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Intra-Empyema Antibiotic Administration: A Case Report

Laleh Hakemi[1]*, Pirhossein Kolivand[2], Naereh Khodashenas Firoozabadi[1]

[1]Pain Department, Shefa Neuroscience Research Center, Tehran, Iran

[2]Department of Health Economics, School of Medicine, Shahed University, Tehran, Iran

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***Corresponding author:** Laleh Hakemi

E-mail address: *lalehakemi@yahoo.com*

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A B S T R A C T

Untreated empyema has a great morbidity and is potentially lethal. The usual approach for treatment is intravenous antibiotics together with chest tube insertion or thoracoscopic/ surgical interventions. The patient was a 32-year-old man with spinal muscular atrophy and severe intercostal muscular weakness presented with localized pleuritic chest pain and fever. The cause was diagnosed to be parapneumonic empyema. In this case, invasive approaches would potentially exacerbated chest musculature weakness. Other non-invasive managements CT-guided needle drainage and intravenous antibiotic therapy were tried and proved to be ineffective for the patient and the next invasive step would pose considerable risk to the patient. Intra-empyemic antibiotic administration through a small tube catheter was successfully effective without any recurrence. This method was non-invasive with no significant risk to the patient, had a low cost and was accepted by the patient. It was shown to be effective without any complication and saved the patient from performing an invasive procedure. This non-invasive approach may be used in those patients who are of high risk for invasive procedures. Also, potentially it may be used as one of the first line approaches before performing more invasive modalities.

1. Introduction

Spinal muscular atrophy (SMA) is a lower motor neuron disease with autosomal recessive inheritance that results in progressive proximal muscle weakness and skeletal muscle atrophy. In 1891, Werdnig first reported two cases of SMA with common features, and genetic homogeneity was proved in 1990. SMN1, the gene responsible for it, was identified in 1995.[1] SMA is a neurodegenerative disease characterized by the degeneration of spinal cord motor neurons, atrophy of skeletal muscles, and generalized weakness. It is caused by homozygous disruption of the survival motor neuron 1 (SMN1) gene by deletion, conversion, or mutation. No effective treatment is available. Advances in medical technology have changed the standard of care for patients with SMA, primarily through supportive care measures.[2]

The most important cause of morbidity and mortality in spinal muscular atrophy (SMA) is respiratory complications. The key respiratory problems in SMA are impaired cough resulting in poor clearance of lower airway secretions, hypoventilation during sleep, chest wall and lung underdevelopment, and recurrent infections that exacerbate muscle weakness, eventually causing respiratory failure.[3] Empyema is usually classified as complicated parapneumonic effusion with a high mortality rate.[4,5] Pleural infection is an ancient disease that remains an important clinical problem. In recent decades, with unknown reasons and probably influenced by aging and more survival in the underlying conditions, the

incidence of pleural infection has increased worldwide. Early surgical decortication is superior to chest tube drainage in advanced empyema in terms of outcome and duration of hospitalization.[6] Patients with symptom duration of less than 4 weeks showed better outcomes of Video-assisted thoracoscopic surgery compared to those with a duration greater than 4 weeks.[7]

2. Case Presentation

A 32-year-old man with spinal muscular atrophy type II was referred to the ED with severe left chest and flank pain for 1 month. The pleuritic chest pain increased with inspiration and disrupted normal sleep. Mild non-purulent cough and dyspnea accompanied the pain. Till one week before, he would be able to walk with the aid of an accompanying person or wall; however, several days before hospital admission, he had been prescribed methocarbamole for pain relief, subsequently he had a fall and ankle sprain. On hospital admission, left ankle and knee were painful and he could not walk and was wheelchair bound. His vital signs were stable and he was not febrile. Because of chest wall pain he received intravenous apotel in the ED which caused urticarial skin rashes. His height was 165 cm and weight 30 kg. In his medical history, when he was just 1 year old, muscular weakness emerged, and SMA was diagnosed through genetic testing several years later. He had osteoporosis and frequent fallings due to weakness, with subsequent multiple fractures in the skeleton. He has a history of hospital admission 20 years ago because of a left femur fracture. Now, he was taking calcium-D as a daily oral supplementation.

Physical examination revealed severe generalized muscular atrophy. Also, he had urticarial lesions scattered all over the body. Mild erythema on the lower left chest wall was present. There was moderate tenderness on palpation in the left chest. On auscultation, lung fields were clear. Cranial nerves were intact. The muscular force was 4/5 in the upper and 3+/5 in the lower extremities. Deep tendon reflexes were decreased generally (0-1+). Urticarial lesions were managed with antihistamines and a corticosteroid shot.

On laboratory evaluation, the sedimentation rate was increased to 94 mm in the 1st hour, hemoglobin was 11.5 g/dl and MCV 91.2 fl. White blood cell count showed significant leukocytosis 27940/micL with toxic granulation and 85% neutrophil on differentiation count. The platelet count was 854000/micL with giant platelets. In spiral computerized tomography scanning of the thorax without contrast, there were two thick wall loculated lenticular pleural fluid collections in the left hemithorax with slight pleural effusion in the left main fissure as well as pleural thickening. (Figure 1) Spirometry showed a severe mixed obstructive + restrictive pattern. (FEV1/ FVC = 92.42% – FEV1= 1.61 L- FVC= 1.74 L).

