CASE REPORT

CONFUSION IN A PATIENT WITH MYCOPLASMA PNEUMONIA LOWERS THRESHOLD TO LUMBAR PUNCTURE DESPITE HYPONATREMIA

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ABSTRACT
A 55 year old Indonesian male known case of DM on oral hypoglycaemic drugs presented to ER with productive cough for 3 days. He had no history of SOB, neither haemoptysis, vomiting, haematuria nor bleeding. On physical examination, he appeared disoriented agitated, pale, jaundiced and with good body built. He was not distressed without any cyanosis, clubbing or sign of dehydration. His temperature was 39.3˚C. Laboratory investigation revealed WBC count of 22 /ml, HB 7, 5 gm/dl normochromic normocytic anaemia, normal platelet and normal AB, random blood sugar 320 ml/dl, urea 140 ml/dl, creatinine 2.5 ml/dl , LDL 1500, total bilirubin 3 mainly indirect. CT brain was clear. CSF analysis showed 10 cells 100% lymphocytes, protein 100mg/dl (high) and glucose 160 mg/dl (normal). The patient was initially treated with I.V hypertonic saline, ceftriaxone, vancomycin, acyclovir and dexamethasone, insulin and diet management. Two days later. patient showed improvement in his level of conscious as his Na become 121 mg/dl, urine output 600ml/day, but patient still had high grade fever on-going haemolysis, erythromycin was started and an obvious improvement happened, he become communicating, afebrile, LDH decrease from 2000 to 750, with increase HB level from 6,5 g/dl to 9 g/dl. Conclusively, Aseptic meningitis should be considered in patient with mycoplasma pneumonia presented with confusion despite he has hypernatremia.

KEY-WORDS: Lowers Threshold; Lumbar Puncture; Mycoplasma Pneumonia

Introduction

Mycoplasma pneumoniae is a bacteria causes the disease mycoplasma pneumonia, a form of atypical bacterial pneumonia, is increasingly recognized as a common and an important pathogen in community-acquired respiratory tract infections (RTIs) and pneumonia.[1] The infection affected all age groups but was most common in infants (32.5%) and preschool children (22.5%).[2] It occurred year round but was common in the fall (35%), and spring (30%),[3] which appear as respiratory symptoms but rarely develop CNS manifestation, as studies show low incidence of CNS involvement which is range from 0.1 % to 5% Encephalitis is the most frequent manifestation, but cases of meningitis, myelitis, and polyradiculitis, as well as many other symptoms (e.g., coma, ataxia, psychosis, and stroke), have been reported. The onset of these manifestations is usually acute, with lowered consciousness, convulsions, paresis, and other neurological signs. Severe, even fatal, cases are known. The pathophysiology of CNS manifestations remains clearly unknown. But it could be caused by one of three categories which shown in Table 1.[1]

Table-1: Neurologic Manifestations due to Mycoplasma Pneumoniae Infection, Classified According to the Type of Pathomechanisms that may be involved

	Direct	Indirect	Vascular Occlusion	Unclassifiable
Definition	Inflammation at the local site through the function of cytokines.	Inflammation through autoimmunity, allergy, formation of immune complexes, and similar mechanisms.	Vasculitic or thrombotic vascular occlusion*	—
Manifestations	Early-onset encephalitis; early-onset myelitis; aseptic meningitis	Late-onset encephalitis; late-onset myelitis; cerebellar dysfunction; Guillain-Barre´ syndrome; cranial and peripheral neuropathies	Striatal necrosis; stroke; psychologic disorders	Acute disseminated encephalo-myelitis

* Either vasculitic and thrombotic, or both; with or without systemic hypercoagulable state

Case Report

A 55 year old Indonesian male known case of DM on oral hypoglycaemic drugs presented to ER with productive cough for 3 days. He had no history of SOB, neither haemoptysis, vomiting, haematuria nor bleeding. On physical examination, he appeared disoriented agitated, pale, jaundiced and with good body built. He was not distressed without any cyanosis, clubbing or sign of dehydration. His temperature was 39.3◦C, pulse 100 /min, BP 135/90 mmHg, RR 18/min and O2 98% in room air. There was a decrease in breathe sound with crackles in middle and lower zone bilaterally in chest examination, cardiovascular examination was unremarkable (normal s1, s2, no murmurs), abdomen was soft and lax, no organomegaly. On central nerve examination, GCS=10/15, moving all limbs, neck stiffness, meningeal sign are positive. The remainder physical examinations were unremarkable.

Laboratory investigation revealed WBC count of 22 /ml , HB 7, 5 gm/dl normochromic normocytic anaemia , normal platelet and normal AB, random blood sugar 320ml/dl, urea 140ml/dl, creatinine 2.5 ml/dl , LDL 1500, total bilirubin 3 mainly indirect, NA109, k 3, 7, AST 90 and ALT 88. Culture of sputum was negative. Chest radiograph show sign of airways disease in both lungs most likely consolidation. ECG shows sinus tachycardia with regular rhythm. CT brain was clear. CSF analysis showed 10 cells 100% lymphocytes, protein 100mg/dl (high) and glucose 160 mg/dl (normal).

