



SUCCESSFUL TREATMENT OF DESCENDING NECROTIZING MEDIASTINITIS IN INTENSIVE CARE UNIT

Mehmet Salih SEVDI	M.D. , Specialist Anesthesiologist
Meltem TURKAY	M.D., Specialist Anesthesiologist
Tumay ULUDAG YANARAL	M.D., Specialist Anesthesiologist
Dogan KILIC	M.D., Specialist Anesthesiologist
Kerem ERKALP	M.D., Assc. Prof., Specialist Anesthesiologist
Aysin ALAGOL	M.D., Prof., Specialist Anesthesiologist

ABSTRACT

Mediastinitis is inflammation of the tissues in the mediastinum due to infection and external causes other than infection, it can be either acute or chronic. Its most common cause is perforation of the esophagus but it can develop after a lung infection, pleural effusion or descending necrotising mediastinitis (DNM). Descending necrotising mediastinitis develops when odontogenic infections create abscess formations in retropharyngeal space and extend to the mediastinum. Descending necrotising mediastinitis is a serious disease that may result in empyema, pleural-pericardial effusion, pericarditis, intrathoracic hemorrhage and cardiac tamponade due to acute polymicrobial infections and may lead to a mortality rate of 30-50%. Early diagnosis, effective antibiotherapy against GR (-), GR (+) and anaerobic microorganisms, thoracotomy, cervical drainage and debridement of necrotic tissue are life savers. In this paper, we intend to present 2 cases where mediastinitis developed, one following a common neck infection after tooth extraction and the other due to a throat infection.

KEYWORDS

Mediastinitis, tooth extraction, intensive care unit

INTRODUCTION:

Mediastinitis is inflammation of the tissues in the mediastinum due to infection and external causes other than infection, and can be either acute or chronic. Acute mediastinitis is observed at the rate of 0.5-5%. Primary reasons for acute mediastinitis are esophagus perforation (90%), tracheobronchial injuries and infections after cardiac surgeries. Secondary reasons include lung infection, pleural effusion and descending necrotising mediastinitis (DNM) (10%).^{1,2}

Descending necrotising mediastinitis develops when odontogenic (second and third teeth) and oropharyngeal infections transform into abscess formations in the peritonsillar and retropharyngeal spaces due to insufficient antibiotherapy and reach posterior mediastinum through parapharyngeal lymphohematogenous route.³⁻⁵

The most commonly isolated anaerobic agents in DNM development due to head and neck infection are viridans streptococci and peptostreptococci. Bacteroides fragilis and Fusobacterium spp. are less commonly observed agents. And the most frequently isolated aerobic agent is Pseudomonas aeruginosa.^{6,7}

In this paper, we aim to present our clinical observations and treatment experience in the intensive care unit (ICU) about 2 cases which developed mediastinitis after tooth extraction and throat infection.

Case I: While the male patient, 49, had a tooth extracted five days ago and was being followed in the otorhinolaryngological clinic because of complaints of redness in the left side of the neck, pain and fever, he was hospitalized in the ICU when he developed general condition (GC) disorder and respiratory distress. In the first physical examination (PE) of the patient

