Letters

severe pulmonary embolism. Post mortem investigation grossly showed brain oedema. Irregular periventricular lesions, adjacent to the 4th ventricle extended to the hilus of the right dentate nucleus (fig 1). These lesions consisted of inflammatory necrosis and/or myelin loss. As a consequence of the lesion of the right dentate nucleus the left inferior olive was hypertrophic (fig 2). Additional findings were a fibrous gliosis in the inferior vermis and a prominent lymphocytic infiltration in the mesencephalon and the rostral pons. The neuropathological data indicated an atypical subacute panencephalitis. Again, special stainings for bacterial or fungal microorganisms and immunocytochemical investigations for viral inclusion bodies failed to clarify the aetiology of the panencephalitis.

Considering the clinical and laboratory findings in adult coeliac disease and associated neurological complications the following speculations about the pathogenesis seem to be reasonable. First, might vitamin E deficiency which was also found in our case cause encephalopathy?⁶ Second, might malnutrition which is often found as a result of coeliac disease cause or promote cerebellar disturbances, myelopathy and neuropathy?^{1-3,7,8} Third, is coeliac disease known to be frequently associated with a genetically determined alteration in immune functions?^{2,8} This might be suggested by a high coincidence of coeliac disease and lymphoma, carcinoma and multifocal leucoencephalopathy.⁹ A high susceptibility of the patients for jejunally mediated infection might be due to a deficiency of mucosal IgA antibodies.

The myoclonic movement disorder of our patient is explained by the neuropathological changes in structures that are known as Guillain-Mollaret's triangle.¹⁰

In conclusion, our case of adult coeliac disease does not provide any further clues to

the pathogenesis of the brainstem encephalitis. Our report merely documents an uncommon relationship between coeliac disease and an atypical subacute encephalitis.

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References

- Cooke WT, Smith T. Neurological disorders associated with adult coeliac disease. *Brain* 1966;**89**:638-722.
- Morris JS, Ajdukiewicz AB, Read AE. Neurological disorders and adult coeliac disease. *Gut* 1970;**11**:549-54.
- Kristoferitsch W, Pointner H. Progressive cerebellar syndrome in adult coeliac disease. *J Neurol* 1987;**234**:116-8.
- Ward ME, Murphy JT, Greenberg GR. Celiac disease and spinocerebellar degeneration with normal vitamin E status. *Neurology* 1985;**35**:1199-201.
- Stokes PL, Asquith P, Holmes GKT, Mackintosh P, Cooke WT. Histocompatibility antigens associated with adult coeliac disease. *Lancet* 1972;**ii**:162-4.
- Muller DPR, Lloyd JK, Wolff OH. Vitamin E and neurological functions. *Lancet* 1983;**i**:225-8.
- Finelli PF, McEntee WJ, Ambler M, Kestenbaum D. Adult coeliac disease presenting as cerebellar syndrome. *Neurology* 1980;**30**:245-9.
- Kinney HC, Burger PC, Hurwith BJ, Hijmans JC, Grant JP. Degeneration of the central nervous system associated with coeliac disease. *Neurol Sci* 1982;**53**:9-22.
- Kepes JJ, Chous S, Prince LW. Progressive multifocal leukoencephalopathy with 10-years survival in a patient with non-tropical sprue. *Neurology* 1975;**25**:1006-12.
- Van Woert MH, Chung Hwang E. Myoclonus. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*. Vol 38. Amsterdam: North-Holland 1979:575-93.
- Jellinger K. Hypertrophy of the inferior olives. *Z Neurol* 1973;**205**:153-74.

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Brain stem glioma mimicking progressive basilar artery thrombosis

Sir: Vascular insufficiency in the posterior cerebral circulation often presents with

visual disturbances, ataxia, dysequilibrium, and alternating motor and sensory signs.¹⁻³ The anatomical origin of these symptoms and signs can be confirmed by neurophysiological testing such as brainstem auditory evoked responses (BAERs), and high resolution cranial computed tomography (CT) or magnetic resonance imaging (MRI) of the brain.⁴ The aetiology of the dysfunction can be confirmed by demonstrating diminution of vertebral-basilar flow via angiography.⁵⁻⁷ Diminution or absence of flow, however, is only presumptive evidence as flow may also be impeded by non-atherosclerotic disorders such as extravascular compression due to mass lesions. We report such a case.

A 70-year-old male initially presented with a 3 month history of diplopia, dysarthria, and gait ataxia. Although of rapid onset, the symptoms were progressive. He appeared slow, but otherwise had a normal general physical examination. His mental status and speech were normal except for a mild scanning dysarthria. Cranial nerve examination showed right medial and bilateral upward gaze palsies. Extraocular movements and gait were ataxic. No other neurological abnormalities were recorded.

Initial CT suggested a sagittal sinus thrombosis and revealed a small basilar artery (fig). Intravenous digital subtraction angiography (DSA) excluded the sinus thrombosis but detail of the posterior fossa circulation was not obtained. Cerebrospinal fluid (CSF) studies were normal. He was discharged with a presumptive diagnosis of brainstem ischaemia after investigation had not shown a systemic malignancy.