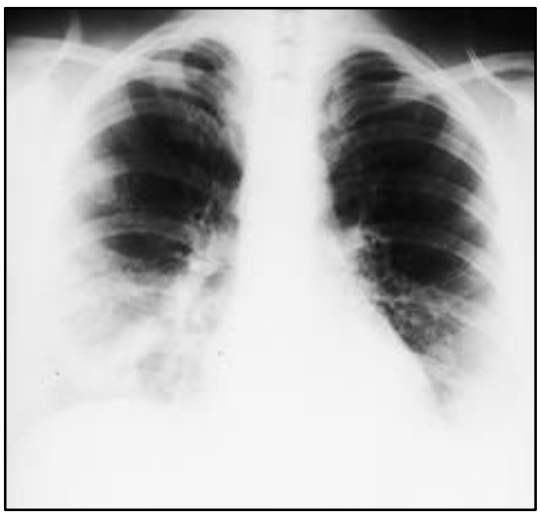

Figure-1: Chest Radiograph Showing Sign of Airways Disease in Both Lungs

Hospital Course

On admission we seek full blood count with differentiation, ESR, peripheral smear, coombs test, electrolytes: potassium 4 mEq/L (normal 3,5 – 5,1), plasma uric acid concentration 1 mg/dl (normal 2-8,5) and hypernatremia 109 mMol/L (normal 135-145), serum osmolarity 200 mosm/kg (normal 280-300 mosm/kg) and urine osmolarity 500 mosm/kg (normal 500-800 mosm/kg). Urine sodium concentration was 68 mEq/L (normal >20 mEq/L). Septic screen was negative, coombs test was positive and urine output 200 ml /day (normal 1 L/day).

The patient was initially treated with I.V hypertonic saline, ceftriaxone, vancomycin, acyclovir and dexamethasone, insulin and diet management. Two days later. patient showed improvement in his level of conscious as his Na become 121 mg/dl, urine output 600 ml/day, but patient still had high grade fever on-going haemolysis, erythromycin was started and an obvious improvement happened, he become communicating, afebrile, LDH decrease from 2000 to 750, with increase HB level from 6,5 g/dl to 9 g/dl.

Discussion

Mycoplasma pneumonia is contagious disease of young adult and children caused by mycoplasma pneumoniae, it's characterized by 9 - 12 days incubation period and followed by a symptoms of an upper respiratory infection.[1]

Aseptic meningitis refers to patients who have clinical and laboratory evidence for meningeal inflammation with negative routine bacterial culture, the most common cause is interovirus, additional etiology include other infections mycobacteria fungi and spirochetes.[4]

There are few cases reported with aseptic meningitis in a patient with mycoplasma pneumonia, according to our research there is a scarcity of reports from Saudi Arabia.

Aseptic meningitis said to occur when the patient have headache, fever, decrease level of consciousness, photophobia, neck stiffness,

malaise, myalgia, chills, sore throat, abdominal pain nausea and vomiting.[5] The CSF manifestation WBC >250 cells/micro L, elevation protein level (generally less than 150 ml/L, normal glucose concentration (45- 80). The CSF analysis and most of clinical manifestation was present in our case and CSF culture was done and it gives negative result, while PCR detect mycoplasma pneumonia antibodies.

Mycoplasma pneumonia produce hydrogen peroxide which is thought to be responsible for much of the initial cell disruption in respiratory tract and for damage of erythrocyte membrane and that probably explain the normocytic hypochromic anaemia.[1]

The clinical examination confirm that the hypernatremia is not the result of decreased effective intravascular volume from volume depletion or from states of volume excess such as congestive heart failure and cirrhosis. While the workup shows hypernatremia, serum hypo-osmolality, urine osmolality >100 mosm/kg (serum osmolality is lower than urine osmolality), decrease urine output, normal urine sodium concentration and potassium and acid base concentration which are going with SIADH criteria.

SIADH (syndrome of inappropriate antidiuretic hormone) is a hyponatremia in a patient due to water retention secondary to increase of antidiuretic hormone.[6] Mycoplasma pneumonia can lead to SIADH although the mechanism by which this occur is not clear.[6]

The SIADH occur when urine osmolality of more than 100 msom/kg in the context of plasma hypo-osmolality is sufficient to confirm ADH excess.[7] Unappreciated water retention causes dilutional hyponatremia. Urine sodium concentration in persons with SIADH is usually more than 40 mEq/L because, in SIADH, sodium handling is not abnormal and the urine sodium concentration reflects sodium intake, which is generally more than 40 mEq/d (usually 50-100 mEq/d).[6] However, the urine sodium concentration in persons with SIADH can be modulated by dietary sodium intake. Thus, on a low- sodium diet, patients with SIADH may have a urine sodium level of less than 40 mEq/L.

Conclusion

This was case of Mycoplasma pneumonia that was complicated by aseptic meningitis. The diagnosis couldn`t made without do lumber puncture which showed a picture of aseptic meningitis. Aseptic meningitis should be considered in patient with mycoplasma pneumonia presented with confusion despite he has hyponatremia .

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