Mediastinal Abscess Resulting from Dental Infection

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SUMMARY: A case of dental sepsis is presented which resulted in a deep neck infection and eventually caused a mediastinal abscess. The microbiology of such a mixed infection is briefly discussed with emphasis on the synergistic activity of the organisms. The anatomy of the fascial spaces is also described to draw attention to the possible routes of spread of this potentially life threatening condition.

Introduction

That dental infections can cause life threatening complications is well accepted. However, because of greater public awareness of oral hygiene requirements and the use of antibiotics, most of these infections are prevented or aborted before they can spread into the fascial spaces of the neck and beyond. Other potential sources of deep neck infection are infected salivary glands, tonsils, neoplasms, fractures of the mandible and penetrating injuries to the floor of the mouth.

Ludwig's angina is one complication which can result from periapical infection of a tooth. It is characterised by a massive, brawny cellulitis occurring bilaterally in the submandibular region and also involving the sublingual tissues. However, if the sepsis extends into the parapharyngeal and retropharyngeal spaces it is then able to track into the superior mediastinum and the posterior mediastinum as far as the diaphragm(1). Other possible life threatening complications of deep neck infection are cavernous sinus thrombosis and meningitis, as a result of carotid sheath and pterygopalatine space involvement(2).

The most common signs and symptoms of deep neck infection are pain, swelling, dysphagia, dyspnoea, regurgitation and possible nuchal rigidity. If the precursor infection involved the submandibular region there could be, in addition, a board like swelling of the neck and floor of the mouth with elevation of the tongue. Spread to the mediastinum causes chest pain, severe dyspnoea, fever with X-ray evidence of mediastinal widening. Other possible sequelae of these infections include haemorrhage, rupture of the airway with aspiration, laryngeal spasm, bronchial erosion, septicaemia, metastatic abscesses, thrombosis of the jugular vein, empyema and pericarditis. In order to prevent the unremitting advance of such an infection vigorous treatment must be instituted at the earliest opportunity if a fatal outcome is to be avoided.

Case Report

A 67 year old caucasian male presented to the Accident and Emergency Department on the 23rd June 1989 complaining of pain and swelling in the submandibular region. He was an American citizen visiting London and had developed, 48 hours prior to presentation, a sore throat and mouth, with external

swelling, for which he had taken self-prescribed oral phenoxymethyl penicillin. By the time he was admitted his symptoms had become worse, he was pyrexial and had a massive swelling of the submandibular region extending intraorally (Fig 1). However, his airway was not compromised, he could swallow saliva but had difficulty drinking. Also, he was producing foul, green sputum. Other relevant findings were that he was taking disopyramide 150 mg bd following a myocardial infarction 10 years previously and that he had been a coal miner all his working life. He also smoked 20 cigarettes a day.

On referral to the Oral Surgery Department, in addition to the previous findings, it was noted that there was some elevation of the floor of his mouth but tongue

Fig 1. Patient preoperatively showing massive swelling of neck.

mobility was essentially unaffected. All his remaining teeth, three canines plus the root of the lower left canine and three upper molars, were mobile with pus around the lower canines. Radiographs revealed radiolucencies associated with the apices of most of the teeth but were much more extensive in relation to the lower canines. The patient volunteered that he had been delaying treatment recommended by his dental surgeon for many months. Specimens of the pus were sent for culture and sensitivity and he was commenced on an intravenous infusion of benzyl penicillin 600 mgs qds and metronidazole 500mgs tds.

Over the next 24 hours there was no improvement and his airway was becoming obstructed when he lay supine. Therefore, on the 25th June 1989 he was taken to the operating theatre where his remaining teeth were extracted and two incisions made on each side of his neck through which drains were inserted to the medial side of his mandible (Fig 2). All four drains produced copious amounts of thick grey pus which microscopy showed to contain gram negative bacilli and gram positive cocci. Later results of culture and sensitivity demonstrated heavy growths of *S. milleri*, α -haemolytic Streptococci (*S. viridans*) and Diphtheroids together with a light growth of coagulase negative Staphylococci all of which were sensitive to penicillin and erythromycin. He was returned from theatre, nasally

Fig 2. Patient showing drains in place.

intubated, to the Intensive Therapy Unit (ITU) for care of his airway and intensive physiotherapy. He did extremely well and was extubated on the 27th June. However, over the next 48 hours his pO_2 gradually dropped despite 50% oxygen supplementation, signs of central cyanosis appeared with reduced respiratory movement and widespread wheezes and crackles. The chest radiograph (CXR) was unchanged from admission and it was difficult to decide whether its appearance was due to the present infection or his previous occupation as a coal miner. Assay of his immunological status was within normal limits and, on the recommendation of the chest physicians, erythromycin 500mgs qds by mouth and terbutaline nebuliser 1ml qds were added to his drug regime.

