ported in the Queensland patients following 8 weeks to 9 weeks treatment with albendazole.[3] The total number of parasites within the muscles in our patient depends not only on the duration of the infection, but also the severity of auto-reinfection and the duration (5 months) of immunosuppressive therapy. Auto-reinfection occurs when third-stage larvae rupture through the body wall and cuticle of the female in the cephalic region.[2] Administration of corticosteroids in three of the reported patients prior to the diagnosis of *H. perplexum* probably resulted in a substantial proliferation of the nematodes. Our patient has responded very well with weight gain, improvement in muscle strength and bulk and decreased serum CK concentrations, and 3 months after finishing albendazole therapy, his serum CK was only mildly elevated at 470 U/L.

We report this sixth known patient with myositis due to *H. perplexum*, and highlight the clinical severity of the infection, with potential for refractory disease if the inflammation is detected late in the illness when significant muscle fibrosis has occurred. This is a uniquely Australian disease, which can mimic idiopathic polymyositis but with potential for mortality and significant permanent morbidity if the diagnosis is delayed. Although all patients except ours were associated with peripheral blood eosinophilia, all biopsies did show eosinophils in the muscle biopsy, which should alert the pathologist to a possible parasitic infestation. Patients with a polymyositis phenotype who deteriorated on immunosuppressive therapy should be investigated for the possibility of parasitic myositis.

Acknowledgements

We thank Drs. Craig Costello, Phillip Marshall, John Archer and Jon Reimers for their follow-up of the previously reported cases from northern Queensland.

References

1. Dennett X, Siejka SJ, Andrews JR, et al. Polymyositis caused by a new nematode. *Med J Aust* 1998;**168**:226–7.
2. Spratt DM, Beveridge I, Andrews JR, et al. *Haycocknema perplexum* n.g., n. sp. (Nematoda: Robertdollfusidae): an intramyofibre parasite in man. *Syst Parasitol* 1999;**43**:123–31.
3. Basuroy R, Pennisi R, Robertson T, et al. Parasitic myositis in tropical Australia. *Med J Aust* 2008;**188**:254–6.
4. Spratt DM, Nicholas WL. Morphological evidence for the systematic position of the order Muspiceida (Nematoda). *Trans Roy Soc SA* 2002;**126**:51–62.

doi:http://dx.doi.org/10.1016/j.jocn.2012.06.019

L-dopa responsive parkinsonism secondary to a subdural haematoma

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ARTICLE INFO

Article history:
Received 10 June 2012
Accepted 17 June 2012

Keywords:
L-dopa
Parkinsonism
Subdural haematoma

ABSTRACT

We report a 53-year-old woman who developed a reversible, L-dopa responsive parkinsonian syndrome in the context of a recurrent right-sided subdural haematoma. The syndrome occurred during a prolonged stay in hospital and resolved completely during the following year. Parkinsonism is a rare but recognised complication of subdural haematoma.

1. Case report

A 53-year-old woman was admitted following a 10 day history of constant and worsening headache in the frontoparietal region. There was no significant past medical history or family history, and her only current medication was hormone replacement therapy. Clinical examination was normal. Following a head CT scan she was diagnosed with a right-sided subdural haematoma with midline shift, and she underwent burr hole drainage.

Over the next 3 days, two further evacuation procedures were required, followed by a right craniotomy, with implantation of a cranial flap in the anterior abdominal wall. The patient was then admitted to the neurosciences critical care unit (NCCU) as she had begun to develop left-sided focal seizures and pneumonia. A few days later she had some left-sided weakness with increased tone in all four limbs, clonus and brisk reflexes. After 3 weeks at the NCCU she stabilised and was returned to the neurology ward. At no point was she given any dopamine-blocking drugs.

