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Imported Case of Nephropathia Epidemica – A Case Report

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ABSTRACT

A 41-year-old patient was hospitalized in hypotensive phase of nephropathia epidemica. The diagnosis was confirmed by serology. It is highly likely that the patient was exposed to infected aerosol of bank voles in endemic area of Sweden. The disease clinically manifested with fever, headache, abdominal and back pain, vision disturbances. Pathologic laboratory findings included thrombocytopenia, leukocytosis, increased values of C-reactive protein, blood urea and creatinine, abnormal chest X-ray and transient electrocardiogram abnormalities.

Key words: *nephropathia epidemica, Puumala virus, Sweden*

Introduction

Nephropathia epidemica (NE) is a mild form of hemorrhagic fever with renal syndrome (HFRS) caused by Puumala virus, a member of the *Hantavirus* genus in the *Bunyaviridae* family¹. Puumala virus is carried by the bank vole, *Myodes glareolus*. NE is clinically characterized by fever, headache, backpain and gastrointestinal symptoms. Main symptoms in NE is impairment of the renal function, while hemorrhages are possible but very rare. Hypotension is present in 10% of patients. The renal involvement results in transient proteinuria, hematuria and impairment of the renal function. Common laboratory findings are leukocytosis, thrombocytopenia, hypoproteinaemia and moderate elevation of liver enzymes, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). The mortality rate is very low; however lethal cases have been reported². The disease is highly endemic in central and northern Sweden, especially the region near 64 degrees N^{3,4}. The highest incidence rate in Sweden was found in the county of Vasterbotten⁵. However, the number of cases further south seems to increase⁶. Two different hantaviruses, Puumala and Dobrava viruses, were found to cause HFRS in Croatia⁷.

Case Report

A 41-year-old male was admitted to the University Hospital for Infectious Diseases in Zagreb Croatia, with a

four day history of fever (40 °C), chills, shivering, headache and back pain. Before hospitalization he was taking antipyretic drugs (paracetamol and acetylsalicylic acid). The patient, a musician from Sweden, arrived to Croatia on November 25, 2005 for a concert. Two days after his arrival he got ill. He lives with his fiancée in the northern part of Sweden (Kiruna), and sometimes they visit a cottage in the forest between Kiruna and Skaulo (near Gällivare Kommun). Their last visit was two weeks before the onset of the current disease.

His past medical history was unremarkable. On admission the patient was afebrile – Tax. 36.6 °C (after antipyretic therapy), hypotensive (RR 80/50 mmHg), dehydrated, prostrated. Lumbar region was sensitive on succussion. Other clinical status was normal.

On admission several laboratory findings were notable: thrombocytopenia and leukocytosis, followed by changes in the level of haemoglobin, C-reactive protein (CRP), urea, creatinine, and sodium (Table 1). Haematuria and proteinuria were also recorded. Blood and urine cultures were negative. Chest X-ray revealed a small interstitial infiltrate in the right basal segment with pleural reaction. Electrocardiogram (ECG) showed sinus rhythm (58/min), right bundle branch conduction disturbance and anterior left hemiblock. Abdominal ultrasound showed oedematous and enlarged kidneys.

Following admission, appropriate symptomatic and supportive therapy (infusion of crystalloid solutions and