Lung perfusion scanning showed intermediate probability of pulmonary thromboembolism; color doppler sonography of lower extremities both for artery and vein were within normal limits. Multi-slice spiral chest CT angiography showed patent arteries; however, evidence of an extrapulmonary large cystic mass in the paraspinal space in the left hemithorax together with a small similar cyst in the left oblique fissure was evident. The whole body bone scan did not show any active lesion in the thorax; however, it showed active growth plates at the lower extremities, especially at the distal left femur and distal right tibia. Multi-slice spiral CT scanning of the right ankle joint without contrast revealed diffuse osteopenia and coarse trabeculation and thinning of cortex due to disuse, with no evidence of fracture or dislocation.

Left pleural space sonography showed 180 ml fluid with internal echoes; 160 cc thick creamy olive-colored fluid was aspirated through a no.16 gray angio-catheter and sent to the laboratory. Pleural fluid culture showed *Streptococcus pneumoniae* and anaerobes. The blood culture did not show bacterial growth. Because of the general status of the patient, chest tube insertion would further compromise chest wall muscle weakness and be evaluated to be high risk. Antibiotic therapy (intravenous targocid 400 mg

once per day + meropenem 1g three times per day) was ordered, however, pain did not vanish and signs of infection and inflammation like leukocytosis, high erythrocyte sedimentation rate and C-reactive protein did not show a favorable change. In addition, the empyemic loculation re-emerged in the sonographic examination.

This approach of drainage and intravenous antibiotic was repeated the next time, again with no desirable clinical and para-clinical outcomes. So, we modified the standard therapeutic approach and the abscess was discharged by CT-guided insertion of a drain remained for 10 days and 20 cc of metronidazole was injected daily intra abscess and the catheter was clamped for 1 hour. Clinical, laboratory and radiological signs of the empyema came back to normal. The patient fully recovered from the empyema, and there was no recurrence in the follow-up visits. No adverse effect was noticed, and the process was highly tolerable for the patient.

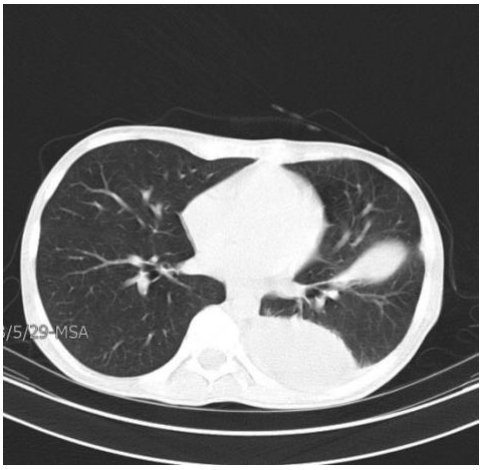

Figure 1. Computerized tomographic scanning of the chest.

3. Discussion

Empyema is a subtype of pleural effusion due to bacterial infections. The diagnosis relies on chest X-ray, ultrasonography, and/or computerized chest tomography.[4-6] Usually, there are three stages of pleural infection. The first stage is the early exudative stage with simple, freely moving exudate with increased capillary permeability resulting in the movement of fluid into pleural space without a bacterial infection in pleural fluid. in this stage, intravenous antibiotics may be sufficient, and a chest tube is not required. If this stage is left untreated or not sufficiently treated, it progresses to the fibrinopurulent stage, in which the bacteria invade and multiply in the pleural cavity, accelerating the migration of neutrophils and activation of coagulation cascades. Fibrin deposition and septation formation occur in the pleural fluid. The last step is the organizing step, in which fibroblast proliferation forms a thickened fibrous pleural peel encasing the lung and preventing lung expansion.[7-10]

Untreated empyema may progress to a fibrothorax with pleural peels. Empyema necessitans is a rare condition in which an empyema extends into the soft tissues of the thorax after eroding through the parietal pleura.[11,12] Depending on the patient's clinical status and underlying diseases, it has a high mortality rate of 5.4% to 22%.[5] The mainstem of treatment is antibiotic administration together with appropriate drainage of the loculation. The MIST1 large, double-blind, randomized trial did not show significant beneficial effects of intrapleural streptokinase in mortality, surgical referral rate, and the length of the hospital stay. The MIST2 used recombinant tissue plasminogen activator (t-PA) and DNase. Combined t-PA and DNase therapy significantly outcomes

compared to the placebo group, but t-PA alone or DNase alone did not produce any significant difference compared to the placebo group. However, there is an absolute difference in a number of surgical referrals.[13-15] In a study on 111 subjects with empyema, Surgical decortication showed a better treatment success rate in all subjects compared with simple drainage (P <.0001) even after adjustment for compounding factors (odds ratio (P = .014).[6-9]

The patient was shown to be affected by purulent empyema, diagnosed by computerized tomography and chest sonography. The stage of the empyema was the second stage (fibrinopurulent) not responded adequately to intravenous antibiotic and loculation drainage with recurrences. The patient was experiencing pain and was clinically toxic. Because of extensive muscular atrophy including chest wall and intercostal musculature, general anesthesia and/or any surgical approach involving the chest even with local anesthesia would cause consequent deterioration in chest muscular strength and cough. Subsequently, intra-empyemic administration of metronidazole together with intravenous antibiotics showed favorable outcomes, no side effects without any recurrence.[16-20]

4. Conclusion

Intra-empyemic, together with intravenous antibiotic administration, may be used in those patients who are at high risk for invasive procedures. Also, potentially, it may be used as one of the first-line approaches before performing more invasive modalities.

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