

Table 1 Motor nerve conduction for right ulnar nerve

	Site	Latency	Amplitude	Duration	Distance mm	CV m/s
1	Wrist	4.6 ms	0.426 mv	6.2 ms		
2	Below elbow	11.3 ms	0.373 mv	5.6 ms	200	30
3	Above elbow	18.2 ms	0.251 mv	6.4 ms	100	14

Impression: Motor nerve conduction study shows ulnar nerve compression in the cubital fossa. MS; + milliseconds; MV, + millivolts; CV, + conduction velocity.

DISCUSSION

Involvement of a peripheral nerve by a ganglion cyst is uncommon.¹ The most frequent location is the common peroneal nerve at the head of fibula, which comprise 75% of the total reported cases in the literature.¹⁻³ Suprascapular nerve involvement is the next most common.⁴ Involvement of the ulnar nerve at the elbow in the cubital tunnel is extremely rare.⁴⁻⁶ In the largest series, comprising 27 cases of ganglion cyst involving peripheral nerve, not a single case was observed at the cubital tunnel.²

In the majority of the cases, the diagnosis was not made until the time of surgical exploration,² as happened in our case.

Cystic degeneration of neurilemmomas, progressive enlargement of synovial rests displaced during embryological development, herniation of joint synovium and traumatic intraneural haemorrhage are some of the theories of pathogenesis.¹⁻⁵ Ganglion cysts may or may not be associated with an obvious connection with the neighboring joint. Our case had no connection with the elbow joint and the cyst was entirely in the cubital tunnel, flattening and compressing the ulnar nerve. Motor weakness and pain in the distribution of the involved peripheral nerve are the most common presenting symptoms, which were also observed in the present case. Detailed history, physical examination and electrophysiological studies are important in the initial evaluation of cases. Except in isolated instances of cyst recurrence, imaging studies by and large are not performed before operative exploration, although ultrasonography, CT and MRI have been performed for diagnosis in isolated cases. MRI⁴ is able to provide detailed morphology of the cyst and the nerve and other adjacent structures. Ganglion cysts are usually multilocular and nonenhancing lesions, usually hypointense to muscle on T1 weighted MRI and hyperintense on T2 weighted MRI. Ganglion cysts are well differentiated from nerve sheath tumours, glomus tumours and lipomas on MRI morphology.⁵

Complete excision is possible in the majority of cases² of epineural ganglion cyst, as in our case. However, in cases of intraneural cyst a complete excision may not be possible and the chance of recurrence exists.¹

The majority of patients benefit from microsurgical excision of a ganglion cyst of a peripheral nerve; however, motor recovery may not be complete if the ganglion cyst extensively involves the nerve and symptoms have been present for more than one year. In our case, symptoms were present for 3 years but fortunately the patient recovered completely following the microsurgical excision.

CONCLUSION

An epineural ganglion cyst of the ulnar nerve in the cubital tunnel is extremely rare. Progressive sensory and/or motor deficits and pain are the chief presenting symptoms. MRI is diagnostic. Microsurgical excision may be curative even in the presence of advanced neural compression and severe neurological deficits.

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Bilateral medial medullary infarction

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Summary We present a case of bilateral medial medullary infarction demonstrated by magnetic resonance imaging (MRI) and review 12 previously reported cases. We classify these 13 cases (including the present case) into two groups according to the extent of the ischemic region: type 1, the ischemic lesion developed from the medullary pyramid to the medial longitudinal fasciculus; type 2, the lesion was confined to the medullary pyramid.

A 71 year old man presented with slight disturbance of consciousness, dysarthria, disturbance of leftward gaze, no gag reflex and tetraparesis. He developed nearly complete horizontal ophthalmoplegia. MRI revealed upper medial medullary infarction bilaterally that extended to the pontomedullary junction. We propose that the prognosis of type 2 bilateral medial medullary infarction is better than that of type 1. Furthermore, the prognosis of bilateral medial medullary infarction itself may be better than previously indicated. © 2000 Harcourt Publishers Ltd

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INTRODUCTION

Bilateral medial medullary infarction is a rare condition. Only 12 anatomically confirmed cases^{1,2,4-13} have been reported.

We report a patient with slightly impaired conscious state, dysarthria, disturbance of horizontal ocular movement, left hypoglossal nerve palsy and a left dominant tetraparesis.

