CASE REPORT

UNUSUAL MANIFESTATION IN PARALYTIC RABIES

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Paralytic rabies is far less familiar than the classical type. The presenting feature of paralytic rabies is ascending paralysis, resembling acute polyneuritis, or ascending myelitis. Cerebrospinal fluid (CSF) examination usually presents as aseptic meningitis or normal finding (Hurst and Pawan, 1931; Chopra et al, 1980; Phuapradit et al, 1985). To our knowledge, low CSF sugar profile in initial presentation of paralytic rabies has never been reported. We here report a case of paralytic rabies which presented as subacute meningomyelitis with lymphocytes and low sugar level in CSF.

A 41-year-old Thai man was admitted to Srinagarind Hospital on 26 August 1990 with the chief complaint of urinary retention for 2 days prior to admission. He was healthy until about 9 days before admission when he experienced high fever, chills, myalgia, generalized headache and mild conjunctivitis. He was treated at a local hospital without improvement. Three days prior to admission he complained of lower abdominal pain and mild weakness of both legs and on the next day retention of urine occurred. He denied a history of animal bite or anti-rabies vaccination.

Physical examination revealed an alert man with body temperature of 39.5°C. The conjuntivae of both eyes were injected, and he had generalized muscle tenderness. He had a stiff neck, generalized muscle weakness (muscle power grade IV in upper limbs and grade III in lower limbs) with hyporeflexia of both knee and ankle jerks, and loose anal sphincter tone. Pin prick sensation and joint positional sensation were intact.

Laboratory results of complete blood count, blood glucose, urea, creatinine and electrolytes were within normal limits. CSF showed high initial opening pressure, mild lymphocytic pleocytosis, mild elevation of protein and low sugar ratio profile (Table 1). No CSF blockage was demonstrated by the Queckenstedt maneuver.

Intravenous doxycycline 200 mg per day was given for 3 days without improvement. On 27 August, the respiration rate was 36-40 per minute and mechanical respiration was performed. On 28 August he developed drowsiness. Myoedema at deltoid and thigh regions and hypersalivation were noted. Serum and CSF for anti-rabies virus titer were requested. Treatment was switched to combined antituberculous drugs and intravenous dexamethasone 20 mg per day for treatment of tuberculous meningitis because of persistent low sugar ratio profile in CSF (Table 1). On the next two days his condition improved, including consciousness and muscle power. Sensation to pin prick was lost in the legs and trunk up to the level of T₉ level. However on 2 September his consciousness was diminshed again with a rise of body temperature of 40.5°C. On 4 September he developed coma and hypotension. Myoedema was remained detected. Blood pressure was maintained by inotropic drugs. The result of anti-rabies virus titers in the serum and CSF, performed by indirect immunofluorescent technique, were 1:8192. Also the corneal touch preparation, performed by indirect immunofluorescence technique, demonstrated rabies virus antigen. His condition did not improve and he died on 5 September from cardiac arrest.

Brain tissue from autopsy revealed Negri body both by direct immunofluorescence and routine paraffin sections.

The clinical features of rabies may be classified as encephalitic rabies and paralytic rabies. The encephalitic form is well known to most clinicians.

Table 1						
Cerebrospinal	fluid	analysis.				

	Pressure (mmH ₂ O)	WBC (per cu mm)	Protein (mg/dl)	Glucose (CSF/blood)
August 26	280	164 (84% lymphocytes)	> 200	47/129 (36.43%)
August 27	-	300 (80% lymphocytes)	131	49/115 (42.60%)
August 29	110	326 (82% lymphocytes)	104	67/141 (47.51%)
September 3	90	90 (85% lymphocytes)	58	66/133 (49.62%)

It is easily diagnosed by its typical manifestation as aerophobia and hydrophobia, alternating intervals of full comprehension and confusion, and signs of autonomic dysfunction such as hypersalivation. However paralytic rabies is far less familiar. The presenting feature is ascending paralysis resembling the Guillain Barré Syndrome (GBS) or ascending myelitis. Consciousness is fully alert in the early stage. Aerophobia and hydrophobia may present in only half of the cases (Hemachudha et al, 1989) and usually occur during the terminal stage (Hurst et al, 1931; Hemachudha et al, 1989). The helpful sign of paralytic rabies during the early phase is percussion myoedema (Hemachudha et al, 1987). It is best demonstrated by percussion at the deltoid and thigh regions with a tendon hammer or a finger and consists of mounding of a part of muscle at the percussion site which then flattens and disappears over a few seconds. It is noted from the early phase to the preterminal stage of paralytic rabies and this sign is not found in GBS patients. The other useful sign in paralytic rabies is acute fasciculation (Phuapradit et al, 1985) which does not appear in acute transverse myelitis.

Cerebrospinal fluid analysis in rabies usually presents with normal finding or mild lymphocytic pleocytosis, mild elevation of protein and normal sugar profile. To our knowledge there is only one report of human rabies which presented with a low sugar profile in CSF (Roine *et al*, 1988). However this abnormality presents in the late stage of the disease.

Our patient had symptoms compatible with paralytic rabies. However the initial presentation

with CSF lymphocytosis, low CSF sugar profile suggested to another diagnosis such as tuberculous meningitis which could have the clinical manifestation of subacute meningomyelitis and similar abnormal CSF findings. Thus in the patient who presents with GBS or transverse myelitis and CSF lymphocytosis and low sugar profile in CSF, paralytic rabies is one of the differential diagnoses. Percussion myoedema and fasciculation should be look for to confirm the diagnosis.

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