After several weeks on the ward, the patient had continued fever and a reduced Glasgow Coma Scale score of 10 and she began to have right-sided focal seizures affecting the hand and face. She was diagnosed with an abdominal abscess associated with the cranial bone flap, which was drained and the flap removed. She initially improved, but 3 weeks later, when she had been in hospital for 4 months, she was re-admitted to the NCCU for a second time with pneumonia. At this stage it was noted for the first time that she had developed bilateral parkinsonian signs including tremor, bradykinesia, rigidity, cogwheeling and hypomimia, in the absence of any drugs that could cause this. There was also a residual left hemiparesis. A dopamine transporter scan showed diminished visualisation of the corpus striatum on both sides, right more than left (Fig. 1). She was tried on a low dose of co-careldopa (25 mg/100 mg 3 times daily) without any improvement initially. However, on higher doses of co-careldopa (50 mg/250 mg 5 times daily) and cabergolene (7 mg once daily) her axial stiffness improved, followed by her rigidity.

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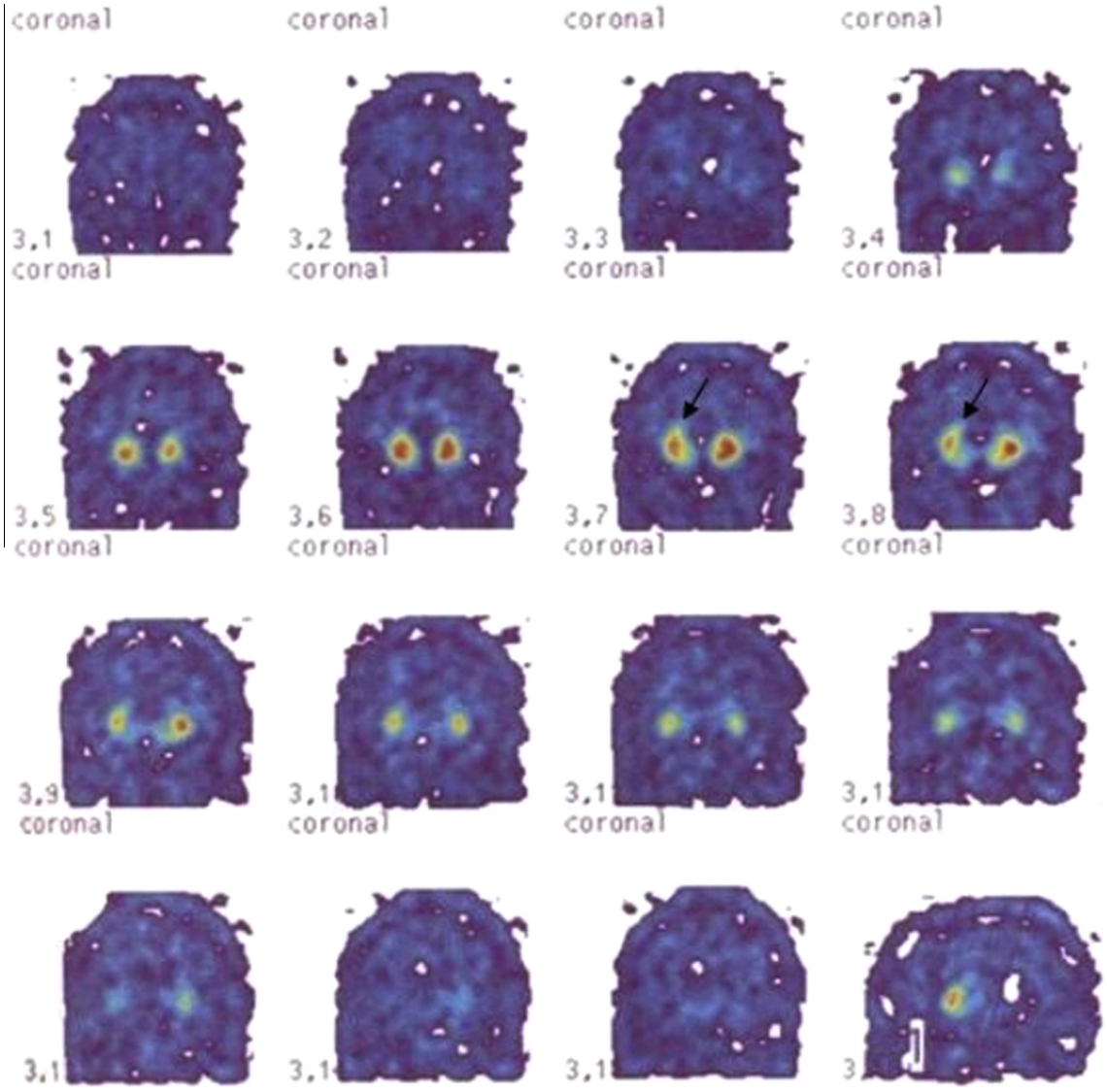