CASE REPORT

A 71 year old man with a history of hypertension awoke with dizziness on 2 June 1998. A depressed conscious state and tetraparesis were noted at an out-patient office of a local hospital and he was transferred to our hospital by ambulance. On admission he had a blood pressure of 130/100 mm Hg, a pulse rate of 96 beats per min and a respiratory rate of 32 per min. He was drowsy and his speech was dysarthric. The visual fields were intact and pupils were equal, with a normal light reflex. He could neither direct his gaze voluntarily nor follow a target for leftward gaze. Corneal reflexes were intact, and there was no loss of sensation to pin, temperature or light touch on either side of his face. Facial movements were symmetric and active and hearing appeared intact. No gag reflexes were observed. His left side was flaccid, while the right side exhibited decreased muscle tone. There were no sensory deficits to touch, pain, temperature, vibration or position sense.

On 3 June the disturbance of extraocular movement had deteriorated in both horizontal directions. Brain magnetic resonance imaging (MRI) with a 0.5-T superconducting magnet performed on 4 June revealed a high intensity area on fluid attenuated inversion recovery (FLAIR) imaging, which was noted in the medulla oblongata extending to the lower pons (Fig. 1). The lesion involved the medial medulla oblongata bilaterally, mainly the ventral half with partial extension dorsally.

The disturbance of ocular movement gradually improved, and only mild leftward gaze limitation was detected. Moreover, improvement of tetraparesis was observed, and only a mild left hemiparesis was detected a year after the onset.

DISCUSSION

The medial medullary syndrome is characterised by contralateral hemiparesis and hemisensory disturbance of posterior column function with ipsilateral paralysis of the tongue.³ It results from infarction of the paramedian region of the medulla oblongata due to occlusion of the vertebral or anterior spinal artery or their small branches. Bilateral medial medullary infarction is a rare condition and only 12 anatomically confirmed cases^{1,2,4-13} have been reported. The clinical findings of these 12 cases are summarised in Table 1. Including our case, there were 9 male and 4 female patients, ranging in age from 23 to 71 years, and the mean age was 53.7 years. The first 8 cases were diagnosed by autopsy, but in vivo diagnosis by MRI became possible in the 1990s. In addition, the treatment improved with advances in diagnostic technology and this condition is not necessarily associated with a poor prognosis. Motor pareses were recognised in all cases, and tetrapareses were noted in 10 of 12 cases in which precise neurological findings were available. Caudal/cranial nerve palsy was detected in 6 of 13 cases, including those described as dysarthria or dysphagia, and may result in respiratory infection. Three studies on this lesion described the presence of oculomotor disturbance. Upward extension of the lesion, especially to the pontine medial longitudinal fasciculi (MLF) and caudal paramedian pontine reticular formations (PPRFs), may be responsible for horizontal gaze palsy. However, some previous studies^{12,13} indicated that a vertical gaze

