Fatal descending necrotising mediastinitis

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Descending necrotising mediastinitis rarely develops and this variety of mediastinitis is a highly lethal disease. A case is reported of descending necrotising mediastinitis caused by an odontogenic infection. The importance is emphasised of prompt diagnosis and aggressive surgical mediastinal drainage for the survival of these patients. Most acute mediastinal infections result from oesophageal perforation, either secondary to oesophagoscopy or tumour erosion. Mediastinitis occasionally develops as descending necrotising mediastinitis originating from the complications of cervical or odontogenic infections. Descending necrotising mediastinitis usually has a fulminant course, leading commonly to sepsis and death.

A 16 year old woman presented to our accident and emergency (A&E) department complaining of dyspnea, fever, pleuritic chest pain, and swollen throat. Her initial complaints had been a right sided premolar toothache and swollen cheek. Initially she had been treated as an outpatient with parenteral antibiotics for five days in a local medical facility. Upon worsening of her condition (orthopnea, high fever, chest pain), the patient was brought by ambulance to our A&E department.

On admission, blood pressure was 110/70 mm Hg, pulse rate was 110 beat/min, respiration rate was 30 breath/min, and her body temperature was 38.9℃. She appeared to be sick and orthophneic. Physical examination revealed dyspnea, orthopnea, diffuse painless cervical swelling, and decreased breath sounds on the right side of the chest.

Laboratory tests were as follows: white blood cells 17.600/mm^3, haemoglobin 9.6 g/dl, platelets 481000/mm^3. Blood glucose, urea, sodium, potassium, creatinine, and liver function tests were in the normal range.

Chest roentgenogram showed a pneumomediastinum and pneumohydrothorax on the right side. Computed tomography (CT) of the neck and the chest revealed gas bubbles in the neck region, marked inflammation in the mediastinal soft tissues and azygo-oesophageal recessus suggesting a mediastinal abscess and a pleural effusion and pneumothorax on the right side were noted.

In the A&E department, 1300 ml empyema was drained from the right hemithorax via a chest tube. She was then taken to the operating room and her mediastinal abscess was drained. Intravenous triple antibiotic treatment (ceftazidime 2×1 g IV, amikacin 1×1 g IV, and metronidazole 3×500 mg IV) was started. The following day, repeat neck CT was done and revealed a submandibuler abscess, which was also drained. Bacteriological results from samples obtained from the neck, pleura, mediastinum, and blood did not reveal any micro-organism. On hospital day five, another cervicothoracic CT was done and revealed residual pleural empyema and effusion around the major vessels, gas in the soft tissues of the neck and the mediastinum, and a pneumonic infiltration in the posterior lobe of the left lung.

Although she continued to receive intravenous antibiotics, she remained febrile for nine days. At this time her general condition and arterial blood gas pressures deteriorated. She was in significant respiratory distress and she, once again, needed endotracheal intubation. But this was not possible because of massive laryngeal oedema. A tracheostomy was attempted but meanwhile she developed cardiorespiratory arrest. CPR was not successful.

DISCUSSION

DNM is an uncommon complication of oral and pharyngeal infections,[1–3] spreading caudally through the retropharyngeal space, which is limited by the middle and deep layers of the deep cervical fascia.[4]

The criteria for the diagnosis of DNM were accurately defined by Estrera and colleagues.[5] These criteria include: 1—clinical manifestation of severe oropharyngeal infection (odontogenic, peritonsillar, or retropharyngeal abscesses, Ludwig's angina, or infection secondary to traumatic pharyngeal perforations); 2—demonstration of characteristic roentgenographic features of mediastinitis; 3—documentation of mediastinitis at operation or postmortem examination, or both; and 4—establishment of relation between oropharyngeal infection and development of the necrotising process in the mediastinum.[1 3 5 6] Our patient met all these criteria.

Mediastinitis occurring secondary to descending odontogenic or deep cervical infections is polymicrobial and mixed, caused by a combination of aerobic and anaerobic organisms reflecting the oropharyngeal flora.[4 6 7 8] Review of the literature shows that although DNM is quite rare, this variety of mediastinitis is a highly lethal disease. The mortality rate of this disease is between 40% and 50%.[1 3 4] Marty-Ane and associates reported that aggressive surgery provides improved results with an 83% survival rate.[1 3 4]

Delayed diagnosis and inadequate drainage are the main causes of the high mortality rate in DNM.[5 7 9] Because of the absence of early clinical or radiological signs, diagnosis is usually delayed. The diagnosis of acute mediastinitis from conventional radiological studies may be difficult. CT is an extremely useful tool in the treatment of cervical infections and the diagnosis of mediastinitis.[1 3 7 10] CT is also extremely useful in the postoperative assessment of the results of surgical drainage and the timing of the re-operations in patients with continued sepsis.[1 3 5 7]

The mainstay of treatment in patients with DNM is aggressive and adequate surgical drainage of the cervical and mediastinal collections and intravenous broad spectrum antibiotic treatment.[1 3 4 11]

Mediastinitis secondary to descending odontogenic and deep cervical infections is rare. It should be suspected in patients whose conditions are disproportionately severe to be justified by a trivial pharyngeal or odontogenic infection, or conversely, in whom a mediastinitis develops in the absence of known risk factors, such as previous surgical and diagnostic procedures involving the oesophagus or the upper airways or pharyngeal foreign bodies. At present, CT is a

valuable tool for early diagnosis of DNM in all patients with deep neck infections to determine the presence of mediastinal involvement.

