

**A RETROSPECTIVE STUDY OF DESCENDING NECROTIZING
MEDIASITINIS OF ODONTOGENIC ORIGIN.**

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Witwatersrand, Johannesburg, in partial fulfilment of the requirements for the degree
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1. DECLARATION

I, Elias Tinyiko Mabaso, declare that this research report is my own work. It is being submitted for the degree of Master of Dentistry in the branch of Maxillofacial and Oral Surgery at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other University.

.....

Date..... Place.....

2. ABSTRACT

This retrospective study evaluated the causative factors, treatment, complications and management outcomes of patients with Descending Necrotising Mediastinitis (DNM) of odontogenic origin treated at Charlotte Maxeke and Chris Hani Baragwanath Academic Hospitals for comparison with other published international studies.

It is based on the management of 11 patients with DNM treated over a period of 3 years (2007 to 2010). Five patients had tracheostomies, 10 patients had postero-lateral thoracotomies and 1 patient had a Chamberlain procedure. One patient died of septic shock giving a mortality rate of 9.10%. Future studies are needed to monitor this trend. The management of DNM in this study is comparable with that reported by other clinicians.

3. ACKNOWLEDGEMENTS

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4. INTRODUCTION

The majority of acute mediastinal infections result from oesophageal perforation or following trans-sternal cardiothoracic surgical procedures.(1) Occasionally, acute mediastinitis follows oropharyngeal abscesses with severe cervical infection spreading along the deep fascial planes into the mediastinum. This is a particularly virulent form of mediastinal infection, described as Descending Necrotizing Mediastinitis (DNM).

The criteria for diagnosis of DNM were defined by Estrera *et al* (1);

1. Clinical signs and symptoms of severe neck infection
2. Typical radiologic signs including fluid and/or gas collection on chest CT scans
3. Intraoperative or post-mortem documentation of mediastinal infection
4. Continuity between the oropharyngeal infection and the mediastinal infection.

The most common primary infection preceding Descending Necrotizing Mediastinitis is odontogenic (Wheatley *et al* reported 25 of 43 cases).(2) Infections originating in the fascial planes of the head and neck spread downward into the mediastinum along the cervical fascia, supposedly facilitated by gravity, breathing and negative intrathoracic pressure.(3)

The anatomic proximity of the cervical fasciae, consisting of the superficial fascia and the three layers of the deep cervical fascia (superficial, visceral and prevertebral) provides a conduit for the spread of infection from the neck into the mediastinum.

These layers partition the neck into several potential spaces (pretracheal space, retropharyngeal space, perivascular space, and parapharyngeal space) that can all serve as portals of entry into the mediastinum.(4) According to Brunelli *et al* (5) and Mibos *et al*(6)

the retrovisceral space is affected in 70%, the perivascular compartment in 21%, and the pretracheal space in 8% of cases. The most common anatomic pathway is the lateral pharyngeal space through the retrovisceral space, inferiorly into the mediastinum.(5)

The microbiology of DNM is polymicrobial reflecting the indigenous microflora of the oral cavity. The organisms most commonly implicated include Prevotella, Peptostreptococcus, Fusobacterium, Veillonella, Actinomyces and oral Streptococcus,(6) There is an anaerobic predominance.

Surgical management, (particularly the optimal form of mediastinal drainage), remains controversial with advocates of cervical drainage only, or cervical drainage and routine thoracotomy.(5)

With early clinical diagnosis, use of Computed Tomography, airway management, adequate drainage of the abscess, appropriate use of intravenous antibiotics and postoperative intensive care unit management, there has been a reduction in mortality rates.

Mortality rates have dropped to 20% - 40% in the antibiotic era, as opposed to more than 60% in the pre-antibiotic era. Delayed recognition and insufficient drainage have been recognised as the primary causes of high mortality rates.(7) Hyperbaric oxygen therapy can be beneficial, but the logistic challenges can be prohibitive in the acute phase of the disease.(8)

5. LITERATURE REVIEW

5.1 Risk factors

Although the dogma is that risk factors for DNM include poor dental hygiene, diabetes, I.V drug abuse, excessive alcohol intake, AIDS and recent steroid use,(9) the reported experience is that co-morbidities are often not found and thus their role in DNM is doubtful.

5.2 Microbiology

Both the aerobic and anaerobic bacteria found in the oral flora are the predominant microorganisms isolated from DNM patients.(8, 10) The most common aerobic bacteria include alpha haemolytic Streptococcus, Staphylococcus Aureus and Klebsiella pneumonia.(11) The most common anaerobic bacteria include Peptostreptococcus, Bacteroides Fragilis, Prevotella and Porphyromonas.(6) Less commonly described organisms are Streptococcus Viridans, Serratia Marcescens, Enterobacter, Neisseria, Fusobacterium, Pseudomonas Aeruginosa, Escherichia Coli (9), Stenotrophomonas (12) and Veillonella (13)

5.3 Antibiotic Therapy

Antibiotic therapy should be started immediately and be broad enough to cover Gram-positive cocci, Gram-negative rods and anaerobic bacteria. First choice empirical regimens include Piperacillin–Tazobactam and Vancomycin, Clindamycin with either Ceftriaxone or ceftazidime, or in penicillin-allergic patients, a quinolone plus Clindamycin.(14) This is modified subject to clinical procedure and culture and sensitivity tests.

