Spinal cord ischaemia complicating meningococcal meningitis

SONIA S. SWART
M.B., M.R.C.P. (UK)                      IAN F. PYE
M.B., M.R.C.P. (UK)

Department of Neurology, Leicester Royal Infirmary, Leicester LE1 5WW

Summary
An extensive ischaemic cord syndrome developed in a patient with meningococcal meningitis complicated by 2 respiratory arrests but not by any period of prolonged hypotension or other signs of cardiovascular collapse. Excellent functional recovery occurred after intensive rehabilitation.

Case history
A 15-year-old schoolboy presented with one day’s history of severe headache and progressive confusion following several days of ‘flu-like illness. The clinical findings on admission were a temperature of 40°C, marked neck stiffness, a depressed conscious level with response to pain only, and a generalized erythematous macular rash over the arms and trunk. Lumbar puncture confirmed purulent meningitis due to Neisseria meningitidis.

He was treated with intravenous benzyl penicillin and probenecid. Within 7 hr he was responding to command, drinking unaided, and his temperature had fallen to 38.7°C. At this point he had a respiratory arrest but intubation was followed by the immediate return of spontaneous respiration and recovery of his pre-arrest state. Twelve hours later after a further respiratory arrest he required assisted ventilation and did not regain consciousness for several hours. Neither arrest was accompanied by hypotension. After the second episode, it was noted that he was unable to move. The neurological findings were as follows: he was alert and well orientated and able to mouth words; there was no attempt at spontaneous respiration; neck stiffness remained marked; the visual axes diverged at rest, although external eye movements were full, and there was nystagmus on lateral gaze bilaterally; facial sensation to light touch and pin-prick was impaired bilaterally and the corneal reflexes were sluggish; visual fields and fundi were normal; the gag reflex was absent but function of the 7th and 12th cranial nerves was normal and hearing was intact; he was unable to shrug his shoulders or turn his head, had a flaccid quadriplegia and was areflexic with absent plantar responses; sensation was normal in the upper limbs; there was altered light touch and pin-prick below T4 with absent position sense in the feet but preservation of vibration sensation; he developed urinary retention and required an indwelling catheter.

Tracheostomy was performed and the patient was ventilated. Four days later the conjugate gaze defect and nystagmus had improved and sensation to light touch and pin-prick was returning in the feet. Artificial ventilation was discontinued after 2 weeks. Neurological recovery proceeded in the following sequence: motor power on the left side and sensation on the right returned simultaneously; pin-prick and light touch sensibility recovered before temperature appreciation; the tendon reflexes returned slowly as did some power on the right and sensation on the left. Four weeks after admission he was transferred to a spinal unit for rehabilitation. At that time there was wasting and flaccid weakness of all 4 limbs, more marked distally and worse on the right. The supinator and knee jerks were absent bilaterally but the other tendon reflexes had returned and both plantar responses were extensor. The appreciation of light touch, vibration and joint position sensation had recovered but pin-prick and temperature sensibility remained impaired in the left leg. Eight weeks later he could walk holding parallel bars and 5 months later he was walking unaided. When reviewed 11 months after the illness he was able to jog and had no functional disability in the arms. There were some residual neurological signs consisting of minimal wasting of the intrinsic muscles of the left hand with slight weakness but normal power above the wrist. Formal testing of power in the legs was normal and he was able to walk on tiptoes but there was slight impairment of heel walking and heel toe walking. There was minimal clumsiness of alternate rapid movements in the right arm and leg and minimal right hyperreflexia. Both plantars were extensor and the abdominal reflexes were absent. Sensation
was intact in the limbs apart from some contact
dysaesthesia over the soles of the feet.

**Discussion**

The neurological signs following the respiratory
arrest were compatible with an ischaemic lesion at
the cervico-medullary junction plus patchy involve-
ment of the brainstem rostrally. Within a few days
the brainstem signs had resolved and the clinical
picture suggested an infarct of the upper cervical
cord in the anterior spinal artery territory. This was
considered to be secondary to an infective arteritis,
and although hypoxia may have contributed to
the neurological deficit, it seemed more likely that the
respiratory arrest itself was the result of involvement
of the respiratory centres in the brainstem since on
each occasion he was resuscitated immediately
without any period of hypotension. The presence of
transient signs of brainstem ischaemia may indicate
occlusion of the anterior spinal artery close to its
origins from the vertebral arteries.

Ischaemia of the cord, particularly in the vulner-
able territory of the anterior spinal artery, has been
noted in a variety of clinical situations. Early
reports focused on syphilitic arteritis as a cause of
the syndrome of anterior spinal artery occlusion
(Spiller, 1909). More recent reviews emphasize the
importance of atheromatous vessels (Henson and
Parsons, 1967) with or without a period of prolonged
hypotension as an aetiological factor (Silver and
Buxton, 1974). Hughes (1978) describes spinal cord
infarction as an unusual but important complication
of Pott's disease and thrombosis of the radicular
artery may occur in herpes zoster. Although
ischaemic cord damage secondary to small vessel
endarteritis may occur in pyogenic meningitis,
reports of extensive infarction of the cord have not
been found on reviewing the literature.

This case is reported for 2 reasons, firstly as an
unusual complication of bacterial meningitis, and
secondly, as a reminder that a young previously fit
subject may have a remarkable potential for
neurological recovery from a catastrophic situation.

**Acknowledgment**

We should like to thank Dr C. P. Alexander for referring
the patient.

**References**

Henson, R.A. & Parsons, H. (1967) Ischaemic lesions of the
spinal cord: an illustrated review. *Quarterly Journal of
Medicine, 36*, 205.


Spiller, W.G. (1909) Thrombosis of the cervical anterior
median spinal artery: syphilitic acute anterior polio-
myelitis. *Journal of Nervous and Mental Disorders, 36*, 601.
Spinal cord ischaemia complicating meningococcal meningitis.

S. S. Swart and I. F. Pye

*Postgrad Med J* 1980 56: 661-662
doi: 10.1136/pgmj.56.659.661

Updated information and services can be found at:
http://pmj.bmj.com/content/56/659/661

These include:

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/