On the 30th June the swelling was somewhat smaller and his respiratory status was improving. Pus had ceased from the left drains but was still flowing at a remarkable rate from those on the right. He was returned to theatre where the drainage incisions on the right were joined to produce one 10cms incision. Tissue spaces were opened from the medial side of the submandibular gland superiorly to the lateral aspect of the larynx inferiorly. Pus was present in all areas with black, necrotic tissue present around the carotid sheath, over the surface of the sterno-cleido-mastoid muscle as well as on the lateral and posterior aspects of the pharynx and larynx. The prevertebral fascia could not be identified. Debridement was carried out to remove all traces of necrotic tissue from fascial spaces as well as from the surfaces of exposed viscera. After the whole area had been thoroughly irrigated with eusol a Whitehead's vacuum pack was placed and the patient was returned to ITU. The endotracheal tube was left in place to protect his airway and breathing was spontaneous with some assistance from continuous positive airway pressure (CPAP).

Total parenteral nutrition (TPN) was now started and the intravenous antibiotics were continued awaiting the microbiological examination of the pus and necrotic tissue. The production of pus continued from the right and further debridement of the neck and paratracheal region was carried out via a second supraclavicular incision. Irrigation using eusol and dilute hydrogen peroxide was repeated on the 3rd, 5th, and 7th July. In addition, two passive suction drains were secured in place with their tips at the level of the clavicle. These allowed two hourly irrigation with eusol, one drain being used to deliver the eusol and the other to aspirate.

Despite the antibiotics and vigorous physiotherapy his respiratory function was slowly deteriorating and he was therefore commenced on intermittent positive pressure ventilation (IPPV). Organisms isolated from the pus and necrotic tissue were reported as *S. milleri*, *Proteus morgani*, *Citrobacter diversus* and *Morganella morganii*. The sputum also contained *Citrobacter diversus* as well as *Serratia marcescens*. Therefore, the antibiotic regimen was altered to benzyl penicillin 1.8g qds, metronidazole

500mgs tds, flucloxacillin 500mgs tds, cefotaxime 2g tds and tobramycin 120mgs bd, all given intravenously. Within 24 hours his respiratory function improved and he was again breathing spontaneously on CPAP. However, on the 9th July a CXR showed a widened mediastinum (Fig 3) and a Computerised Tomography (CT) scan revealed a mass in the mediastinum, at the level of the aortic arch (Fig 4), suggestive of an abscess or necrotic lymph nodes. A cardiothoracic opinion advised a right thoracotomy and drainage of the mediastinal abscess which was shown to contain *S. milleri*, Diphtheroids and Bacteroides. He had a stormy postoperative period requiring a great deal of ventilatory support to maintain satisfactory blood gases. An adrenaline infusion was also necessary for cardiovascular support. Over the next two weeks he remained relatively stable on intermittent ventilation but despite disopyramide and digoxin he still suffered frequent supraventricular tachycardias.

On the 23rd July a CXR showed a recurrence of mediastinal widening which was confirmed on a CT scan as a fluid collection. *Serratia*, sensitive to ciprofloxacin and ceftazidime, were now cultured from the blood as well as from the sputum and further cultures of early neck swabs were growing Bacteroides, sensitive to metronidazole. On review of his antibiotic therapy it was decided to substitute intravenous ciprofloxacin 200mgs bd and ceftazidime 2g bd for the flucloxacillin and tobramycin.

On the 26th July a second, right thoracotomy was performed at which a collection of pus was found in the mediastinum and chest drains were positioned to allow continuous irrigation of the site. Postoperatively he was fully sedated, ventilated and remained stable for 36 hours until the 28th July when he again presented a severely septic picture being pyrexial and jaundiced with cardiovascular instability and deteriorating gas transfer. There was very little change over the next few days with a continuous adrenaline infusion being required to maintain a blood pressure of 110/50 and a prostacycline infusion to improve oxygen delivery to the tissues.

