MENDELSON’S SYNDROME COMPlicated BY ACUTE RESPIRATORY DISTRESS SYNDROME

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ABSTRACT The article presents a clinical case of aspiration pneumonia (Mendelson’s syndrome) complicated by the acute respiratory syndrome. Clinical symptomatology of patient S., 39 years old, hospitalized at the University Clinic of Ivano-Frankivsk on November 5th, 2012, included shortness of breath and cough predominantly, and hemoptysis was observed. Extensive (almost total) radiological changes in both pulmonary fields dissonated with relatively poor auscultatory data, namely, only weakened vesicular respiration was heard throughout the clinical observation period. The data of X-ray examination somewhat resembled the disseminated process, and therefore in some cases were interpreted as a case of the firstly diagnosed pulmonary tuberculosis (FDPTB), and later as viral and bacterial pneumonia due to the epidemiological situation regarding the outbreak of California influenza in 2009-2011. However, with the scrupulous survey, we managed to find out that a prerequisite for breathlessness and coughing was repeated vomiting in a patient in the evening and night because of frequent epileptic attacks. This fact contributed to the formation of a proper clinical diagnosis of aspiration pneumonia complicated by acute respiratory distress syndrome. The peculiarity of this clinical observation was the fact that it occurred on the comorbidities background (epilepsy; chronic internal haemorrhoids, complicated with rectal bleeding on October 3rd, 2012 and posthemorrhagic anaemia; acute deep vein thrombosis and acute ascending thrombophlebitis of the varicose diluted saphenous veins of both lower limbs). However, with adequate mask mechanical ventilation, antibiotic therapy, mucolytics, clexane, cardiomagnyl, normoven, elastic compression of the lower extremities, dosed exercises, a rapid clinical and particularly radiological dynamic on November 9th, 2012 was noted. Due to this, on December 13th, 2012, the patient was discharged in satisfactory condition for outpatient treatment.

KEYWORDS Mendelson’s syndrome, aspiration pneumonia, acute respiratory distress syndrome (ARDS), epilepsy, X-ray examination, treatment

Introduction

Acid-aspiration pneumonia (Mendelson’s syndrome), or acute exudative pneumonitis, was described in 1946 as the most severe complication of anaesthesia in obstetric practice during childbirth. [1] Issues of diagnosis, treatment and prevention remain relevant today due to high mortality (40-50% and above). [2] Its development is based on the aspiration of acidic gastric juice with a pH of 2-2.5 in a volume of fewer than 25 ml. [3] With increasing volume and its acidity, the severity of the clinical manifestation and the probability of lethal outcome increase. [4] It is established that damage to the epithelial and endothelial layers of cells and edema can occur at low pH - 5.0, such as during bile aspiration. [5] There is a chemical burn of the mucous membrane of the respiratory tract with damage to the epithelium of the trachea, bronchi, bronchioles, burns of the alveoli and capillary endothelium, which can cause pulmonary edema and the development of acute respiratory distress syndrome. At the same time, edema of the mucosal and submucosal layers of the bronchial tree causes obstruction, total bronchospasm and hypoxic respiratory failure. [3] This usually occurs during anaesthesia, intoxication, use of sedatives, coma, convul-

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sions, during emergency child delivery surgical interventions, trauma of face and neck, tumors of the esophagus and trachea, gastro-oesophageal reflux, conditions after gastrectomy, diseases of the central and peripheral nervous system, mechanical and iatrogenic interventions (endotracheal tube, nasogastric tube, tracheostomy), airway obstruction by a foreign body, vomiting of various genesis and in elderly and old age people, etc. [6] The respiratory tract is contaminated in 50% of cases by anaerobic flora, in 40% by combined, and only in 10% by aerobic flora. [7],[8] The clinical presentation is characterized by acute onset, obvious signs of respiratory failure or bronchospasm, possible expiratory dyspnea, grey cyanosis, even when 100% oxygen is given. The typical triad includes tachycardia, tachypnea, cyanosis. [9] Often a drop in blood pressure, cardiovascular disorders, wheezing over the lungs, crepitation in the lower lungs, the radiograph resembles a 'shock lung' - diffuse darkening of the lung tissue are present. When infectious factor combines, the considerable increase in temperature, cold and even, development of abscessing might possibly occur. [2],[3] The effective treatment consists of ventilation with high pressure at the end of exhalation, and in severe cases, hyperbaric oxygenation; large doses of corticosteroids, as well as antibacterial drugs. [10]

