INTRODUCTION

Tick-borne encephalitis (TBE) is caused by a flavivirus with three subtypes: Siberian, European and Russian Spring Summer encephalitis virus, they are transmitted to humans through the tick bite, species Ixodes ricinus or persiculatus, there are sporadic cases described in Europe and in the eastern part of Asia. In 2006 numerous cases have been reported in Russia, Poland, Germany, Lithuania and Slovenia. The type of clinical picture developed is meningoencephalitis, sometimes associated with myelitis and a severe evolution in half of cases occurred in adults with possible neuropsychiatric sequelae and risk of death in 1% of cases. The evolution is biphasic, with general symptoms accompanied by asymptomatic period (1), then in the second week meningocerebral manifestation appear like tremor, cerebellar ataxia, disorders of the state of consciousness, spinal cord impairment, paralysis of manifestation appear like tremor, cerebellar ataxia, disorders of state of consciousness, spinal cord impairment, paralysis of 

THE PHYSICAL EXAMINATION shows a patient with alteration of general condition, fever 38.6°C, with pale skin, first degree of dehydration, stetastic bilateral basal lung emphasized murmur without rales, respiratory rate (RR) 26/min, SaO₂ 93%, HR 72 / min, BP 120/80 mmHg, white tongue, painless abdomen on palpation, with signs of meningeal irritation: neck stiffness, outlines Kernig, Babinski bilateral present and osteotendon hyperreflexia.

Lumbar puncture has been performed, obtaining a clear, hypertension cerebrospinal fluid with 83 elemente/mm³, 100% represented by lymphocytes, proteinorahie was 0.77 mg/dl, 61 mg/dl glucorhie, 113.2 mg/dl chlororhie, on growth medium there was no development of germs.

There have been taken samples for TBE and Lyme disease serology-which were positive. Hydroelectrolytic disturbance treatment has been initiated, cerebral depletion treatment was performed, antibiotics were administered until obtaining the bacteriological examination, have been administered also steroidal antiinflammatory, proton pump inhibitors, symptomatic treatment; under treatment the evolution was favourable and rapid, the patient requested to be discharged on the fourth day of hospitalization.

After 2 days from discharge headaches, emesis reappear, the patient was brought back to the emergency room the next day, when suddenly aplasia and right hemi-body motor deficit was installed. At physical examination the patient shows a rapid alteration of general condition, cardiopulmonary between normal limits, hepatomegaly at 1cm, persistent neck
stiffness, right diminished osteotendinous reflexes, right Babinski sign (+), mixed aphasia, right deficit motor.

Cranial CT scan was performed: cranial MDCT examination native and with iv contrast was performed, with contiguous sections that do not shows infra or supratentorial focused lesions. Median structures were in normal position. Asymmetrical and normotensive ventricular system. Vascular structures were more pericerebral dilated. Without hematic intracranial densities, without localized or diffused areas of edema without mass effect. Conclusions: pericerebral vascular hyperemia

Lumbar puncture was repeat obtaining clear, slightly hypertensive CSF with 9 elemente/mm3, proteinorahie 0.73 g/l, clorurorahie 115.6 mEq/L, glicorahie 87 mg/dL and no development of germs.

Other laboratory examinations: leukocytes: 10200-7960/mm³, erythrocytes 5.35-4.56 mil/mm³, HGB =16.4g-13.6/dl, HCT=47.5-40.6%, MCV=88.8-89.0 fl, MHC=30.7-29.8 pg, MCHC=34.5-33.5/g/dl, PLT= 198000-251000/mm³, NEU 43.4%, LYM 46.1%, MONO=8.7%, BASO 0.3%, EOS1.5%, glucose=7.99mg/dl, BUN(Blood Urea Nitrogen ) 26-16mg/dl, creatinine 0.72-0.67 mg / dL, ESR 55mm/h, fibrinogen 411.8-478.7 mg/l, CPK 89U/L, SGOT 16-52U/L, SGPT 40-133U/L, Bilirubin(total)0.37mg/dl, amylase 46U/l, Sodium =135.6 mEq/l, Potassium =4,13 mEq/l, Chlorde =96,5 mEq/l.

Urinalysis (UA): density 1015, LEU25/ul LEU, KET 5 mg/dl, sediment: rare leukocyte, frequent flat epithelial cells, rare crystals of calcium oxalate.

Abdominal ultrasound: liver, gall bladder, kidney, pancreas, spleen between normal limits, no fluid collection in the peritoneal cavity.

Under antibiotic therapy with ampicillin 8g/day+ ceftriaxone 4g/i, dexamethasone, cerebral deplition treatment, antifungal therapy, PPIs, symptomatic treatment, aphasia and the motor deficit improves in the next 3 days after admission, at the end of the first week of hospitalization the patient became febrile with rare and irritating cough, in parallel with the development of fluid type bilateral basal dullness and evolving crackles rales disseminated in both lung fields.

Pulmonary radiography initially shows the clouding of the costodiaphragmatic sinus then on left lower lobe localized in the posterior and lateral segment opacities with fine drawings of air bronchogram on the profile image, imprecisely defined, inomogen “padded” looking with medium intensity, more obvious on oblique incidence (subsegmentar condensation process). Right perihilar opacity imprecisely defined with the same characteristics. Bilaterally emphasized peribronhovascular interstice and levelled up heart and prominent left middle heart arch (see figures no. 1, 2).

Blood cultures collected and bacteriological examination of sputum was sterile. The antibiotic therapy was escalated associating meropenem with vancomycin, iv antifungal therapy becoming afebrile and imagistic improvemt, the patient was discharged after 23 days of hospitalization.

The serological results and CSF examination confirmed the presence of IgM antibodies for TBE and from serum analyze positiiv IgM for B burgdorferi.

DISCUSSIONS

The biphasic evolution type of our case was suggestive for TBE considering the ocupational risk of exposure. Surprising was the concomitance of borreliosis at this patient that was probably responsible for the biological changes-thrombocytopenia, hepatocytolisis syndrome; we can not sustain the possible involvement in impairment meninocerebral in the absence of examining the CSF for B. Burgdorferi for which the therapeutic attitude (antibiotic therapy) was beneficial. The occurrence of bronchopneumonia as the cardiac involvement are described in the literature for the association with TBE.(2) In our case, the improvement after association of vancomycin suggested a staphylococcal etioly, without having any bacteriological evidence.

The patient did not return to his check up, his health condition is good, he did not consider to be necessary the reassess.

REFERENCES