The most frequently prescribed diuretics were indapamide (45.1%), furosemide (18.9%), hydrochlorothiazide (18.4%) and amiloride (12.7%); 29.9% of diuretics were prescribed as monotherapy. Of the angiotensin converting enzyme (ACE) inhibitors 31.5% were monotherapy, compared with 9.5% of the calcium-channel blockers and 54% of the beta-blockers. Among β-blockers, atenolol accounted for 49.2%, propranolol 44.4%, and bisoprolol 6.4%; 174 prescriptions (42.3%) included non-antihypertensive drugs.

Table II compares the retail costs of generic and brand name products for some of the widely used antihypertensives.

The low percentage of generic prescribing, infrequent use of fixed drug combinations, high number of incomplete prescriptions, tendency to prescribe more drugs for medical aid patients relative to those paying cash, and inconsistency of our findings with available guidelines are troubling. For instance, although diuretics were the commonest prescribed group, indapamide, not hydrochlorothiazide, was the most popular, and more than half the β-blockers were prescribed as monotherapy. South African and international guidelines, on the other hand, call for the use of a thiazide diuretic, followed by β-blocker plus thiazide, but not β-blocker alone.[7-10] The guidelines discourage the use of propranolol in favour of atenolol,[8] yet we found that the two were prescribed equally.

Most prescriptions had no diagnosis, which limits the evaluation of appropriateness of drug use.[11] The greater prescription of brand name drugs relative to the generics, with attendant higher costs, may result in irregular supply of drugs and poor control of blood pressure.[12,13] The polypharmacy noted (10% with five or more items) raises the possibility of irrational prescribing with higher likelihood of non-compliance.[14,15]

Prescribers need to write complete prescriptions to ensure proper and safe use of drugs. These concerns should be addressed through CPD activities, which should incorporate issues of treatment guidelines for common conditions such as hypertension.

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Superficial siderosis — case report and review

To the Editor: Superficial siderosis (SS) is a rare but distinct syndrome afflicting the central nervous system (CNS) characterised by sensorineural deafness (SND), cerebellar ataxia, dementia and myelopathy and caused by chronic bleeding into the subarachnoid space.[1-4] The source of this bleeding is identified in only half of cases described. Pathologically there is deposition of haemosiderin in those parts of the CNS lying in close proximity to the cerebrospinal fluid (CSF), including the subpial and subependymal margins.[1] The haemosiderin deposition causes gliosis, neuronal loss and demyelination.[1] The peripheral nervous system is not affected.[1]

Hamill first described this condition in 1908, and in 1995, 87 cases of superficial siderosis were reported in the literature worldwide.[1] It occurs more commonly in males (male/female 3:1) and has been described in patients with an age range of 14 - 77 years.[1] No specific racial predilection has been noted.

We describe here a 58-year-old South African woman with SS in whom no cause has been identified. As far as we are aware there is no report of a case(s) of SS from South Africa.

Case presentation

A 58-year-old woman presented with a history of deafness and gait disturbance. The deafness had started in the left ear 6 years before presentation, and within 6 months of onset it

affected the right ear. She was investigated for the deafness but no cause was identified. A diagnosis of idiopathic or hereditary hearing loss was made at the time. She was referred to us because she subsequently developed difficulty with walking. She described the gait disturbance as one in which she experienced stiffness in her legs, with loss of balance and co-ordination. These symptoms began 1 - 2 years after the deafness. More recently, she has developed forgetfulness.

The patient has a longstanding history of hypertension and is on treatment with perindopril and hydrochlorothiazide. There were no other vascular risk factors and no family history of deafness or any neurological disorder. There was no history of occupational or environmental toxin exposure.

Clinically she had a blood pressure of 160/90 mmHg. Her general examination was normal. She had bilateral deafness (SND) with short-term memory loss and impairment of intellectual abilities. She was unable to perform simple arithmetical tasks and had difficulty recalling events described during the interview. She had prominent cerebellar dysfunction, which included both axial (truncal and gait) ataxia and appendicular signs (dysmetria and dysdiadochokinesis). There was no spasticity but lower limb reflexes were exaggerated. The plantar responses were flexor. She had anosmia and low gain saccadic eye movements. There was no extrapyramidal involvement.

Blood investigations including full blood count, erythrocyte sedimentation rate (ESR), urea and electrolytes, liver function tests, calcium phosphate and magnesium, pyruvate and lactate, serum iron, serological tests for syphilis, antinuclear factor, thyroid function tests and HIV enzyme-linked immunosorbent assay (ELISA) were all normal or negative. A computed tomography (CT) scan of the brain showed bilateral basal ganglia calcification. CSF examination was normal. Audiometry confirmed bilateral sensorineural basis for the deafness. The diagnosis of SS was made after cranial magnetic resonance imaging (MRI). On the T_2-weighted fast spin echo (FFE) imaging (Fig. 1) hypointense signals were noted in the basal ganglia, tentorium, basal cisterns, around the brainstem and cervical spinal cord. The signals were in keeping with haemosiderin deposition. A four-vessel cerebral angiogram was normal. The source of bleeding could not be identified. A spinal MRI performed at a later stage showed leptomeningeal haemosiderosis throughout the spinal cord and cauda equina. The patient is currently receiving treatment for hypertension with no specific treatment for the SS. Her condition is stable.

