

## West Nile Virus Infection - Encephalitis - Lower Respiratory Tract Infection - Respiratory failure type II-CASE REPORT

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### Introduction

West Nile virus (WNV) is the leading cause of mosquito-borne disease in the continental United States. It is most commonly spread to people by the bite of an infected mosquito. Cases of WNV occur during mosquito season, which starts in the summer and continues through fall. There are no vaccines to prevent or medications to treat WNV in people. Fortunately, most people infected with WNV do not feel sick. About 20% of people who are infected develop a fever and other symptoms. About 1% infected people develop a serious, sometimes fatal, illness.

### Patient's Medical History

- Type II DM (treated with metformin)
- Class III obesity
- Hyperuricemia (treated with allopurinol)

### Present Illness

This is a female patient, aged 58 years old, who was admitted to the Emergency Department on 19/08/2018 due to a reported fever up to 38.5°C and dysarthria. The patient had already been receiving cefuroxime pos for 3 days. In the Emergency Department, Type II Respiratory Failure was also observed. The patient was admitted to the pathology clinic with a diagnosis of possible West Nile Virus Encephalitis due to clinical and epidemiological evidence. During her hospitalization, as it was impossible to perform a lumbar puncture due to her body type, Abs against West Nile Virus were sent to the serum.

However, the patient presented a gradual deterioration of her clinical picture with persistence of febrile illness, a picture of profundity and worsening of respiratory failure and was led to tracheal intubation and Mechanical Ventilation. On imaging examination, mild cerebral oedema was noted (verbal response) by CT brain and by CT chest atelectasis at the bilateral lung bases and a picture of thickening mainly on the right (CD attached). Also reported by a heart triplex: good contractility and good LV dimensions with EF: 60%, increased LA dimensions, mild mitral and tricuspid regurgitation and RVSP: 40-45mmHg. Also attached is an ultrasound of kidneys, ureters and bladder which shows no abnormal findings. The

patient was receiving moxifloxacin for 3 days, meropenem and acyclovir for 2 days and had received piperacillin/tazobactam and ceftriaxone for 1 day.

On 23/08/2018 she was transferred to the ICU for vital functions support and further hospitalization and treatment.

### Course of Illness

On admission to the ICU the patient was intubated under Mechanical Ventilation and was receiving propofol sedation (RASS-4). Pupils in mid-position, miosis with a positive optokinetic reflex (APACHE II: 16, SOFA:9). She was on mechanical ventilatory support with severely impaired gas exchange (PO<sub>2</sub>/FiO<sub>2</sub>=87). ABGs: pH=7.44, pCO<sub>2</sub>=45mmHg, PO<sub>2</sub>=87mmHg, HCO<sub>3</sub>=30mEq/l, Lac=1.3mmol/dl with FiO<sub>2</sub>=100% and PEEP=14. Cardio vascularly, the patient was hemodynamically unstable with need for low dose vasoconstrictor administration (noradrenaline 6g/min) and maintained satisfactory hourly diuresis.

Due to the dense features on chest CT and the presence of leukocytosis, empirical antimicrobial treatment with meropenem was continued and linezolid was empirically added. Treatment with acyclovir was not continued as antiviral agent treatment is not recommended in case of West Nile Virus Infection, but a conservative treatment. As the patient remained febrile with a temperature of 38.5°C, had increasing infection markers and had purulent bronchial secretions, colimycin iv was empirically added to her treatment on the 5<sup>th</sup> day of hospitalization, adjusted to her renal function (last urinary creatinine clearance: 70ml/min on 28/08/18). Urine antigen testing for legionella and pneumococcus was negative and no pathogen was isolated from bronchial secretions and urine.

On day 6 (28/8/18) we were informed by the laboratory of the Athens Medical School that positive IgM and IgG antibodies against West Nile Virus were detected, which is consistent with acute infection. During her hospitalization, Hepatitis B virus (HBV) infection was also detected as she was positive for HbsAg, anti-Hbc, anti-Hbe.

The patient showed gradual improvement in her respiratory function with gradual restoration of gas exchange and improvement in her hemodynamic status, maintaining satisfactory renal function. In the last few days, she has presented low-grade fever reaching 38.0°C, she has had minimal mucopurulent bronchial secretions, restoration of atelectasis of the right lower lobe clinically and imaging. She underwent weaning and on T-piece test with FiO<sub>2</sub> 50% on 28/08/18, she maintained satisfactory gas exchange.

At this stage, the patient is without sedation (she had been receiving propofol until 25/8/18), awake, with a good level of communication, calm with GCS: 11T/15. She remains intubated with Mechanical Ventilation with PS: 14cmH<sub>2</sub>O and PEEP: 6cmH<sub>2</sub>O and has adequate ventilation and oxygenation. The patient maintains a satisfactory cough. Hemodynamically she is stable (on noradrenaline 1-2 g/min). She is feeding from the nasogastric tube, and she is voiding her bowels (last voiding 29/08/18). Satisfactory serum glucose control with crystalline insulin on a drip.

She needs respiratory physiotherapy, movement therapy.

### Conclusions

Infection with WNV is either asymptomatic (no symptoms) in around 80% of infected people, or can lead to West Nile fever or severe West Nile disease.

About 20% of people who become infected with WNV will develop West Nile fever. Symptoms include fever, headache, tiredness, and body aches, nausea, vomiting, occasionally with a skin rash (on the trunk of the body) and swollen lymph glands.

The symptoms of severe disease (also called neuroinvasive disease, such as West Nile encephalitis or meningitis or West Nile poliomyelitis) include headache, high fever, neck stiffness, stupor, disorientation, coma, tremors, convulsions, muscle weakness, and paralysis. It is estimated that approximately 1 in 150 persons infected with the West Nile virus will develop a more severe form of disease. Serious illness can occur in people of any age, however people over the age of 50 and some immunocompromised persons (for example, transplant patients) are at the highest risk for getting severely ill when infected with WNV.

The incubation period is usually 3 to 14 days.

West Nile virus can be diagnosed by a number of different tests:

- IgG antibody sero-conversion (or significant increase in antibody titers) in two serial specimen collected at a one week interval by enzyme-linked immunosorbent assay (ELISA);
- IgM antibody capture enzyme-linked immunosorbent assay (ELISA);
- neutralisation assays;
- viral detection by reverse transcription polymerase chain reaction (RT-PCR) assay, and
- virus isolation by cell culture.

IgM can be detected in nearly all cerebrospinal fluid (CSF) and serum specimens received from WNV infected patients at the time of their clinical presentation. Serum IgM antibody may persist for more than a year.

Treatment is supportive for patients with neuro-invasive West Nile virus, often involving hospitalization, intravenous fluids, respiratory support, and prevention of secondary infections. No vaccine is available for humans.

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