not having an additional pathologic disease, the GC was mild; he was conscious, cooperative; his orientation was full, his Glasgow Coma Scale (GCS) was 15/15; and when they were listened to, the respiratory sounds in the lower lobes in both hemithoraxes were low. The records were as such: Body temperature (BT): 38.5 °C, arterial blood pressure (ABP): 110/70 mmHg, heart rate (HR): 120/min., peripheral arterial oxygen saturation (SpO₂): 85%. C-reactive protein (CRP) in the blood biochemical sample: 401 mg/dL, white blood cells (WBC): 11.92 10/mm³, and other laboratory values were normal. In the arterial blood gas, the records were as such: (ABG) pH: 7.33, PaO₂: 71 mmHg, PaCO₂: 58 mmHg, Be_b: 3.6 mmol/L, SaO₂: 59%, HCO₃: 27 mmol/L. 4 L/min of oxygen mask was administered. Cultures of blood, urine and sputum samples were taken. Common air and liquid collection was observed in the thorax through radiological imaging of the neck and mediastinum by computerized tomography (CT) (Figure 1). In the neck ultrasonography (USG), we observed an abscess consistent with inflammation in the range of 4x2.5 cm soft tissue, starting from the supraclavicular region and extending from the posterior of the left sternocleidomastoid (SCM) to the thyroid gland posteriorly and to the thyroid gland vicinity and to the right SCM posteriorly. As an empirical treatment, we started antibiotherapy by administering meropenem (3 gr/day), vancomycin (2 gr/day) and metranidazol (2 gr/day). The patient was examined by the thoracic surgery clinic, diagnosed with mediastinitis and taken to surgery. Right posterolateral thoracotomy, mediastinal abscess drainage and necrotic tissue debridements were made. A thorax tube was inserted from the anterior for mediastinal irrigation. The patient under the post-operative mechanical ventilation support was administered midazolam 5 mg/hour and remifentanyl 0.3 mg/hour infusion with the aim of sedation. The patient in the mechanical ventilator was examined in the mode of synchronized intermittent mandatory ventilation pressure control (SIMV-PC), and his val-

ues were as such: Fraction of inspired oxygen (FiO₂): 50%, inspiratory pressure (PI): 12 cmH₂O, frequency (F): 14/min, positive end-expiratory pressure (PEEP): 6 cmH₂O, P-Support (PS): 10 cmH₂O. During control posteroanterior lung graphy and infiltrative areas in the right side were observed. During neck ultrasonography (USG), a collection whose widest range was 2 cm was observed in the area from the left supraclavicular region to the 1 cm posteriority of the SCM muscle. Clindamisin (1.2 mg/day) was added to the antibiotherapy. Postoperative third day sedation was finished. After a follow-up in the mode of continuous positive airway pressure (CPAP), he was taken to the T tube for 30 minutes. His arterial blood gas controls were as normal and so he was extubated by applying a leakage test. His treatment continued with 2 L/min of oxygen with nasal cannula. Place, time and space orientations were assessed completely. Staphylococcus haemolyticus and staphylococcus epidermidis were reproduced in his blood culture sample. No pathologic agents were reproduced in his sputum and urine culture. His arterial blood, gas and biochemical samples were normal and his control thorax CT and vital findings were stable; he was transferred to the thoracic surgery clinic in the postoperative 14th day, fully recovered.

Case II: When a male patient, age 50, was followed due to complaints of a sore throat, he was hospitalized in the ICU when neck infection, pleural effusion, pneumonia, and acute renal failure were detected. In the first physical examination of the patient with coronary artery disease and hypertension, the GC was mild; he was conscious, cooperative, his orientation was full, his Glasgow Coma Scale (GCS) was 15/15; and when listened to, the respiratory sounds in the lower lobes in both hemithoraxes were low. There were ralls and rhoncus in the bilateral apical regions of the patient who has his thorax tube on his right side. The records were as such: BT: 38.0 °C, ABP: 130/90 mmHg, HR: 130/min, SpO₂: 90%. C-reactive protein (CRP) in the blood biochemical sample: 359 mg/dL, white blood cells (WBC): 20.95 10/mm³, and other laboratory values were normal. Regarding the arterial blood gas, the records were as such: pH: 7.37, PaO₂: 71 mmHg, PaCO₂: 50 mmHg, Be_b: 0.6 mmol/L, SaO₂: 90%, HCO₃: 24 mmol/L. In the sporadic CPAP mode, FiO₂: 40%, PEEP: 5 cmH₂O, PI: 5 cmH₂O were planned with non-invasive mechanical ventilation. Cultures of blood, urine and sputum samples were taken. In the thorax CT, the left major fissure and pleural effusion in bilateral hemithorax were tracked. Free air images in the right paracardiac area and the collection (nearly 4.5x2.2 cm) was observed (Figure 2). In the neck USG, a collection was observed in the right side of the middle line of the neck. Percutaneous abscess drainage was applied to the collection in the neck. Ceftriaxone sodium (2 gr/day) and clindamisin (1.2 gr/day) treatment was stopped. We started antibiotherapy by administering meropenem (3 gr/day), vancomycin (2 gr/day) and metranidazol (2 gr/day). No pathologic agent was reproduced in his culture samples. Hemodynamics and vital findings of the patient were stable on the tenth day of his hospitalization, thus he was transferred to the thoracic surgery clinic on the 10th day.

