Saunders
Department of Medicine,
Russells Hall and Guest Hospitals,
Dudley, West Midlands, UK
Correspondence: H. Jousif (Al-Najjar)
P.O. Box 4078
Al Shuwaikh 13041,
Kuwait

References

Chemical de-bulking of desmoid tumours
Sir,
Surgical treatment of desmoid tumours is accompanied by a high recurrence rate. Recent reports have suggested that the use of tamoxifen may induce a remission. Our case adds to the very limited world literature supporting its use in this condition.
A pre-menopausal 51 year old woman with polyposis coli had required a colectomy and ileo-rectal anastomosis for (Duke's A) carcinoma of the ascending colon. She re-presented 18 months later with abdominal pain and distension. A solid, tender, bi-lobed mass was palpable per abdomen. Computerized tomography demonstrated a mass in the left upper quadrant and a second, 11 x 8 x 8 cm mass to the right of the aortic bifurcation (Figure 1). At laparotomy the former tumour was resected, but the latter was inoperable due to the direct involvement of the inferior vena cava. Histological examination confirmed the diagnosis of desmoid tumour.

![Figure 1](http://pmj.bmj.com/) Computerized tomogram of the abdomen and pelvis showing a large mass occupying the left upper quadrant.

![Figure 2](http://pmj.bmj.com/) Section of the resected desmoid showing a moderately cellular spindle cell tumour with very occasional mitotic figures (H&E x 41).
Partially reversible nerve deafness due to vincristine.

H. Yousif, S. G. Richardson and W. A. Saunders

doi: 10.1136/pgmj.66.778.688