On the 3rd August it was decided to start imipenem 1g iv qds instead of the ciprofloxacin and ceftazidime. He was given methyl prednisolone and started on a course of the anabolic steroid nandrolone decanoate. A CT scan of his head, neck and chest showed no fluid collections and this proved to be the turning point. Over the next two weeks his temperature settled, he was weaned off the adrenaline, his hepatic and renal function improved as did his level of consciousness and TPN was replaced by nasogastric feeding which he had previously failed to tolerate. He was then gradually mobilised from his bed, although still nasally intubated.

By the 21st August the neck infection had resolved sufficiently to allow a tracheostomy without risk of compromising his recovery. His respiratory drive and function continued to improve and he began eating solids with the tracheostomy cuff deflated. On the 5th

Fig 3. Chest radiograph showing mediastinal abscess 'A'.

Fig 4. CT scan of chest showing mediastinal abscess 'A', and pleural effusion 'E'.

September this tube was changed for a fenestrated tube to facilitate communication and by the 17th September, the time of his transfer to the USA, he only required oxygen supplementation at night, was afebrile with a blood pressure stable at 130/70 and a pulse of 80. All antibiotics had been stopped except for benzyl penicillin. The digoxin and disopyramide were also continued as was the terbutaline nebuliser. It is gratifying to report that since his transfer he has recovered sufficiently to continue in his previous life style.

Discussion

This patient developed the classical signs and symptoms of a retropharyngeal abscess occurring as a result of a virulent, mixed dental infection and in spite of vigorous medical and surgical therapy it spread to involve the mediastinum and necessitated twelve weeks intensive care before the patient was sufficiently well to allow transfer home to the United States.

Dental infection has been cited as the cause in 90% of cases of Ludwig's angina(3). This is a severe cellulitis

primarily affecting the submandibular space and secondarily involving the sublingual and submental spaces. The organisms most commonly incriminated are β -haemolytic *Streptococcus*, *Staphylococci*, gram negative bacilli and obligate anaerobes such as *Bacteroides melaninogenicus* and *Bacteroides oralis*. In this case the streptococci were identified as *S. milleri* which is non-haemolytic and *S. viridans* strains which are α -haemolytic. Neither of these has the invasiveness of the β -haemolytic strains as they do not produce hyaluronidase or streptokinase which destroy the intercellular matrix and the fibrin inflammatory barriers. However, the fact that gram negative bacilli were seen on microscopy but not cultured in the early stages suggests that they were likely to have been anaerobes, which are notoriously difficult culture. Indeed, later swabs and aspirates were shown to contain *Bacteroides*, which are obligate anaerobes, as well as *Proteus* and *Serratia*. Both of these are facultative anaerobes with *Serratia* being saprophytic. The significance of anaerobes in deep neck infections was highlighted by Sprinkle et al. who advised that negative cultures from cervical abscesses should arouse suspicion of anaerobic infection, particularly when they originated from dental infection(4). Therefore, it is wise to assume, when gram staining exposes gram negative bacilli, which do not appear on culture, that they are anaerobes and the inclusion of the relevant antibiotics in the initial empirical prescription is to be greatly recommended. Crogan introduced the term 'synergistic necrotizing cellulitis' for these infections where a mixture of aerobic and anaerobic organisms cause a rapidly spreading cellulitis which would be most unlikely if any one of the organisms was acting independently(5).

The spaces through which infection can spread from the submandibular space were described by Grodinsky and Holyoke(1) and re-emphasised by Levitt in 1970(6). He likened the fasciae to roads in the countryside saying: "they both separate one area from another and provide readily accessible communication routes from one area to another" and hence are important not only in the limitation of disease but also in its spread (Fig 5). They described five fascial compartments of which the first two are superficial and not relevant to this case. However, the others, which run the entire length of the neck are created by the layers of the deep cervical fascia which for practical purposes has superficial, middle and deep layers (Figs 6,7).

The superficial layer completely encircles the neck with a sheet of fibrous tissue, extending into the face superiorly and into the pectoral and axillary regions inferiorly.

The middle or visceral layer surrounds the pharynx, larynx, trachea, oesophagus, and thyroid gland as well as the infrahyoid strap muscles, extending from the base of the skull and hyoid bone to be continuous with the fibrous pericardium inferiorly.