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REFERENCES

1 **Marty-Ane CH**, Berthet JP, Alric P, *et al*. Management of descending necrotizing mediastinitis: An aggressive treatment for an aggressive disease. *Ann Thorac Surg* 1999;**68**:212–17.
2 **Brunelli A**, Sabbatini A, Catalini G, *et al*. Descending necrotizing mediastinitis. Surgical drainage and tracheostomy. *Arch Otolaryngol Head Neck Surg* 1996;**122**:1326–9.
3 **Wheatley MJ**, Stirling MC, Kirsh MM, *et al*. Descending necrotizing mediastinitis: transcervical drainage is not enough. *Ann Thorac Surg* 1990;**49**:780–4.
4 **Marty-Ane CH**, Alauzen M, Alric P, *et al*. Descending necrotizing mediastinitis. Advantage of mediastinal drainage with thoracotomy. *J Thorac Cardiovasc Surg* 1994;**107**:55–61.
5 **Estrera AS**, Landay MJ, Grisham JM, *et al*. Descending necrotizing mediastinitis. *Surg Gynecol Obstet* 1983;**157**:545–52.
6 **Berlot G**, Tomasini A, Cioffi V, *et al*. Fatal streptococcus viridans descending mediastinitis: case report and review of the literature. *Eur J Emerg Med* 1997;**4**:111–14.
7 **Levine TM**, Wurster CF, Kresp YP. Mediastinitis occurring as a complication of odontogenic infections. *Laryngoscope* 1986;**96**:747–50.
8 **Moncada R**, Warpeha R, Pickleman J, *et al*. Mediastinitis from odontogenic and deep cervical infection. Anatomic pathways of propagation. *Chest* 1978;**73**:497–500.
9 **Morey-Mas M**, Caubet-Biayna J, Iriarte-Ortabe JI. Mediastinitis as a rare complication of an odontogenic infection. Report of a case. *Acta Stomatol Belg* 1996;**93**:125–8.
10 **Kim HJ**, Park ED, Kim JH, *et al*. Odontogenic versus nonodontogenic deep neck space infections: CT manifestations. *J Comput Assist Tomogr* 1997;**21**:202–8.
11 **Payne WS**, Larson RH. Acute mediastinitis. *Surg Clin North Am* 1969;**49**:999–1009.

Troponin testing: beware pulmonary embolus

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Serum troponin estimation is widely used in the diagnosis and management of coronary syndromes, but it is possible to be misled by a positive result unless it is put carefully into clinical context. The serum troponin can be positive in pulmonary embolus and carries prognostic significance. A case report is presented and a review of the relevant literature.

Troponin assays have had a considerable impact in the diagnosis and risk stratification of patients presenting with an acute coronary syndrome. A correctly timed troponin assay has 95%–100% sensitivity and 85%–90% specificity for myocardial damage.[1–4] However, there are many other causes of a positive troponin test that should be remembered when assessing these patients in order to avoid misdiagnosis.

CASE REPORT

A 77 year old woman was admitted to hospital with an exacerbation of chronic obstructive pulmonary disease. She had a background of controlled hypertension, paroxysmal atrial fibrillation, and had been a heavy smoker for 30 years. She responded well to initial treatment with antibiotics, bronchodilators, and corticosteroids.

Five days after her admission, she deteriorated acutely with tight central chest pain and dyspnoea. On examination, she was tachycardic (rate 110, sinus rhythm), hypotensive (BP 90/50), but heart sounds were normal and the jugular venous pressure was not elevated. Her ECG showed ST segment depression and T-wave inversion antero-laterally, suggestive of myocardial ischaemia. The chest radiograph showed hyperinflated lung fields but was otherwise unremarkable; her peripheral oxygen saturations were 85%. She was treated with diamorphine, low molecular weight heparin, and aspirin. There was a small rise in creatine kinase (52 to 209 IU/l (normal range 24–195 IU/l)) and the troponin T 12 hours after the pain was significantly raised at 2.1 μg/l (normal <0.01 μg/l), consistent with significant myocardial damage. Two days later she was still limited by significant dyspnoea and hypoxia (po_2 8.3 on air) and the diagnosis of pulmonary embolus was suspected. An echocardiogram was normal in particular demonstrating normal left and right ventricular dimensions and no regional wall motion abnormalities. There was no significant tricuspid regurgitation to permit an estimation of the pulmonary artery pressure. A D-dimer assay was strongly positive at 3872 ng/ml (normal <500 ng/ml) and a ventilation-perfusion scan showed multiple unmatched defects, indicating a high probability of pulmonary embolus.

Joint European and American guidelines on the diagnosis of myocardial infarction[8]

- A typical rise and fall in troponin or CK-MB
- Plus at least one of the following:
  - ischaemic symptoms
  - development of pathological Q waves on the ECG
  - ECG changes suggestive of ischaemia—ST elevation or ST depression
- Coronary artery intervention, for example, coronary angioplasty

DISCUSSION

An increased serum troponin concentration is found not only in acute coronary syndromes but in several other conditions: myocarditis, stroke, myocardial trauma, post-CABG, chronic renal failure, ischaemic heart failure, ischaemic arrhythmia,