Case report

Patient S., 39 years old, unemployed, due to California flu epidemic in 2009-2011 was admitted to the clinic of the Medical University of Ivano-Frankivsk on November 5th, 2012 with a preliminary diagnosis of viral and bacterial pneumonia of group IV, severe form. ARDS. Respiratory failure, third degree. On admission, he complained of a cough with pink and purulent sputum, severe shortness of breath, general weakness, loss of appetite, weight loss, the appearance of painful longitudinal condensed cord on the inner surface of the left thigh and lower leg, periodic rectal bleeding, and epileptic seizures. Upon obtaining anamnesis carefully, it was determined that on November 1st, 2012, on the background of an increase in epileptic seizures, he had repeated vomiting, after which there was severe shortness of breath, cough, temperature 39 °C, cold. Vomiting in epileptic seizures had been observed repeatedly before. From the of ambulance referral, from November 1st, 2012 was treated in the intensive care unit of the central district hospital, in serious condition, with shortness of breath, RR 35/min, blood oxygen saturation 51%, and on November 3rd, 2012 was transferred to the internal medicine department. The anamnesis revealed that the patient had been suffering from epilepsy for 17 years (received finlepsin medication), had internal haemorrhoids for a long time, and had periodic rectal bleeding after defecation. Allergic anamnesis was not burdened; the patient denied a history of infectious hepatitis, tuberculosis and hereditary diseases in himself or his family members.

The full blood count (FBC) performed on November 1, 2012 in the Central District Hospital showed: Hb - 74 g / l, erythrocytes - 2.5 x 10¹² per liter, colour index (CI) - 0.9, leucocytes - 11.9 x 10⁹ per liter, rod-nuclear neutrophils - 20%, segmental neutrophils - 72%, lymphocytes - 6%, monocytes - 2%, ESR - 20 ml/h, hematocrit - 0.19.

Biochemical blood analysis done on November 1, 2012: total bilirubin - 11.3 mcml/l, direct - 2.2 mcml/l, ALT - 0.48 and ALT - 0.42 mlmol/l, urea - 7 mlmol/l, creatinine - 121 mcml/l, total protein - 69.6 g/l, Ca - 2.4 mlmol / l, chlorides - 99 mlmol/l. Dynamics of the coagulogram on November 1, 2012: prothrombin time - 14, 23 and 24 °, prothrombin index - 108, 66 and 63%, respectively.

Chest X-ray (CXR) performed on November 1 and 2, 2012 revealed: on both sides diffused foci of infiltration and consolidation with blurred contours, sinuses are free. The heart is spread to the left. Conclusion: disseminated pulmonary tuberculosis? Radiographs were consulted on November 2nd, 2012 by a radiologist of the tuberculosis dispensary. Conclusion: bilateral non-hospital pneumonia? Pulmonary tuberculosis is not excluded (Figure 1 and 2).

Abdominal ultrasound was done on November 2nd, 2012: liver - right lobe 15 cm, left lobe - 7.7 cm, echostructure was compacted. The gallbladder was deformed, the walls were compacted, the constriction was closer to the bottom, content was stagnant. Pancreas - partially visualized in the body region 1.1 to 5 cm, not widespread, the echostructure was compacted. The spleen was slightly enlarged, 11.2 by 7.3 cm, the echostructure was somewhat compacted. Kidneys were normal in size, compaction of pelvicalyceal complexes with small echo (+) inclusions. Free fluid in the abdomen and pleural sinuses was not detected.

The ECG performed on November 2nd, 2012 revealed: sinus tachycardia with a heart rate of 100/min, normal position of the electrical axis of the heart, disorders of myocardial repolarization, incomplete left his bundle branch block.

Echocardiography of the heart on November 2, 2012: aorta - 3.0 cm, left atrium - 3.7 cm, mitral valve - M-type, left ventricle: end-diastolic size - 4.8 cm, end-systolic size - 3.2 cm, ejection fraction - 56%, intraventricular septum in diastole - 1.1 cm, posterior wall of left ventricle in diastole - 1.1 cm, right ventricle - 2.7 cm. Conclusion: heart cavities within normal limits, good myocardial contractility, left ventricular hypertrophy. Upper gastrointestinal endoscopy on November 2nd, 2012: the esophagus was freely passable, the stomach was straightening satisfactorily, the mucosa was hyperemic, numerous hemorrhagic erosions were noticed. The pylorus gaped. No abnormalities were detected in the duodenum. Conclusion: erosive hemorrhagic gastritis. The patient was consulted by a surgeon on November 3rd, 2012. Chronic internal hemorrhoids, complicated with rectal bleeding. Posthemorrhagic anemia.

