Infection of the brainstem by Listeria monocytogenes

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SUMMARY A case of brainstem infection by Listeria monocytogenes is described. The patient was a 63 year old man previously in good health and his illness did not follow the usual bi-phasic pattern. There was no prodromal phase, and the progressive brainstem signs with a lymphocytosis and a normal sugar level in the CSF led to a tentative diagnosis of viral brainstem encephalitis. Ampicillin was begun only when signs of pulmonary infection developed. Clinical diagnosis is difficult but ampicillin should probably be used in any doubtful case in which a “viral” brainstem encephalitis is being considered.

Meningeal infections by Listeria monocytogenes are well recognised in neonates and in immunosuppressed, debilitated, and elderly subjects (Medoff et al., 1971; Gantz et al., 1975). Listeria may also involve the brainstem (Eck, 1957), producing a clinical presentation resembling that of viral brainstem encephalitis.

Case report

A 63 year old man, previously in good health, was admitted as an emergency. Five days earlier he had noticed a pattern of numbness around his left upper lip, and during this period the numbness had gradually spread to involve most of the left side of his face. Two days before admission he noticed horizontal diplopia on looking to his left and found his gait was unsteady. His walking deteriorated rapidly and, shortly before admission, he noticed that his voice had become hoarse and that swallowing was difficult. Six weeks previously his left upper canine tooth had been extracted. Healing of the gum had been uneventful.

On examination he was alert and orientated. There was no fever or neck stiffness. There was a left sixth nerve palsy with coarse grade 1 nystagmus in both vertical and horizontal planes. There was impaired perception of pain and temperature in the maxillary division of the left trigeminal nerve, but light touch sensation was normal in this area. There was an incomplete left facial weakness, left sided palatal weakness, and weakness of the left side of the tongue. In addition, there was cerebellar ataxia of the left arm and leg with marked truncal ataxia and broad-based, unsteady gait. General examination was normal.

The haemoglobin was 14.7 g/dl, the white cell count 7700 mm$^3$ (neutrophils 88%), and ESR 20 mm/hr. A lumbar puncture, performed on the day of admission, revealed clear CSF containing 110 leucocytes/mm$^3$ (90% lymphocytes). The protein content was 0.5 g/l and the sugar was normal.

The CSF pressure was normal. No organisms were found with Gram stain and cultures were negative. A CAT scan was normal.

During the next 48 hours the patient developed increasing difficulty with speech and swallowing. A low grade fever appeared with bilateral basal lung crepitations, and ampicillin therapy was begun. On the third day of admission he suddenly collapsed and died.

The next day Gram positive rods, identified as Listeria monocytogenes, were found in a blood culture taken on admission.

Necropsy findings

The leptomeninges, cerebral hemispheres, and cerebellum appeared normal. The pons and medulla were slightly distended and softened. The brain was sectioned after immersion fixation. The lateral ventricles were small, and the brain was oedematous. There was a confluent multifocal abscess in the left side of the pons and medulla, consisting of several smaller abscesses, each 2 to
6 mm in diameter (Figure, a).

Microscopy of the brainstem showed multiple abscesses containing polymorphs and lymphocytes in the left side of the pons and medulla (Figure, b). A few similar abscesses were present on the right side. There was marked perivascular leucocytic cuffing (Figure, b), and some small vessels contained concentric layers of fibrinoid material. Pus in the abscesses contained both intracellular and extracellular Gram positive bacilli (Figure, c).

The general necropsy revealed pulmonary congestion and a mild basal pneumonia.

Discussion

Since Eck’s (1957) description of brainstem infection by *Listeria monocytogenes* only a further nine cases have been fully described (Benazet et al., 1957; Hirasawa, 1958; Christ et al., 1961; Duffy et al., 1964; Ford et al., 1968; Mahony et al., 1974), although an abstract noting a further six cases has also appeared (Kocen et al., 1977). In all these cases, except two (Ford et al., 1968; Mahony et al., 1974), the patients had been previously healthy. By contrast, focal suppuration elsewhere in the central nervous system due to *Listeria monocytogenes* has usually occurred in debilitated or immunosuppressed patients (Johnson and Colley, 1969; Halkin et al., 1971; Chow et al., 1975; Crocker and Leicester, 1976).

The pathological findings in our patient were similar to those previously reported (Benazet et al., 1957; Eck, 1957). Duffy et al. (1964) noted the similarities in distribution and microscopic appearance of lesions in patients with brainstem involvement from *Listeria monocytogenes* infection to those occurring in animals with Listeria encephalitis, known as “circling disease” (Gill, 1933). This disorder can be produced experimentally in mice and goats either by conjunctival

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Figure (a) Sagittal section through the pons and medulla showing a large abscess in the pontine tegmentum and retro-olivary region of the medulla. (b) Abscess with a large number of inflammatory cells around blood vessels. Haematoxylin and eosin, original magnification ×160. (c) Abscess showing large numbers of intracellular and extracellular Gram positive bacilli. Gram stain, original magnification ×630.
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instillation or by inoculation of the bacteria into small wounds in the mouth (Asahi et al., 1957). Inflammatory changes found in the trigeminal nerve of these animals suggest that the organism invades the brainstem by this route. Unfortunately, the trigeminal nerve and geniculate ganglion were not available for study in our case. However, the first symptom was numbness of the face in the territory of the maxillary division of the left trigeminal nerve, occurring six weeks after removal of the left upper canine tooth, and it is possible, therefore, that infection may have spread from the gingivae to the brainstem via the trigeminal nerve. The incubation period in our patient was similar to that found in experimental studies (Asahi et al., 1957).

In the previously reported cases the CSF has contained more than 100 white cells/mm³, consisting mainly of lymphocytes, but in some cases a predominantly polymorphonuclear response has occurred. The CSF protein is usually only slightly raised, and the sugar has always been normal, as it was in our patient. The organism has never been seen on the initial Gram stain film and has only rarely been cultured from the cerebrospinal fluid. As in our case, positive identification of Listeria infection has usually been made from the blood cultures, often too late to allow effective therapy. The delay in obtaining positive blood cultures is probably a reflection of the very poor growth of Listeria monocytogenes in ordinary broth. This is greatly improved by the addition of 0.5 to 1% of glucose (Wilson and Miles, 1975), and such supplementation to routine blood culture media should be considered in cases of possible encephalitis.

In most reports of brainstem infection by Listeria monocytogenes a bi-phasic illness has been described. A phase of headache, vomiting, fever, and leucocytosis lasting four to 10 days is followed by signs of brainstem involvement (Eck, 1957). In our patient, however, there was no prodromal phase, and the progressive brainstem signs with a lymphocytosis and a normal sugar level in the CSF led to a tentative clinical diagnosis of viral brainstem encephalitis. Ampicillin was begun only when signs of pulmonary infection developed. Clinical diagnosis is difficult but the organism is sensitive to ampicillin and this antibiotic should probably, therefore, be used in any doubtful case in which a diagnosis of "viral" brainstem encephalitis is being considered.

References


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