Fig. 1. Coronal dopamine transporter scans showing diminished visualisation of the corpus striatum on both sides, right more than left (arrows). (This figure is available in colour at www.sciencedirect.com.)

Table 1
Patients with parkinsonism caused or worsened by subdural haematoma reported in the literature

Patient no.	Age (years)	Sex	Other features	Responsiveness to levodopa	Outcome	Reference
1	52	M	Parkinsonism only	Not given	Complete remission	1999[1]
2	62	M	Headache, confusion, fatigue	Not given	Complete remission	
3	66	M	Headache, confusion, fatigue	Yes	Complete remission	
4	38	F	Headache, confusion	Not given	Complete remission	
5	74	M	Memory deficit	Not given	Complete remission	
6	72	M	Parkisonism only	Not given	Marked improvement	
7	78	F	Confusion, incontinence	Decreased response in pre-existing PD	Recovery to pre-morbid function	
8	48	M	Headache, fatigue	Not given	Complete remission	
9	73	M	Parkinsonism only	Not given	Complete remission	
10	61	M	Parkinsonism only	Not given	Complete remission	
11	83	M	Incontinence	Not given	Partial remission	
12	60	F	Headaches, lethargy	Not given	Partial remission	
13	58	M	Headache	Not given	Complete remission	
14	75	M	Headache	Not given	Complete remission	
15	66	M	Parkinsonism only	Not given	Complete remission	
16	75	M	Confusion, hemiparesis (right)	Yes	Complete remission	
17	63	M	Parkinsonism only	No	Complete remission	
18	70	M	Incontinence, somnolence	Not given	Recovery to pre-morbid function	
19	82	M	Apathy	Decreased response	Recovery to pre-morbid function	
20	81	M	Confusion	No	Complete remission	2006[2]
21	65	F	Headache	Not given	Complete remission	2009[3]
22	73	M	Confusion, headache	Not given	Complete remission	2009[4]

F = female, M = male, PD = Parkinson's disease.

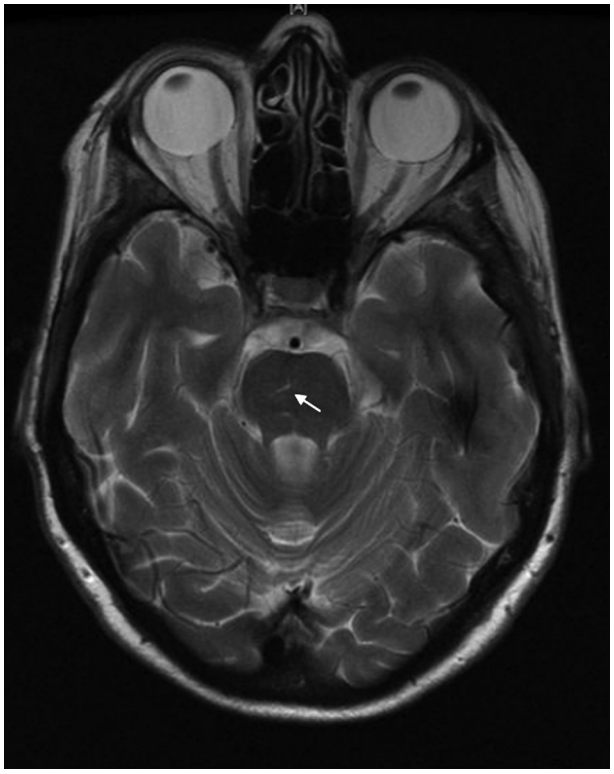

Fig. 2. Axial T2-weighted MRI showing areas of hyperintensity in the pons (arrow).