He received treatment: mask inhalation with oxygen, magnesium sulfate, L-lysine, mexidol, rheosorbil, sodium bicarbonate, cytochrome, ceftriaxone, sumamed, levofloxacin, thiotriazoline, mucolvan, euphylline, and controloc.

On November 4th, 2012, the patient underwent control radiography of the chest. The radiograph features remained with diffuse infiltrative changes and insignificant dynamics of the pathological process in the lungs (Figure 3), after which the patient November 5th, 2012 was transferred to the regional Pulmonology center with the diagnosis of first diagnosed TB. Chronic disseminated pulmonary tuberculosis, category I, Destruction (-), Mycobacterium tuberculosis (-). Hemoptysis. Bilateral non-hospital bacterial pneumonia, severe form?

Looking at the clinical presentation, distinctive radiological picture and the recent California influenza epidemic of 2009-2011 in Ukraine, the patient was hospitalized on November 5th, 2012 to the university clinic with a previous diagnosis of viral and bacterial pneumonia of group IV. ARDS. Respiratory failure, III degree. In addition to the above complaints of cough with pink and purulent sputum, severe shortness of breath at rest and during exercise, the patient noted the appearance on the inner surface of the lower third of the left thigh and upper part of shin painful hyperemic longitudinal seal in the form of a cord.
A careful survey revealed that the patient had significantly more frequent epileptic seizures, and on November 1st, 2012 during an epileptic seizure in the evening he had vomiting, after which there was severe shortness of breath, temperature 39 ° C, cold.

Objectively: the patient's condition was severe. The position was passive, mostly lying down. The respiration rate at rest was 26, SPO2 at rest 80-82%, with mask ventilation 94-95% with FIO 2 - 0.6. Mucous membranes were pale and cyanotic. The skin had increased moisture. Peripheral lymph nodes, thyroid gland, were not enlarged. The veins of both lower extremities were varicose. On the left, on the inner surface of the lower third of the thigh and the upper part of the shin, there was a longitudinal painful compacted cord 10 cm long with redness of the skin on its surface. In almost all areas of the chest on both sides, the dull percussion sound was found. Significantly weakened vesicular breathing was determined here, other pathological physical changes over the lungs were not heard. The boundaries of the heart were not determined due to severe dullness found above. Heart tones were rhythmic, weakened, heart rate was 100/min, blood pressure was 120/80 mm Hg. The abdomen was not painful, and the liver was near the edge of the costal arch; the spleen was not palpable, the segments of the intestine had no abnormal features. Pasternatsky's symptom was negative on both sides.

In the analysis of blood on November 6, 2012: Hb - 70 - g/l, erythrocytes - 3,26x1012 per liter, CI - 0,65, leukocytes - 6,8 x109 per liter, eosinophils - 1%, rod-nuclear neutrophils - 4%, segmental neutrophils - 72%, lymphocytes - 15%, monocytes - 8%, ESR - 30 mm/h; in the biochemical analysis of blood: thymol test - 2.77 units, total bilirubin - 8.38 mcmol/l, direct bilirubin - 2.1 mcmol/l, ALT - 1.66 and AST - 1.35 mmol/l, creatinine - 112 mcmol/l. In coagulum on November 8, 2012: Prothrombin time of 16.9”, the prothrombin ratio - 1.0, INR - 1.1, prothrombin index - 84%, APTT - 17.9”. In the analysis of urine on November 6, 2012: specific gravity - 1025, pH - 6.5, protein - 0.3 g/l, leukocytes - 3-5 in the view field, squamous epithelium - 2-7 in the view field, mucus homogenous ++, amorphous urates +++ were also found in the sediment.

CXR performed on November 1, 2, 4 in 2012 were reviewed again and consulted by a specialist of the clinic. X-ray changes on them were interpreted as diffuse disseminated focal and infiltrative shadows of different sizes and densities, places of
consolidation in the upper lungs and basal areas. In the projection of the costo-diaphragmatic sinuses, the transparency of the lung tissue was preserved. Due to the consolidation changes in the lungs, the roots of the lungs were not contoured. The heart was within normal limits. Conclusion: bilateral pneumonia. Hemogenous disseminated tuberculosis?

According to urgent indications, on November 6th, 2012, the patient underwent dopplerography of the vessels of the lower extremities. It was found that the posterior tibial veins on both sides were filled with thrombotic masses, without recanalization. Varicose saphenous veins were also filled on both sides from the middle of the thigh in the distal direction with thrombotic masses, about which the patient was urgently consulted by an angiologist.