After the 20th day of his hospitalization, the free air image and collection were found to be extending in the right paracardiac area, according to the control thorax CT. Right posterolateral thoracotomy, mediastinal abscess drainage and necrotic tissue debridement were made. The patient who was brought to the ICU intubated was administered with midazolam 5 mg/h and remifentanil 0.3 mg/h infusions with the aim of sedation. He was connected to the mechanical ventilator by the parameters in the mode of SIMV-PC: FiO₂: 50%, PI: 10 cmH₂O, f: 12/min, PEEP: 5 cmH₂O, PS: 10 cmH₂O. Due to his low arterial blood pressure, dopamine 10 µg/kg/min ve noradrenaline 2 µg/kg/min were infused as inotropic agents. In his blood biochemical sample, the values were CRP: 202 mg/dL, WBC: 23.27 10/mm³ while other laboratory values were in the normal level. Clindamisin (1.2 gr/day) was added to the antibiotherapy which used to be planned as meropenem (3 gr/day), vancomycin (2 gr/day) and metranidazol (2 gr/day). No pathologic agent reproduced in his culture samples.

Sedation and inotropic support were closed in the 4th postoperative day. After a follow-up in the mode of continuous positive airway pressure (CPAP), he was taken to the T tube for 30 minutes. His arterial blood gas controls were normal, thus he was extubated by applying the leakage test. His treatment continued with 2 L/min of oxygen with nasal cannula. Place, time and space orientations were assessed completely. His arterial blood gas and biochemical samples were normal and his control thorax CT and vital findings were stable; he was transferred again to the thoracic surgery clinic in the postoperative 5th day with full recovery.

DISCUSSION:

DNM is a serious disease that may result in empyema, pleural and pericardial effusion, pericarditis, intrathoracic hemorrhage and cardiac tamponade due to acute polymicrobial infections in mediastinum tissue.⁸⁻⁹

It is hard to provide an early diagnosis to the patient having mediastinitis. Although some cases have exhibited a slower clinical course, others have had an intense and fast clinical table that may lead to death.¹⁰ The first findings were retrosternal pain, swelling in the neck, redness, toughness, skin crepitation.³ In both of the cases that we followed, there were infection, redness and swelling in the neck.

In the patients having retropharyngeal abscess, if there is fever resistance to the antibiotherapy, it should be thought that it may be mediastinal abscess. If early diagnosis is not provided, mediastinitis and sepsis may develop within the first 48 hours.¹¹ The height of the C-reactive protein (CRP) and WBC in the blood biochemical sample were important.^{2,12} Both of our cases had high CRP and WBC.

Patients having respiratory insufficiency should start early intubation because of the risk of epiglottitis oedema. In order to lower the risk of massive oedema and tracheal injury, tracheostomy is recommended to be planned in the early period.⁹ In our cases, tracheostomy was not needed. Extubation in the early period was planned. In both cases, non-invasive mechanical ventilation sporadically was preferred. We did not observe any airway complications.

Since the infection exhibits prevalence in all patients having neck and cervical infections, cervical and thorax CT is useful for early diagnosis. In CT, extension in the retropharyngeal area, liquid collection in the neck and mediastinal region and free air image, transposition of the tracheal air column to the anterior and loss of cervical lordosis are traces of mediastinitis.⁷ In both of the cases, thorax CT and neck USG are used for diagnosis.