Over the next month, his gait ataxia and dysarthria became worse. New problems developed including dysphagia, inability to sit unsupported, and urinary incontinence. On examination, he appeared dehydrated and confused. His speech was slow and ataxic. Gaze palsies persisted as above. Deep tendon reflexes were brisk throughout, with sustained ankle clonus. Bilateral extensor plantar responses were present with upper motor neuron distribution weakness of the right body parts. The patient had marked dysmetria and could not sit unsupported. Repeat CT revealed lucencies in the basal ganglia. BAERs showed prolonged latencies in the left brain stem. Repeat CSF studies were normal. Repeat DSA during aortic arch injection of contrast was consistent with basilar artery thrombosis because of nonfilling. He was unable to cooperate for a cranial MRI study.

The patient developed nausea, vomiting and intractable hiccups, increasing right-sided motor weakness, and somnolence.

Fig 2 Section through inferior olivary nucleus (left). (Bodian's silver method, $\times 775$) showing some prominent dentritic proliferation in neurons.

Fig CT scan without (a) and with (b) iodinated contrast from patient under present discussion. No areas of low attenuation or contrast enhancement were evident on these particular scans. The basilar artery (arrow) was thought to be reduced in calibre.

Approximately 7 months after the onset of his illness, he lapsed into coma and expired shortly thereafter.

Necropsy was limited to brain examination by family request. The brain weighed 1555 grams. On external inspection, the pons was enlarged to 5 cm between the trigeminal nerve exits on either side, was firm to palpation, and grossly discoloured. The left cerebral peduncle was also enlarged. On cut sections, tumour infiltrates were found in the left medullary pyramid, left pons, caudal

midbrain, both cerebral peduncles, and the left thalamus, extending into the left internal capsule and globus pallidus. Microscopic examination showed a hypercellular, poorly differentiated proliferation, consistent with glioblastoma multiform. All cerebral vessels were free of atheroma. No ischaemic damage was found in brainstem structures.

This 70 year old man presented with signs of brainstem dysfunction, including diplopia, ataxia, and dysphagia, and progressed to the point that a presumptive diagnosis of basal artery insufficiency, secondary to progressive basilar artery thrombosis, was entertained. At necropsy, a primary malignant glioma of the brainstem was found. It was surmised that the tumour compressed the basilar artery, resulting in the radiographic abnormalities described. We present this case to demonstrate how, in spite of radiographic and neurophysiologic abnormalities, the correct diagnosis may be elusive in cases of brainstem ischaemia.

Kinkel, *et al*, reported several cases of cerebral ischaemic infarction, with local and distant enhancement on CT.⁶ Their description of the CT findings is similar to those in our case.

Nonvisualisation of the basilar artery on both venous angiography and contrast CT would normally be convincing evidence of basilar artery insufficiency but, in our case, it was of false localising value. Tumour progression has to be considered always in the differential diagnosis of progressive brainstem syndromes.

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References

- 1 Caplan L. Patterns of posterior circulation infarctions: correlations with vascular pathology. In: Berguer R, Bauer RB, ed. *Vertebrobasilar Arterial Occlusive Disease*. New York: Raven Press, 1984:15-26.
- 2 Caplan L. Vertebrobasilar disease: time for a new strategy. *Stroke* 1981;12:111-4.
- 3 Caplan L. "Top of the basilar" syndrome. *Neurology* 1980;30:72-9.
- 4 Simmons Z, Biller J, Adams HP, *et al*. Cerebellar infarction: comparison of computed tomography and magnetic resonance imaging. *Ann neurol* 1986;19:291-3.

- 5 Lynn GE, Gilroy J. Auditory evoked potentials in vertebrobasilar arterial occlusive disease. In: Berguer R, Bauer RB, ed. *Vertebrobasilar Arterial Occlusive Disease*. New York: Raven Press, 1984:85-94.
- 6 Kinkel WR, Newman RP, Jacobs L. Posterior cerebral artery branch occlusions: CT and anatomic considerations. In: Berguer R, Bauer RB, ed. *Vertebrobasilar Arterial Occlusive Disease*. New York: Raven Press, 1984:117-34.
- 7 Faught E, Oh SJ. Brainstem auditory evoked responses in brainstem infarction. *Stroke* 1985;16:701-4.

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Listeria encephalitis with intermittent symptoms and serological diagnosis

Sir: CNS infection with *Listeria monocytogenes* may be meningitic or encephalitic and typically presents with a history of several days headache, malaise, nausea, vomiting and fever, before the appearance of focal signs of CNS infection.¹ These often reflect the brainstem involvement which characterises listeria encephalitis both in man and in ruminant animals among which it causes "circling disease". In general the symptoms and signs persist until antibiotic treatment is given, and the condition is fatal in 30-40% of cases despite appropriate treatment. Two recent reports of listeria brainstem encephalitis in man highlight the importance of early diagnosis and treatment with high doses of intravenous ampicillin.^{2,3}

We report a case of *Listeria monocytogenes* encephalitis with intermittent symptoms and signs, diagnosed eventually on serological grounds. A 24 year old man was admitted to a district general hospital on 2 February 1986, with a 1 week history of mild headache which had become severe on the previous day. In the early hours of the day of admission, he woke complaining of loss of sensation over his tongue, became disorientated, and vomited. He had a fever of 38.6°C, confusion without meningism or focal neurological signs, and a peripheral neutrophil leucocytosis; the CSF contained $67 \times 10^6/l$ lymphocytes, normal glucose and an increased protein of 0.83 g/l. Though the provisional diagnosis was viral encephalitis he received one intravenous dose of ampicillin; he was transferred to Oxford the following day. On arrival the patient felt fully recovered, was afebrile and had a normal CT scan. However, his EEG was asym-