In the hospital the patient was prescribed mask oxygen therapy with FiO 2 - 0.6, restrictive type of infusion therapy with leflocin 0.75 drops, solumedrol 0.04 per 50 ml of saline, amikacin 0.5 intramuscularly twice a day, mucovalan 4.0 daily twice a day, bronchisan, finlepsin 0.2 in the morning and 0.4 in the evening, omeprazole 0.02 twice a day, sorbifer 1 tablet twice a day, glutargin 0.75 twice a day, and according to the angiologist prescription - clexane 0.4 twice paraumbilical, normoven 1 tablet twice a day, topically on the skin of the lower extremities heparin ointment, elastic bandaging of both lower extremities, dosed exercises, cardiomagnil 0.075 at night. During treatment at 2 o’clock at night of November 6th, 2012, the level of SpO 2 became stable without the need for mask ventilation by 97-98%, pink sputum became less frequently coughed up, and the patient’s condition remained stable-severe in terms of the amount of pathology. Motor activity was limited due to vascular pathology. Breathing remained weakened, wheezing, rhonchi and crepitations were not heard. Respiration rate was 22/min, and the heart rate was 90 beats per minute, blood pressure was 120/80 mm Hg. Looking at the clarified anamnesis data, particularly the past medical history, which included the presence of repeated vomiting caused by a series of seizures, the diagnosis was changed to bilateral aspiration pneumonia complicated by acute respiratory distress syndrome and respiratory failure, III degree. According to Doppler results performed on November 9th, 2012, the data corresponded to the previous study, the treatment prescribed by an angiologist was continued. In the following days, breathing became better over the lungs, and the dull percussion sound disappeared. According to the control X-ray on November 9th, 2012, a significant positive dynamic was noted - only the enhanced pulmonary pattern on both sides remained. The roots of the lungs were without a clear structure; pleural sinuses and diaphragm had normal contours. The boundaries of the heart were normal. Conclusion: Condition after pneumonia (Figure 4).

In the control blood test on November 13, 2012: Hb increased to 98 g/l, erythrocytes - up to 4.3 x 10^12, CI to 0.7, leukocytes - 7.7x10^9, rod-nuclear neutrophils - 2%, segmental neutrophils - 54%, eosinophils - 1%, lymphocytes - 33%, monocytes - 10%, ESR was still elevated to 42 mm/h. Urine analysis was without pathological abnormalities.

Thus, the patient was discharged on November 13th, 2012, in a satisfactory clinical condition to continue treatment in an outpatient setting with the following clinical diagnosis: Bilateral aspiration pneumonia, oxygen-dependent, severe form. Acute respiratory distress syndrome. Respiratory failure, III degree. Hemoptysis.

Erosive hemorrhagic gastritis. Chronic internal haemorrhoids complicated by rectal bleeding. Severe posthemorrhagic iron deficiency anemia. Epilepsy, a condition after a series of seizures that caused vomiting and aspiration of gastric juice. Acute deep vein thrombosis of both lower extremities, tibial segment. Acute ascending thrombophlebitis of varicose saphenous veins of both lower extremities.

Conclusion

1. The peculiarity of this clinical case is that such clinical symptoms as cough, shortness of breath, a sharp decline in SpO 2 the presence of significant physical changes (dull percussion sound) on both sides and extensive radiological changes can simulate the disseminated process.

2. At the same time, although the percussion and radiological changes were extensive and pronounced, wheezing, rhonchi, crepitations and other auscultatory pathological symptoms over the lungs, except for reduced vesicular breathing may not be detected during the entire period of observation of the patient.

3. The difficulties in managing this patient were caused by a presence of comorbidities (epilepsy, complicated by vomiting, erosive gastritis and internal haemorrhoids with secondary posthemorrhagic anemia and acute deep vein thrombosis of the lower limbs and acute ascending thrombophlebitis of varicose saphenous veins of both lower limbs).

4. Adequate antibacterial therapy, mask artificial ventilation, restrictive infusion therapy and corticosteroids led to rapid clinical and radiological dynamics, which resulted in denying other disseminated processes, including disseminated pulmonary tuberculosis.

5. Finally, this clinical observation once again demonstrates that despite numerous modern additional and instrumental examinations and patient being examined by many specialists, the history of repeated vomiting due to increasing epileptic seizures was crucial in the diagnosis. In this case, the gold truth remains absolutely correct, that properly collected anamnesis makes from 50% and even up to 75% of accurate and reliable diagnosis.[11]

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Conflict of interest

There are no conflicts of interest to declare by any of the authors of this study.

References