Descending necrotising mediastinitis has a high mortality rate of 30-50%. Marty-Ane et al.¹¹ reports that mortality can be decreased with aggressive surgical treatment. The survival rate is reported to be 83% in the studies.

Bad prognostic factors include late diagnosis, insufficient medical and surgical drainage treatment. Planning of the antibiotherapy according to the reproduction results in the culture is the most proper way while it is necessary in the empirical treatment to start wide-spectrum antibiotherapy.⁹ We started wide-spectrum antibiotherapy in both of our cases and planned the remaining part of the treatment according to the culture results. Sometimes wide spectrum antibiotherapy or mediastinal drainage treatment alone may not be sufficient. The gold standard for treatment is necrotic tissue drainage and debridement.¹² In both of cases, thoracotomy, mediastinal drainage and necrotic tissue debridement were made.

Consequently, if the DNM diagnosis is made early and properly, wide-spectrum antibiotherapy, hemodynamic monitoring, oxygenation, invasive/non-invasive mechanical ventilation and fast surgical approach decisions are applied, the mortality rate decreases and survival rate increases.

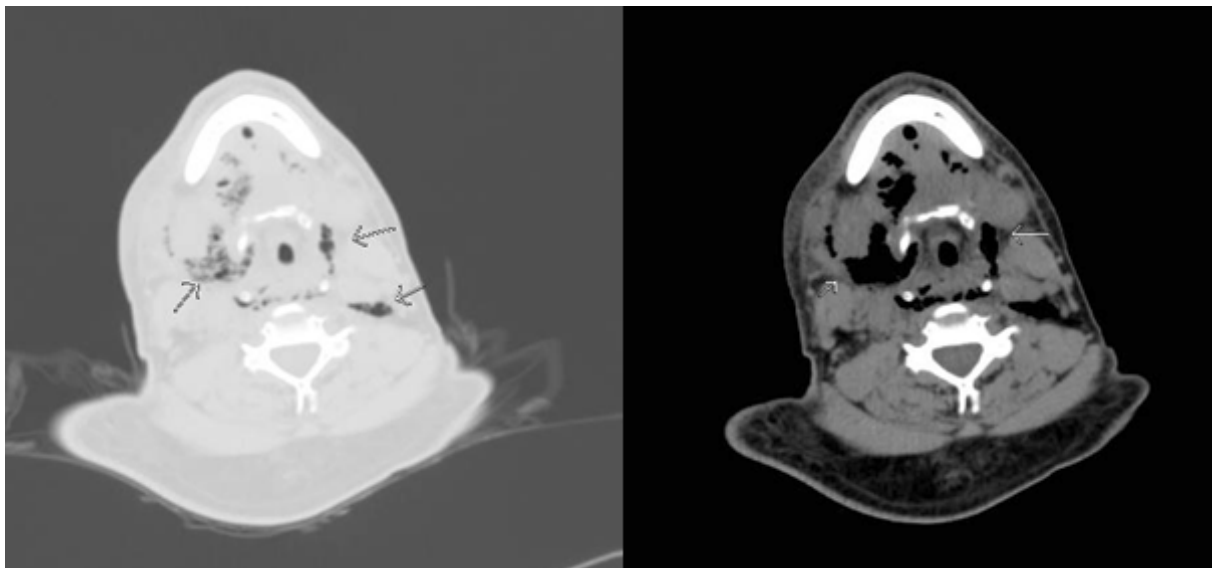


Figure 1: CT of the neck and cervical region.

