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Original research Articles

A Rare Manifestation of Post-Treatment Neuropathy: Facial Diplegia Following Leptospirosis

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Abstract

Introduction:
Leptospirosis, a zoonotic bacterial infection, can occasionally lead to rare post-infectious complications.[1] While the acute phase often involves systemic manifestations, delayed neurological sequelae such as facial diplegia are uncommon and underreported. This case highlights bilateral lower motor neuron facial palsy as a potential post-infectious complication [2]

Case presentation: A 58-year-old man was treated for leptospirosis after presenting with fever, headache, and muscle aches following exposure to floodwaters. Diagnosis was confirmed by positive *Leptospira* serology, elevated liver enzymes (ALT 140 U/L, AST 122 U/L), and mild renal impairment (serum creatinine 1.6 mg/dL). He was successfully treated with intravenous penicillin and oral doxycycline, with full resolution of acute symptoms. Two weeks post-treatment, the patient developed bilateral facial weakness, with difficulty raising his eyebrows, asymmetry in smiling, and incomplete eyelid closure. Neurological examination confirmed bilateral lower motor neuron facial palsy, without limb weakness or other neurological deficits. MRI of the brain and EMG confirmed the diagnosis, with no evidence of stroke, mass lesions, or ongoing infection.

Conclusion: This case underscores the importance of recognizing delayed neurological complications following leptospirosis. The likely etiology in this patient was immune-mediated inflammation triggered by the infection. Treatment with corticosteroids and physical therapy led to significant improvement. Clinicians should maintain vigilance for post-infectious sequelae in leptospirosis patients, as timely intervention can improve outcomes.

Keywords: Leptospirosis, Facial diplegia, Lower motor neuron facial palsy, Immune-mediated inflammation, Electromyography (EMG), Neurology

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Introduction:

Leptospirosis is a widespread zoonotic bacterial infection caused by *Leptospira* spp., typically acquired through exposure to contaminated water or soil. While it primarily manifests as a febrile illness, severe cases may involve multi-organ complications, including liver dysfunction, renal impairment, and pulmonary hemorrhage. [1] Post-infectious neurological complications are rare but can occur as immune-mediated sequelae following the acute phase. Facial diplegia, characterized by bilateral lower motor neuron facial nerve palsy, is an uncommon manifestation in this context. [2] This report describes a 58-year-old man who developed facial diplegia two weeks after completing standard treatment for leptospirosis, highlighting the challenges in diagnosis and management of such post-infectious complications.[3]

Case Report :

A 58-year-old man presented with fever, myalgia, and severe headache following exposure to floodwaters. Laboratory tests confirmed leptospirosis, with positive serology for *Leptospira* spp., elevated liver enzymes (ALT 140 U/L, AST 122 U/L), mild renal dysfunction (serum creatinine 1.6 mg/dL), and thrombocytopenia (platelets 110 × 10^9/L). He was treated with intravenous penicillin for seven days, followed by oral doxycycline, achieving resolution of his acute symptoms.

Two weeks after completing treatment, the patient developed bilateral facial weakness. Clinical evaluation revealed difficulty raising eyebrows, asymmetrical smiling, incomplete eyelid closure, and mild dysarthria. Neurological examination confirmed bilateral lower motor neuron facial nerve palsy, with no other cranial nerve involvement or neurological deficits. [4,5] MRI of the brain was unremarkable, ruling out structural lesions or stroke. Electromyography (EMG) confirmed bilateral lower motor neuron facial nerve palsy (table 1). Inflammatory markers, including C-reactive protein (4.2 mg/L), were normal, and repeat serology for leptospirosis was negative, excluding ongoing infection. Lumbar Puncture was not performed due to absence of meningitis signs.

Table 1: Laboratory values

Test	Values	Reference Range
Hemoglobin	14.2 g/dL	13.5–17.5 g/dL (male)
White Blood Cell Count	8.2 × 10^9/L	4.0–11.0 × 10^9/L
Platelets	110 × 10^9/L	150–450 × 10^9/L
ALT	140 U/L	7–56 U/L
AST	122 U/L	10–40 U/L
Bilirubin (Total)	Normal	0.1–1.2 mg/dL
Serum Creatinine	1.6 mg/dL	0.7–1.3 mg/dL (male)
Sodium	140 mmol/L	135–145 mmol/L
Potassium	4.2 mmol/L	3.5–5.1 mmol/L
C-reactive Protein (CRP)	4.2 mg/L	<5 mg/L
Blood Glucose	98 mg/dL	70–100 mg/dL (fasting)
Serology for Leptospirosis		
IgG (after 2 weeks)	Positive	Negative
IgM (after 2 weeks)	Negative	Negative

Table 2: EMG (Electromyography) findings

Parameter	Left Facial Nerve	Right Facial Nerve
CMAP Amplitude (Orbicularis Oculi)	1.2 mV (reduced; normal >3 mV)	1.1 mV (reduced; normal >3 mV)
Distal Latency (Orbicularis Oculi)	5.8 ms (prolonged; normal <5 ms)	5.9 ms (prolonged; normal <5 ms)
F-wave Latency	Absent	Absent
Spontaneous Activity	Fibrillation potentials observed	Fibrillation potentials observed
Recruitment Pattern	Reduced	Reduced
Blink Reflex R1 Latency	15 ms (prolonged; normal <12 ms)	16 ms (prolonged; normal <12 ms)
Blink Reflex R2 Latency	39 ms (prolonged; normal <35 ms)	41 ms (prolonged; normal <35 ms)
Cranial Nerve Involvement	None detected	None detected

Given the absence of systemic infection or generalized neurological symptoms, a post-infectious immune-mediated mechanism was suspected. Atypical Guillain-Barré syndrome (GBS) with isolated facial diplegia was considered but deemed less likely due to preserved limb strength and reflexes. [6,7] The patient was treated with oral corticosteroids (prednisone) and physical therapy for facial muscle rehabilitation. Over six weeks, his symptoms improved significantly, with near-complete resolution of facial asymmetry and restoration of speech clarity.

Discussion :

Neurological complications of leptospirosis are rare and may result from direct bacterial invasion, systemic inflammation, or post-infectious immune responses [8]. Facial diplegia as a delayed post-infectious manifestation is exceedingly uncommon [5]. Its pathophysiology likely involves immune-mediated inflammation targeting the facial nerves, potentially triggered by molecular mimicry following *Leptospira* infection.[1]
Differentiating between post-infectious facial diplegia and other causes, such as GBS, Bell's palsy, or CNS structural lesions, is crucial. In this case, the absence of limb weakness, normal reflexes, and unremarkable brain imaging ruled out classical GBS and central causes [2,6]. The timing of symptom onset, two weeks post-infection resolution, strongly suggested an immune-mediated etiology.
Management of post-infectious neurological complications is guided by the suspected underlying mechanism. Corticosteroids are commonly used to mitigate inflammatory damage.[9] This patient's improvement with steroids and physical therapy underscores the potential for recovery in immune-mediated facial diplegia. However, residual weakness may persist in some cases, necessitating prolonged rehabilitation.

Conclusion:

This case highlights facial diplegia as a rare but significant post-infectious complication of leptospirosis. Timely recognition and appropriate management with corticosteroids and rehabilitation can lead to favorable outcomes. Clinicians should remain vigilant for delayed neurological sequelae in leptospirosis patients, even after apparent resolution of the acute phase. Further studies are needed to elucidate the mechanisms underlying such complications and optimize treatment strategies.

Conflict of interest: nothing to declare

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