Case report - Thoracic general

Spinal cord compression and epidural abscess extension of pleural empyema

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Abstract

We report a case of epidural extension of pleural empyema with cord compression and neurologic deficit. Surgical decompression was required and emergency bilateral laminectomy was performed with removal of abscess and granulation tissue. Methicillin-resistant Staphylococcus aureus grew in cultures of pleural and epidural specimens and appropriate intravenous antibiotics were started. In spite of early diagnosis and rapid management the patient suffered severe sequelae.

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1. Case report

A 76-year-old woman with depressive disorders, living in an old people’s home, was admitted to our hospital with asthenia, dyspnea, fever and flank pain. Chest X-ray showed a right lower lobar infiltrate and she had a white blood cell count of 27 x 10^9/l. Pneumonia was diagnosed and appropriate intravenous antibiotics were started. Four days later a new chest X-ray revealed a loculate pleural effusion (Fig. 1). Thoracic puncture was performed and purulent thick liquid was extracted for culture. Analytical results revealed glucose levels of 2 mg/dl, LDH 1500 U/l, pH 6.58 and cell count 30 x 10^9/l white cells with 93% neutrophils and no red blood cells. Chest tube was inserted and 700 cc of purulent fluid was evacuated. Methicillin-resistant Staphylococcus aureus (MRSA) sensitive to vancomycin grew in the pleural bacterial culture. The patient was apyretic and pleural fibrinolytics (UROKINASE® Vedin Pharma S.A. Spain) were administered for the next few days. After 3 days, the patient mentioned difficulty in moving her right leg without other symptoms. Neurologic tests showed a sensory and mobility deficit below T8, impaired reflex but conserved sphincter control. Magnetic resonance imaging showed a 5 x 4 cm² lesion in posterior epidural space from conjugate foramen with cord compression from T8 to T11 suggesting an epidural abscess (Fig. 2). Vertebral bodies were partially destroyed. The neurosurgeon performed emergency decompressive bilateral laminectomy from T8 to T11, 20 h after first detecting symptoms. The abscess contained a purulent material and granulation tissue of compressed spinal cord with vascular alteration. A sample was taken for culture while antibiotic treatment was continued and methylprednisolone bolus was administered. After 48 h, the neurosurgeon examined the patient again. She was paraplegic below T8 level without sphincter control. Postoperatory course was uneventful. Chest tube was removed when pleural and sputum cultures were sterile. Oral antibiotics were continued for 6 weeks and the patient was discharged 23 days after neurosurgery. Six months later the patient was still paraplegic without recovery and chest X-ray and CT of longer follow-up were unremarkable. Posteriorly, the patient had a new admission in our hospital to intravenous antibiotics therapy for septic arthritis of the ankle for MRSA and she died of progression of sepsis after 26 days of admission.

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2. Comment

Extension of pleural effusion, caused by microorganisms other than mycobacteria or fungi, to the dural sac is uncommon. Infection of the epidural space occurs in approximately 0.2–1.2 per 10,000 hospitalised patients [1]. Invasion of the dural sac and medullar compression due to extension of pleuropulmonary disease has been described for invasive aspergillosis and some mycobacteria [2]. However, infection by more conventional microorganisms is even rarer. *Staphylococcus aureus* is isolated in 54% of epidural abscesses followed by *Escherichia coli* and other colibacilli [3]. Extension of pleural empyema necessitates towards the epidural space via the conjugate foramen has only been described in infection by invasive aspergillus or mycobacteria. Early diagnosis of the medular compression is essential to prevent irreversible neurologic sequelae. The deficit is produced by compression and, hence, by vascular alteration that can have repercussions on the permanence of the neurologic deficit [1] as in our case. Diagnosis is based on the neurologic examination and the initial symptoms together with NMR that is the elective diagnostic test [4]. Antibiotic treatment is insufficient and surgical medullar decompression is urgent and must be carried out within the first 12 h. Unfortunately, in our case late diagnosis of this process carried late surgical decompression leading to neurologic deficit sequelae.

There is no consensus about the ideal duration of antibiotic therapy but this should be implemented for a minimum of 6 weeks and should be accompanied by monitoring the response with the sedimentation rate and series X-ray controls.

References