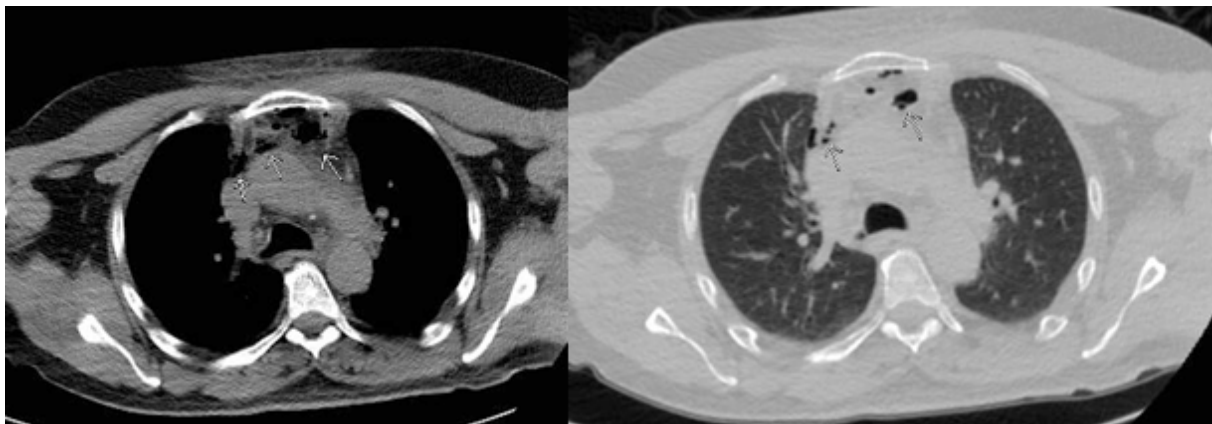


Figure 2: CT of the thorax.

REFERENCES

1. AK Özlem. Mediastinitis. Klinik Journal 2012; 25(1): 2-5 | 2. Kilic D, Findikcioglu A, Ates U, Hekimoglu K, Hatipoglu A. Management of descending mediastinal infections with an unusual cause; A report of 3 cases. Ann Thorac Cardiovasc Surg 2010; 16: 198-202 | 3. Eren , Avci A, ehito ullan A, Eren C. Descending necrotising mediastinitis: case report. J Clin Exp Invest 2013; 1(3): 228-31 | 4. Zachariades N, Mezitis M, Stavrinidis P, Konsolaki-Agouridaki E. Mediastinitis, thoracic empyema, and pericarditis as complications of a dental abscess: report of a case. J Oral Maxillofac Surg 1988; 46(6): 493-5 | 5. Marty-Ané CH, Berthet JP, Alric P, Pegis JD, Rouvière P, Mary H. Management of descending necrotizing mediastinitis: an aggressive treatment for an aggressive disease. Ann Thorac Surg 1999; 68(1): 212-7 | 6. Rupp ME, Mandell GL, Bennett JE, Dolin R, eds. Mandell, Douglas and Bennet's Principles Practice of Infectious Diseases. 7th ed. Philadelphia: Churchill Livingstone, 2005: 1070-8 | 7. Estrera AS, Landay MJ, Grisham JM, Sinn DP, Platt MR. Descending necrotizing mediastinitis. Surg Gynecol Obstet 1983; 157(6): 545-52 | 8. Van Natta LT, Paterson AG, Pearson GF, et al. Acute necrotizing mediastinitis. Pearson's Thoracic and Esophageal Surgery 3rd ed. Edition. Elsevier Inc; 2008: 1521-33 | 9. Yetim TD, Okuyucu , Ako lu SG, et al. Descending necrotizing mediastinitis: A rare case. J Kartal TRH 2012; 23(1): 45-48 | 10. Sponholz C, Sakr Y, Reinhart K, Brunkhorst F. Diagnostic value and prognostic implications of serum procalcitonin after cardiac surgery: a systematic review of the literature. Crit Care 2006; 10(5): R145 | 11. Marty-Ané CH, Alauzen M, Alric P et al. Descending necrotizing mediastinitis. Advantage of mediastinal drainage with thoracotomy. J Thorac Cardiovasc Surg. 1994; 107: 55-61 | 12. Gortlitz M, Grabenwoeger M, Meinhart J, et al. Descending necrotizing mediastinitis treated with rapid sternotomy followed by vacuum-assisted therapy. Ann Thorac Surg 2007; 83(2): 393-6 |