Over the next few months she improved and was able to transfer weight. Neuropsychological testing revealed some deficits with her working memory and attention, with a test of everyday attention score below the 25th percentile. She was transferred to a local rehabilitation centre for 5 months, and then was discharged home, being independent in most activities of daily living. Her parkinsonism completely resolved and all her treatment was stopped. Six months later she underwent cranioplasty without complications.

2. Discussion

There have been 22 patients with parkinsonism caused by subdural haematoma reported since 1963[1–4] (Table 1). After evacuation of the haematoma, complete remission was achieved eventually in 16 of the 22 patients. Our report highlights that such patients can make a full recovery without evidence that the disease might re-emerge. In addition, L-dopa therapy can have a beneficial effect in these patients in the short term, reducing their disability and shortening their recovery time.

Parkinsonism of acute onset is rare and may be secondary to infectious processes, metabolic disturbances, medications or structural lesions. For our patient, there was no evidence of intracranial infection, metabolic disturbances or medications that could have caused the extrapyramidal disorder. MRI revealed only subtle signal change in the pons with no evidence of signal change in the striatum (Fig. 2).

The mechanism by which a subdural haematoma may cause parkinsonism remains obscure. It may be an effect of the space occupying lesion causing partial coning and injury to the nigrostriatal tract. Indeed, similar syndromes have been described secondary to meningiomas and other tumours.[5] Pressure on the midbrain decreases the number of dopaminergic neurons, as has been reported in a patient with parkinsonism secondary to a brain tumour.[6] Alternatively, there could be a decline in the integrity of the nigrostriatal dopamine system, with reduced striatal dopamine content and sprouting with eventual recovery, similar to the changes seen in 1-methyl 4-phenyltetrahydropyridine lesions. Circulatory disturbances may have also occurred, such as disruption of the anterior choroidal artery impairing supply to the basal ganglia. There is some evidence that patients who develop movement disorders secondary to subdural haematoma may have preceding subclinical substantia nigra dysfunction, leaving the system more vulnerable to further insult.[7] However, there was no evidence of a pre-existing parkinsonian syndrome in our patient.

This report illustrates the propensity of subdural haematoma to have a myriad of clinical complications, and demonstrates the possibility of an acute parkinsonian syndrome secondary to an intracranial mass lesion, which is treatable and could easily be missed in the context of a seriously ill patient in an intensive care unit setting.

Conflict of Interest

Justin Cross receives support from the Biomedical Research Centre at Addenbrooke's Hospital. The authors declare that they have no further financial or other conflicts of interest in relation to this research and its publication.

References

1. Wiest RG, Burgunder JM, Krauss JK. Chronic subdural haematomas and Parkinsonian syndromes. *Acta Neurochir (Wien)* 1999;**141**:753–7 [discussion 757–758].
2. Suman S, Meenakshisundaram S, Woodhouse P. Bilateral chronic subdural haematoma: a reversible cause of parkinsonism. *J R Soc Med* 2006;**99**:91–2.
3. Bostantjopoulou S, Katsarou Z, Michael M, et al. Reversible parkinsonism due to chronic bilateral subdural hematomas. *J Clin Neurosci* 2009;**16**:458–60.
4. Giray S, Sarica FB, Sen O, et al. Parkinsonian syndrome associated with subacute subdural haematoma and its effective surgical treatment: a case report. *Neurol Neurochir Pol* 2009;**43**:289–92.
5. Bhatoe H. Movement disorders caused by brain tumours. *Neurol India* 1999;**47**:40–2.
6. García de Y'ebenes J, Gervas J, Iglesias J, et al. Biochemical findings in a case of parkinsonism secondary to brain tumor. *Ann Neurol* 1982;**11**:313–6.
7. Turjanski N, Pentland B, Lees A, et al. Parkinsonism associated with acute intracranial hematomas: an [18F]dopa positron-emission tomography study. *Mov Disord* 1997;**12**:1035–8.

doi:http://dx.doi.org/10.1016/j.jocn.2012.06.019