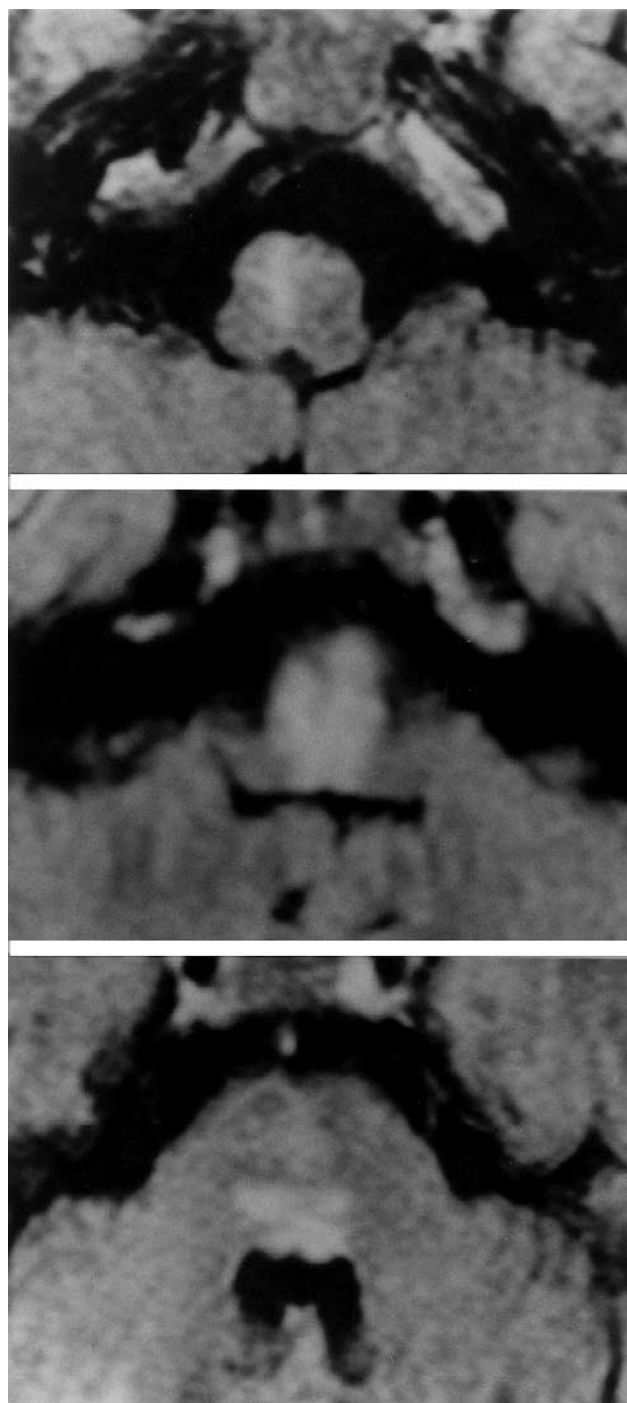


Fig. 1 FLAIR image showing axial sections. Infarction is clearly demonstrated in the medial medulla oblongata bilaterally (arrows).

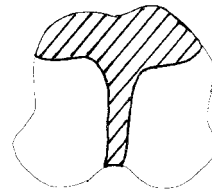
palsy was present. The mechanisms for these symptoms remain unclear.

We classified the 13 cases into 2 groups according to the extent of the ischaemic region (Fig. 2): type 1, the ischaemic lesion developed from the medullary pyramid to MLF; type 2, the lesion was confined to the medullary pyramid. Type 1 infarction may be caused by occlusion of the main artery to the medial medulla oblongata. Type 2 infarction might be caused by occlusion of the arterial branch to the medullary pyramid.

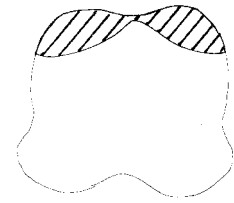
The main cause of death was respiratory infection. Two cases of type 2 did not die of respiratory infection. The prognosis of type 2 might be better than that of type 1.

Table 1 Reported cases of bilateral medial medullary infarction (M: male, F: female, rt.: right, lt.: left)

No.	Report	Year	Age	Sex	Diagnosis	Type	Symptoms	Prognosis	Cause of Death
1	Davison	1937	40	M	autopsy	I	left dominant tetraparesis, lt. Gag reflex (-), sensory disturbance, urinary & faecal incontinence	died, 13 months	attack of coronary occlusion
2	Davison	1944	45	F	autopsy	I	lt. Flaccid hemiplegia, bilateral hyperreflexia, posterior column signs	died, 7 weeks	bronchopneumonia
3	Meyer	1962	45	M	autopsy	I	dysarthria, tetraparesis	died, 7 weeks	aspiration pneumonia
4	Nardelli et al.	1977	57	M	autopsy	I	dysarthria, respiratory failure, flaccid quadriplegia	died, 6 days	bronchopneumonia, renal failure
5	Mizutani et al.	1980	28	F	autopsy	I	caudal nerve palsy, tetraparesis	died, 261 days	pneumonia
6	Kase et al.	1983	23	F	autopsy	I	respiration arrest, quadriplegia, nystagmus	died, 24 hours	pulmonary infection
7	Paulson et al.	1986	60	F	autopsy	II	dysarthria, tetraparesis	died, 7 years	subarachnoid haemorrhage
8	Jagiella et al.	1989	71	M	autopsy	II	pure motor quadriplegia	died, 13 months	urosepsis
9	Toyoda et al.	1992	71	M	MRI	I	quadriplegia, disturbance of ocular movement bed rest	alive, 60 days,	
10	Kleinert et al.	1993	59	M	MRI	I	flaccid tetraplegia, rt. hypoglossal nerve palsy, bilateral disturbance of deep sensation	died, 2 weeks	pneumonia
11	Kim et al.	1995	59	M	MRI	II	dysphagia, facial palsy, nystagmus, severe motor deficit		
12	Wada et al.	1996	69	M	MRI	I	bilateral facial palsy, lt. Hemiparesis, horizontal & upward gaze limitation	alive, 81 days able to walk	
13	Present case	1998	71	M	MRI	I	dysarthria, disturbance of horizontal gaze, lt. Dominant tetraparesis	alive, 5 months able to walk	



Type 1



Type 2

Fig. 2 Type 1: the ischaemic lesion develops from the medullary pyramid to MLF; type 2: the lesion is confined to the medullary pyramid.

The survival rate of medial medullary syndrome may increase with improvements in diagnostic technology. Bilateral medial medullary infarction, which was previously considered to be fatal, can be effectively treated if respiratory management is performed during the acute period.

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Delayed presentation of transorbital intracranial pen

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Summary A 13 year old Fijian boy sustained a stab wound to the left orbit 3 years ago. It was not appreciated by the treating physicians in Fiji that the plastic pen had crossed from the left orbit, through the nose, right orbit and right optic nerve, into the right