An elderly man with dysphasia and pyrexia

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An 85-year-old man presented with a 3-week history of headache, reduced mobility and dysphasia. The headache was unilateral affecting the left side. His reduced mobility was associated with varying levels of weakness of the right arm and leg. His dysphasia was reflected in problems reading the newspaper, and speaking to his wife, as well as understanding what his wife was saying to him. On examination he was found to be alert and orientated in time, place and person, scoring an abbreviated mental test score of 9/10. Neurological examination revealed a right upper motor lesion of cranial nerve VII, increased reflexes in the right arm and leg, and a dysphasia which was both expressive and receptive. In addition he had a temperature of 37.5°C. He had a normal full blood count, routine biochemistry and chest X-ray. Blood cultures were negative. His C-reactive protein was 11 mg/l. Urgent computed tomography (CT) of the brain was organised (figure 1). Following treatment the patient's condition improved gradually over a month. At discharge he had minimal dysphasia and walked independently with ease. Two months later he developed a peripheral sensory neuropathy of a glove and stocking distribution. Serum vitamin B12, folate and thyroid function tests were normal. There was no history of alcoholism, diabetes mellitus or a connective tissue disorder. Following a further adjustment to his treatment he eventually fully recovered (figure 2).

Figure 1 Cranial CT scan with contrast at presentation

Figure 2 Cranial CT scan with contrast after 8 months

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Questions

- 1 Might the medical history help?
- 2 What is the probable initial diagnosis?
- 3 How would you manage his problem?
- 4 Why did he develop peripheral neuropathy?

Answers

QUESTION 1

Yes. His medical history revealed that about 40 years earlier he had been diagnosed as having a left-sided cerebral abscess secondary to mastoiditis. Both were treated surgically. There are three main categories of predisposing factors for cerebral abscesses. Firstly, a contiguous source of infection such as dental abscess or otitis media accounts for approximately 47% of cases. Secondly, haematogenous spread from a distant focus such as bronchiectasis or bacterial endocarditis can account for 25% of causes. Cranial trauma accounts for approximately 8–13%. In 15–20% of cerebral abscess no cause can be found.¹

QUESTION 2

Cranial CT scan showed ring-enhancing lesions in the left temporo-parietal area suggestive of cerebral abscesses or possibly metastatic malignant deposits (figure 1). The clinical picture supported the former diagnosis.

QUESTION 3

In consultation with local neurosurgeons a decision was made to opt for initial medical treatment and assess response with serial cranial CT scans. Treatment initially involved a 2-week regime of intravenous cefotaxime, metronidazole and ciprofloxacin. This was followed by long-term oral ciprofloxacin and metronidazole. There was resolution of the cerebral abscesses on CT scan 8 months after initiating antibiotics (figure 2). As no organism was isolated from the abscesses, treatment was empirical. The organisms isolated in cerebral abscesses include streptococci (*Streptococcus intermedius* group, including *S. angiosus*) in 60–70% of cases, enterobacteriaceae in 23–30% of cases, bacteroides and parvotella in 20–40% of cases and *Staphylococcus aureus* in 10–15% of cases.² Evidence for the use of met-

Learning points

- beware of patients with stroke and a history of ear disease
- cerebral abscesses can recur late and be managed successfully medically
- long-term metronidazole can cause peripheral neuropathy

ronidazole was provided by Ingham *et al*, who showed it to cross the blood–brain barrier and penetrate cerebral abscesses well.³ In Sjolín's series, a combination of cefotaxime and metronidazole was shown to be effective in treatment of cerebral abscesses.⁴ Success of ciprofloxacin in the treatment of cerebral abscesses has been reported.⁵

QUESTION 4

Metronidazole was stopped as it was suspected to be the cause of the peripheral neuropathy, and the patient was continued on ciprofloxacin 250 mg bid. His peripheral neuropathy had mainly resolved 6 months after discontinuation of metronidazole. This has been a well documented side-effect since 1976 when Coxon and Pallis first suggested metronidazole to be a cause of neuropathy.⁶ The degree of neuropathy is dose- and duration-dependent. Signs of neuropathy are reversed within 4–48 months of cessation of metronidazole treatment.⁷

Final diagnosis

Cerebral abscesses, followed by peripheral neuropathy as a side-effect of metronidazole treatment.

Keywords: adverse drug reaction; metronidazole; peripheral neuropathy

1 Wispelwey B, Dacey Jr. RG, Scheld WM. Brain abscess. In: *Infections of the central nervous system*, 1st edn. New York: Raven Press, 1991; p 463.

2 Wispelwey B, Scheld WM. Brain Abscess. In: *Mandel's principle and practice of infectious diseases*, 4th edn. USA: Churchill-Livingstone, 1995; p 889.

3 Ingham HR, Selkon J B, Roxby CM. Bacteriologic study of otogenic cerebral abscesses: chemotherapeutic role of metronidazole. *BMJ* 1977;2:991–3.

4 Sjolín J, Lilja A, Eriksoon N, *et al*. Treatment of brain abscess with cefotaxime and metronidazole: prospective study on 15 consecutive patients. *Clin Infect Dis* 1993;17: 857–63.

5 Mongiardo N, Mussini C, Trenti F, *et al*. Non typhoid salmonella subdural empyema in a patient with acquired immune deficiency syndrome. *Scand J Infect Dis* 1995;2: 173–4.

6 Coxon A, Pallis CA. Metronidazole neuropathy. *J Neurol Neurosurg Psychiatry* 1976;39:403–5.

7 Learned-Coughlin S. Peripheral neuropathy induced by metronidazole. *Ann Pharmacother* 1994;28:536.

Sudden hearing loss following acute hepatitis

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A 35-year-old previously healthy physician was admitted to the emergency room with a fever up to 40°C, extreme weakness and dark urine. The physical examination was remarkable for right upper quadrant abdominal tenderness and mild splenomegaly. He denied sore throat or direct contact with blood products. Laboratory studies disclosed elevated liver enzymes, hyperbilirubinemia and bilirubinuria. A diagnosis of acute hepatitis was established. Initially, all serologic viral studies were negative. However, a late seroconversion for IgM to the early antigen (EA) of Epstein-Barr virus (EBV) in the presence of negative antibodies to Epstein-Barr nuclear antigen (EBNA), followed by a rise in the IgG titres to EA and EBNA were observed, indicating EBV to be the causative agent of the hepatitis. During follow-up examinations in our out-patient clinic, a gradual convalescence and normalisation of the abnormal liver function tests were observed.

Four weeks later the patient complained of a sudden hearing loss in his right ear which he had noticed subsequent to an examination of his patients with a stethoscope and after using a telephone handset with his right ear. He did not complain of tinnitus, nausea, vomiting or vertigo. The physical examination was normal, with a negative Romberg test and without nystagmus. A comprehensive audiologic assessment revealed a bilateral normal ear-drum with a normal pure-tone air and bone conduction (Weber and Rinne tests). The pure tone threshold analysis results are presented in figure 1. Treatment was initiated and the audiogram was repeated 10 days later (figure 2).

Figure 1 Audiogram performed on admission

Figure 2 Audiogram performed 10 days after initiation of therapy

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Questions

- 1 What abnormality is demonstrated on the admission audiogram (figure 1) ?
- 2 What is the diagnosis and its cause ?
- 3 What are the possible additional common aetiologies of the presented disorder ?
- 4 Describe the changes in the audiogram presented in figure 2 compared to that in figure 1.