



Case Report

West Nile Virus Myelitis

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Study design: A case report.

Setting: Department of Rehabilitation Medicine, Reuth Medical Center, Tel Aviv, Israel.

Method: Summary of the clinical course during in-patient and out-patient treatment of a patient with West Nile Virus Myelitis.

Results and conclusion: A healthy young woman, whose medical history revealed only a benign Duane syndrome and a few months' duration of bipolar disorder, contracted encephalo-myelitis due to a West Nile Virus infection. Although she recovered remarkably after long-term rehabilitation treatments, some weakness and pain remained.

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Introduction

West Nile Virus (WNV) was first identified in Uganda in 1937. Until the year 2000, when the virus was isolated in the New York City area, it was common in Africa, the Middle East, West Asia and Southern Europe. *The New York Times* reported that the American strain is 'almost indistinguishable from a strain found in a goose on an Israeli farm'.

Culex Pipiens, or common household mosquito, spreads the virus when it feeds on blood from infected birds. Ten to 14 days after the initial blood meal, the WNV reaches the mosquito's salivary glands and can then be transmitted to birds, animals or humans. There is no evidence that the WNV can be spread from human to human. The human immune system prevents the virus from multiplying in large numbers. The virus causes encephalitis or encephalo-myelitis. Closely related viruses are the St. Louis and the Japanese encephalitis viruses.^{1,2}

There were previous WNV epidemics: in Israel during the 1950s; in South Africa in 1974 and in 1996–97; in the New York area Romania and Russia in 1999–2000.^{3,4}

Symptoms are usually mild: fever, headaches, body aches, skin rashes, swollen lymph nodes. In severe cases, high fever is accompanied by neck stiffness, and rarely, confusion, disorientation, stupor, coma, convulsions, paralysis and occasionally death.

The 1950s epidemic in Israel involved over 500 clinical cases, while the 2000 outbreak caused the death of 12 people.⁵

The WNV can infect anyone, but the elderly, the very young and those with an immune compromised system are at the greatest risk of developing serious disease. We report on a healthy young woman who contracted the severe form of the WNV and on her rehabilitation outcome.

Case report

A 33-year-old woman was admitted on August 26, 2000 to a general hospital's department of neurology with pyrexia of 38°C, cervical pain and left hand weakness. The febrile illness started about 10 days prior to her admission. Meningitis was suspected.

Past history revealed that she had a congenital bilateral Duane syndrome (an eye movement disorder: limited ability to move the eye inwards toward the nose, or outwards or in both directions). A few months earlier she had been diagnosed as suffering from a bipolar affective disorder.

Upon admission, she was found to have the following parameters: 38°C, HR 98 min, BP of 120/80. The following day she became somnolent. Eye fundi, pupil responses and sensory examination were normal, but she slowly developed an incomplete flaccid areflexic tetraparesis (ASIA B). Routine laboratory studies showed no pathology, including anti-cardiolipin Ab, ANF and HIV Ab. No other viruses were identified including the Polio virus.

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Cervical M.R.I. with gadolinium and brain C.T. were normal. EEG showed some general slowness. A pulmonary nuclear perfusion test revealed non-homogenous perfusion [pulmonary emboli?] and anti-coagulant therapy was started [5000 × 2 u/day s.c. of Fragmin (Dalteparin = 1 m.w.h.)]. Repeated lumbar puncture (L.P.) 4 days later showed pleocytosis, higher protein and IgM values (Table 1). One gram of i.v. Solumedrol was administered, then Dexacort 12 mg/day p.o. and Carbamazepine cr 400 mg/day.

On September 6 she was admitted to our rehabilitation ward. She was in good general health, no hyperpyrexia, fully conscious, no sphincter problems and a flaccid tetraparesis (ASIA C). Psychological examination revealed an intelligent and alert woman, with some evidence of manic-depressive behavior and psychotic symptoms.

Functional Independence Measure (FIM) at admission was 81 and a month later 91. Gradual neurological improvement was noticed, more so in the lower limbs. Occupational Therapist (OT) and Physiotherapist (PT) assessments are summarized in Table 2. On discharge she could walk unaided and was almost independent, namely, on ASIA D.

Table 1 Cerebrospinal fluid data

	1st day	2nd day	3rd day
Opening pressure	70 mmH2O	70	85
Analysis	No cells	200 mononuclears	
Glucose	75 mg%	65 mg%	64 mg%
Protein, lactate and cultures			24 mg% lactate 29.5 i.u. WNV+

Table 2 Functional status at admission and discharge

	Admission		Discharge	
	Rt	Lt	Rt	Lt
Muscle power				
Shoulder	1+	0	3-	2
Elbow Flex	3-	0	3+	2
Wrist Extension	2+	1+	4	3-
Sensation	Normal		Normal	
Pain	Some		Severe pain in neck and shoulder	
Ambulation	Wheelchair bound		Walking, no aids	
Activities of daily living				
Transfers	Dependent		Independent	
Personal hygiene	Dependent		Independent	
Feeding	Some help		Independent	
Dressing: upper limb	Dependent		Some help	
Dressing: lower limb	Dependent		Independent	
Bath	Dependent		Supervision	
Instrumental activities of daily living				
Preparing food	Dependent		Independent	
Cleaning the house	Dependent		Some help	
Working on computer	Slow and inefficient		Independent	

She continued her rehabilitation on a day-care basis three times a week. For the last month (December 2000), we did not notice any further neurological changes.

Discussion

We have described a rare case of a healthy young woman who contracted WNV during the 2000 epidemic, which caused her encephalo-myelitis. During the relatively long regular multi-disciplinary rehabilitation period, encephalitis and tetraparesis apparently subsided into incomplete flaccid upper limb paresis. She was discharged home. No substantial changes in her home were recommended. She was advised to return to her previous job as secretary.

As stated before, severe neurological complications due to WNV infection usually occur in the elderly afflicted with disabling diseases or the immunologically compromised.^{6,7} Our patient was rather healthy: neither the benign congenital Duane retraction syndrome (a congenital failure of the abducens nerve to develop normally^{8,9}) nor the very short-term diagnosis of bipolar disorder should be regarded as serious chronic problems. The incomplete recovery in this case is very similar clinically to the outcome of patients with another viral myelitis, ie poliomyelitis.

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