TABLE 1
CLINICAL SYMPTOMS, SIGNS AND LABORATORY FINDINGS IN PATIENT WITH NEPHROPATHIA EPIDEMICA

Symptoms, signs and laboratory findings	Normal values	Day of hospitalization (Day of onset of symptoms)				
		1 (5)	2 (6)	3 (7)	4 (8)	5 (9)
↑ Fever (°C)	> 37 °C	37.4	37.3	36.9	36.8	36.8
Headache		++	++	+	–	–
Abdominal pain		+	++	++	+	–
Backache		+	++	++	+	–
Blurred vision		++	+	–	–	–
CRP (mg/L)	0–10 mg/L	88	55			23
WBC (10 ⁹ /L)	4–10 × 10 ⁹ /L	20.9	14.1	8.3		7.7
RBC (10 ¹² /L)	4.4–5.7 × 10 ¹² /L	5.74	4.74	4.03		4.05
HGB (g/L)	140–180 g/L	177	143	121		121
PLT (10 ⁹ /L)	100–400 × 10 ⁹ /L	35	52	63		175
Urea (mmol/L)	1.8–8.8 mmol/L	9.3	14.1	16.5	17.8	15.2
Creatinine (μmol/L)	35–115 μmol/L	154	286	313	282	252
Sodium (mmol/L)	136–146mmol/L	132		137	141	142
Potassium (mmol/L)	3.8–5.2 mmol/L	4.4		4.4	5.3	4.5
Chloride (mmol/L)	95–105 mmol/L	97		105	107	108
Proteinuria	–	+++	+++	+++		–
Microhematuria	–	++	++	++		–
Diuresis/24 h (mL)		400	1800	3600	6100	

CRP – C-reactive protein, WBC – white blood cells, RBC – red blood cells, HGB – hemoglobin, PLT – platelets, +++ – severe, ++ – moderate, + – mild

paracetamol) was initiated with monitoring of vital parameters. On the first day of hospitalization the patient suffered from visual disturbances, and in the next couple of days from bloatedness and abdominal and back pain. His temperature was normal. During hospitalization, urea and creatinine elevated. On admission to the hospital the patient was oliguric, and until discharge diuresis normalized. Serological testing of antibodies to Puumala virus by enzyme-linked immunosorbent assay (ELISA, Focus Diagnostics Inc., California, USA) was positive (IgM antibody). The patient returned to Sweden in polyuric phase with recommendation to contact his physician for further treatment. Another laboratory check-up (December 12) showed a normalized diuresis and creatinine level (100 μmol/L) (e-mail contact with Magdalena Lindvall, MD, Department of Infectious Diseases Sunderby Hospital Luleå, Sweden).

Discussion

A patient complaining of fever without leading symptoms, and laboratory findings with elevated values of leukocytes, CRP, and thrombocytopenia was hospitalized.

These laboratory findings raised a diagnostic dilemma regarding bacterial infection, especially sepsis. However, substantial epidemiological anamnesis and course of the disease suggested diagnostic approach. Our consideration was confirmed by serology and the patient was cured without antibiotic therapy.

Circulation and persistence of Puumala virus within bank vole populations is influenced by habitat factors⁸. *M. glareolus* seropositive for Puumala virus occurred more frequently near households afflicted with NE than in control areas 10 km away⁹. In northern Sweden the case-patients with NE were more often infected in late autumn, when engaged in activities near or within man-made rodent refuges¹⁰. Contrary to Scandinavian NE, in Croatia HFRS appears during the warm months of the year.

We report this imported case of NE in order to emphasize the importance of regular epidemiological recording of this illness. We believe that the case is interesting since a number of infectious diseases can affect travelers. Health-care providers should consider NE in the differential diagnosis of febrile illness in patients arriving from Scandinavia.

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UVEZENI SLUČAJ EPIDEMIJSKE NEFROPATIJE – PRIKAZ SLUČAJA

SAŽETAK

Četrdesetjednogodišnji bolesnik hospitaliziran je u hipotenzivnoj fazi epidemijske nefropatije. Dijagnoza je potvrđena serološki. Smatramo da je bolesnik bio izložen inficiranom aerosolu voluharice u endemskom području Švedske. Bolest se klinički prezentirala vrućicom, glavoboljom, bolovima u trbuhu i leđima i smetnjama vida. Patološki laboratorijski nalazi uključivali su trombocitopeniju, leukocitozu, povišene vrijednosti C-reaktivnog proteina, ureje i kreatinina, patološki nalaz snimke pluća i prolazni poremećaj elektrokardiograma.