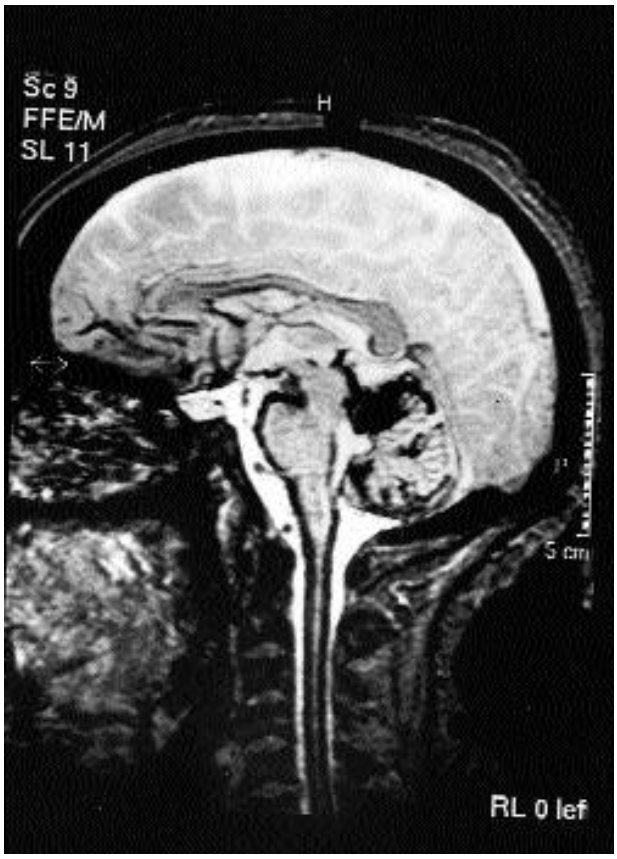

Fig. 1. Saggital T_2 fast spin echo magnetic resonance image showing hypointense rim of haemosiderin around the brainstem, cerebellum and spinal cord.

Discussion

The diagnosis of SS in our patient was made radiologically as is usually the case. Her clinical findings of deafness, cerebellar ataxia, dementia and myelopathy are characteristic of this condition but may occur in a variety of other conditions. A differential diagnosis of sarcoidosis, spinocerebellar ataxia, or mitochondrial cytopathy, among others, has to be considered when one is faced with this combination of symptoms. MRI is, however, diagnostic for SS and has allowed for antemortem diagnosis of this condition.

The MRI features as highlighted in our case include hypointense rims around the brainstem, cerebellum, eighth cranial nerve and spinal cord on gradient echo or routine spin echo T_2-weighted imaging sequences. Gradient echo T_2 studies are superior to routine spin echo sequences; however, either sequence performed routinely during MRI is extremely sensitive to detecting haemosiderin.[3] CT scans are unhelpful and may be normal or show nonspecific cerebellar atrophy.[3]

It is therefore likely that a large percentage of possible cases of SS are missed. MRI is not routinely available and as yet is not available in all tertiary care state and university hospitals in this country.

Laboratory studies on peripheral blood are unremarkable and therefore not helpful in diagnosis.[1] CSF studies are likewise unhelpful. They are normal or occasionally show an elevated CSF protein.[1] Evidence of haemorrhage or

xanthochromia is present in only 50% of specimens.[1] Iron and ferritin levels in blood and CSF are normal.[1,2] Diagnosis of SS therefore rests entirely on clinical suspicion and is confirmed by MRI. In terms of the clinical features, bilateral, progressive hearing impairment is found in 95% of cases.[1] Gait and limb ataxia with or without nystagmus and dysarthria comprise the cerebellar syndrome in 88% of cases.[1] Myelopathy may take the form of bilateral pyramidal tract signs (50%) or hyperreflexia (26%).[1] Additional manifestations include dementia and other cranial nerve involvement. Anosmia is frequently present.[1] Optic nerve involvement, anisocoria, trigeminal sensory loss and facial nerve palsy are rare findings.[1] It is not clear whether SS predisposes to seizures.[1]

The selective vulnerability of certain parts of the CNS can be explained on an anatomical basis. Chronic exposure to haemoglobin and its breakdown products leads to accumulation of ferritin and haemosiderin in subpial microglia and cerebellar Bergmann's glia, which contain the biochemical apparatus for ferritin synthesis.[2] The cerebellar vermal and paravermal regions lie in close proximity to the roof of the fourth ventricle thereby increasing exposure of these surfaces to material circulating in the CSF.[1] Similarly the eighth cranial nerve has a long glial segment and courses through the pontine cistern.[1,3]

Treatment remains unsatisfactory. A source of subarachnoid bleeding should be sought and surgical removal or ablation considered if a potentially treatable cause is identified. Surgery has only been described in a few cases and long-term results are unknown; the possibility exists that the pathological process continues despite cessation of bleeding.[3] Pharmacological therapy with iron chelators, copper chelators and antioxidants has been disappointing.[1]

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