The deep layer, like the superficial layer takes origin

Fig 5. Potential pathway of spread of infection from a dental abscess.

from the cervical spine and nuchal ligament and completely invests the pre- and postvertebral muscles. The part lying in front of the bodies of the vertebrae splits to form two layers. The prevertebral layer is adjacent to the vertebrae and is fused to the transverse processes, from the base of the skull to the coccyx. The alar layer is also fused to the transverse processes but only extends from the base of the skull to the level of the second thoracic vertebra where it fuses with the middle layer.

These layers create three potential spaces. The posterior visceral or retropharyngeal space which extends from the base of the skull to the level of the second thoracic vertebra is the posterior part of the parapharyngeal space, the rest of which is bound by the strap muscles anteriorly and the carotid sheath laterally. The posterior limit of the retropharyngeal space is the weak alar layer. The ease with which this can be breached was demonstrated by Grodinsky and Holyoke when injections they made into this space, to demarcate its limits, readily broke through into the Danger space. This only contains loose areolar tissue and extends from the base of the skull, into the posterior mediastinum and to the diaphragm inferiorly. It is the inferior spread of infection along this space that makes it so potentially dangerous and clinically important.

The last of these spaces is the prevertebral space and lies between the prevertebral layer and the bodies of the vertebrae along the whole length of the spinal column. Acute infections in this space are uncommon and do not usually involve the mediastinum. However it is possible and was seen more commonly in pre-antibiotic days, for posterior neck infections to track along this space and point as far down as the fascial sheath of the psoas major.

The case reported exemplifies the dangerous and

potentially fatal nature of deep neck infections and the resistance that the micro-organisms can demonstrate to modern antibiotics even when used in very high dosages and coupled with intensive care therapy. It can be argued that this patient's defences against infection were depressed but haematological examination and immune system assay failed to show any evidence of it. Bearing in mind the previous description of the fascial planes it is evident that the alar layer was breached; there was no evidence of it at operation, except for the black necrotic tissue, and neither was the prevertebral layer definitely identified. This would place the phrenic nerve, which normally lies just under the prevertebral layer, in very close proximity to the pus and necrotic tissue and must have played a part in his poor respiratory function. Additionally, the myocardial infarct which he had suffered ten years previously, and resulted in him taking the anti-arrhythmic drug disopyramide, undoubtedly had an adverse effect upon his ability to maintain adequate cardiovascular function in the presence of an overwhelming infection. The degree of inotropic support that he required may not have been only due to the underlying heart condition but also to the fact that disopyramide itself impairs cardiac contractility. Nevertheless, even with his reduced ability to cope with

Fig 6. Diagrammatic sagittal section through neck to show fascial spaces.

Fig 7. Diagrammatic horizontal section through neck at level of thyroid isthmus.

the effects of the infection, its spread might have been significantly reduced if he had sought advice at its onset. Bacteroides and other gram negative bacilli play a significant role in this type of infection and, as Willis and Vernon have pointed out, some strains of Bacteroides which were previously sensitive to phenoxymethyl penicillin are no longer so because of the production of β -lactamase(7). Therefore, had he been treated with the standard antibiotic regime of metronidazole together with one of the penicillins at the onset of his symptoms, instead of only the self-prescribed phenoxymethyl penicillin which was probably taken in a haphazard manner, the advance and severity of the infection may well have been curtailed; the gram negative and anaerobic bacilli could have been controlled thus preventing the synergism which typifies this condition.

REFERENCES

1. GRODINSKY M, HOLYOKE E. The fasciae and fascial spaces of the head and neck and adjacent regions. *Am J Anat* 1938; **63**:367.
2. SHAFER W G, HINE N K, LEVY B N. Textbook of oral pathology. 4th Ed. Philadelphia and London: Saunders.
3. TOPAZIAN R G, GOLDBERG M H. Management of infections of the oral and maxillofacial regions. Philadelphia and London: Saunders.
4. SPRINKLE P M, VELTRI R W, KANTOR L M. Abscesses of the head and neck. *Laryngoscope* 1971; **81**: 403.
5. CROGAN I C. Necrotizing mediastinitis secondary to descending cervical cellulitis. *Oral Surg* 1973; **36**:307.
6. LEVITT G W. Cervical fascia and deep neck infections. *Laryngoscope* 1970; **80**: 409.
7. WILLIS P I, VERNON R P. Complications of space infections of the head and neck. *Laryngoscope* 1981; **91**:1129.