CASE REPORT

Facial palsy associated with leptospirosis

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Summary
Introduction: The authors report a rare case of facial palsy associated with leptospirosis.
Case report: A 60-year-old man was admitted to ICU with severe leptospirosis. On the eighth day of hospitalisation, he developed left peripheral facial palsy with a favourable course in response to corticosteroids.
Discussion: Several types of neurological complications of leptospirosis have been reported: encephalitis, myelitis, stroke, cerebral arteritis, mononeuritis, polyradiculoneuropathy, and cranial nerve palsy. Peripheral facial palsy is a rare complication of leptospirosis.
Conclusion: This case illustrates the possible association between leptospirosis and facial palsy.

Introduction

Leptospirosis, considered for a long time to be an occupational disease (sewer workers’ disease), is now an increasingly common infectious disease, particularly in tropical climates and in underprivileged zones of large cities[1]. A wide range of clinical forms of this disease have been described. Neurological features are present in 12 to 40% of cases, usually in the form of febrile meningal syndrome[2]. Facial palsy is only rarely associated with leptospirosis. We report a new case of this association and discuss the pathophysiological mechanisms of this rare clinical entity.

Case report

Mr A.B., 60-year-old, with no significant medical history, reporting a history of contact with rats, developed mucocutaneous jaundice, myalgia and three episodes of minor bleeding gums 5 days before admission to hospital. These symptoms were associated with fever of 39°C. Physical examination on admission revealed a conscious patient with conjunctival hyperaemia, intense mucocutaneous jaundice, fever of 38.7°C, blood pressure of 110/60 mmHg, tachycardia at 100 bpm, tachypnoea at 26 per minute and a soft abdomen on palpation. The initial laboratory work-up revealed leukocytosis with 26,000 leukocytes/mL, thrombocytopenia with 46,000 platelets/mL, haemoglobin: 9 g/dL, CRP: 236 mg/L, blood glucose: 1.02 g/L, serum sodium: 140 mEq/L, serum potassium: 3.7 mEq/L, blood urea nitrogen: 2.57 g/L, serum creatinine: 43 mg/L, total bilirubin: 399 mg/L, direct bilirubin: 205 mg/L, and ALAT and ASAT at 93 and 59 IU/L, respectively. Lumbar puncture was not performed on admission. All findings were suggestive of a diagnosis of leptospirosis. Leptospirosis serology was positive. Abdominal ultrasound, and electrocardiogram were normal. Chest x-ray showed a diffuse alveolar syndrome. The patient was treated with penicillin G at a dosage of 12 million units per day with rehydration by 4 L/day.

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and oxygen therapy. On the eighth day of hospitalisation, the patient presented rapidly progressive House-Brackmann grade IV left peripheral facial palsy (Fig. 1). Physical examination revealed asymmetry at rest with a positive Bell’s sign. ENT examination showed a normal tympanicum on both sides. Head and neck examination did not demonstrate any parotid lesion. Neurological examination was strictly normal and, in particular, did not reveal any other nerve lesions. Head computed tomography was normal. The patient was then treated with corticosteroids at a dosage of 1 mg/kg per day of prednisolone for 2 weeks with progressive tapering, associated with a potassium and calcium supplement with gastric protection. An eye dressing was also applied. The clinical course was favourable with return of apyrexia, marked improvement of jaundice, return of normal renal function (blood urea nitrogen: 0.36 g/L, serum creatinine: 7 mg/L), and resolution of the other laboratory disorders described above. Facial palsy gradually resolved after the third week of treatment.

Discussion

Leptospirosis is one of the most common zoonoses with a worldwide distribution and an annual incidence of about 100,000 cases, responsible for about 1000 deaths each year in the world. The pathogens responsible for leptospirosis are *Leptospira* bacteria and the main reservoirs are rodents but other animal species can sometimes be involved. Man is contaminated either by direct contact with a rodent or its excrements, or, more often, indirectly by contact with contaminated water. The main portal of entry is the skin, via cutaneous excoriations, or more rarely via mucous membranes and exceptionally via the respiratory or gastrointestinal tracts [2]. Leptospirosis is characterized by the development of vasculitis accounting for the wide range of nonspecific and polymorphous clinical manifestations of this disease [2]. However, most patients remain asymptomatic [3]. Clinical signs such as fever and organ involvement, most frequently liver involvement causing jaundice, renal involvement (50–80% of cases), lung involvement (20 to 70% of cases) and meningeal involvement (15–20% of cases), appear after an incubation period lasting 7 to 10 days [2]. Many neurological complications have been described in leptospirosis: encephalitis, myelitis, stroke, cerebral arterial disease, polyneuritis, polyneuropathy, and cranial nerve palsy [4]. Leptospirosis is only very rarely associated with peripheral facial palsy. A review of the literature in 2010 only found six cases of facial palsy associated with leptospirosis, including two bilateral forms [5,6]. This patient developed peripheral facial palsy after the acute phase of the disease. Several hypotheses have been proposed concerning the pathogenesis of these neuropathies. The most widely accepted hypothesis is that, after invading the blood circulation, *Leptospira* spirochaetes induce systemic vasculitis and activate circulating immune complexes [5]. Examination of the neuromuscular biopsy performed by Azouvi et al. found signs of Wallerian degeneration and perivascular and perineural inflammatory infiltrates [7].

In the case reported here, the clinical and laboratory findings were typical of leptospirosis and no other cause of peripheral neuropathy was identified (diabetes, collagen diseases, etc.). The favourable outcome in response to corticosteroids and the chronological relationship with the immune phase of the disease also suggest that the facial palsy was most probably due to vasculitis induced by leptospirosis.

Conclusion

Although a purely coincidental association cannot be formally excluded, this case highlights the possible association between leptospirosis and facial palsy.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References


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