5.4 Radiographic Investigations

Conventional radiographs may demonstrate retropharyngeal soft tissue swelling, widening of the superior mediastinal shadow, gas bubbles in the soft tissues of the neck, pneumomediastinum or air–fluid levels in the prevertebral space.(15) However a retrospective analysis of a series of six patients with DNM found the conventional radiographic finding of superior-mediastinal widening in only four cases and pneumomediastinum in only three.(16) Computed Tomography(CT) imaging to visualise soft tissue and potential spaces in the neck and chest is typically obtained 35–60 seconds following I.V administration of an iodine-based contrast medium(17), with contiguous 3 to 5mm sections to include anatomy from the skull base to the dome of the diaphragm. CT evidence of mediastinitis includes localised mediastinal fluid collections or abscesses, gas bubbles in the mediastinal soft tissues, pleural or pericardial effusions, venous thrombosis and lymphadenopathy.(17) Diffuse mediastinitis may be seen, characterised by increased density of the mediastinal fat with resultant loss of definition of normal fat planes.(18) Mediastinal abscess is diagnosed based on the presence of a well-defined, low-attenuation fluid collection with or without air, which may demonstrate rim enhancement following contrast administration.

It is important to note that some CT signs suggestive of mediastinitis can be seen in asymptomatic patients up to 21 days following median sternotomy.(19) Thus, correlation between clinical and radiological findings is imperative in these patients.

CT findings of mediastinal fluid or air collections have a reported sensitivity of 100% for detection of post-sternotomy mediastinitis (19), and specificity ranging from 33% in the immediate post-operative period to 100% after 15 days post-operative. CT findings in the neck may vary depending on the nature of the infectious focus. Retropharyngeal or

odontogenic abscesses may be confirmed on CT. Cervical CT findings supporting infection include jugular venous thrombosis, lymphadenopathy and abnormal fluid collections.(17)

CT may also assist in diagnosing or excluding necrotising fasciitis. Thickening or enhancement of the sternocleidomastoid muscle and the presence of fluid collections in multiple anatomic compartments of the neck are sensitive features of cervical necrotising fasciitis, demonstrated in all patients in one retrospective analysis of 14 surgically proven cases of necrotising fasciitis of the head and neck.(20) In a series of 34 patients with suspected DNM,(17) the absence of suspicious CT signs of infection in the neck or chest spaces excludes DNM with a sensitivity of 100% based on prediction of a benign non-operative clinical course. However, absence of intrathoracic infection by Computed Tomography does not necessarily ensure a favourable prognosis if concomitant neck infection is present, as two out of 12 patients without initial evidence of intrathoracic infection in the same study ultimately died from subsequent mediastinal spread when conservatively managed with cervical drainage and antibiotics alone.(17)

In addition to confirming a diagnosis of DNM and detecting complications, CT assessment of the extent of infection can aid in determining optimal management and can assist in surgical planning and in assessing response to therapy.(7)

Follow-up CT examinations of treated DNM patients revealed unanticipated evidence of progression of disease requiring further intervention in a majority of cases.(7)

5.5 Surgical Management

Successful surgical management involves a combination of cervical and mediastinal drainage with or without open thoracotomy (1, 9, 10, 12). Endo *et al*(21) proposed a classification scheme to facilitate management of DNM based on CT assessment of the extent of infection. Type I (or localised) DNM, defined as infection localised to the upper mediastinum above the tracheal bifurcation, may be adequately managed with mediastinal drainage via a transcervical approach without opening the chest cavity.(22)

Diffuse DNM is sub-classified as type IIA if infection involves the lower anterior mediastinum or as type IIB if the anterior and posterior lower mediastinum is involved.(21) In cases of type IIA infection, cervicotomy and a subxiphoid incision or anterior mediastinotomy may provide adequate exposure for debridement.(3) For type IIB infection, there is ample support in the literature for combined cervicotomy and thoracotomy with wide debridement to evacuate all infected and necrotic tissue.(4)

When infection involves the anterior and posterior mediastinum, the mortality rate with cervicotomy alone is significantly worse.(5) A meta-analysis by Corsten *et al* (5) found that patients who received combined cervicotomy and thoracotomy had a mortality of 19% versus 47% in patients receiving cervicotomy alone, which was a statistically significant difference. When infection involves both thoracic cavities, bilateral exploration and debridement is required. This has been accomplished with bilateral video-assisted thoroscopic surgery (VATS), unilateral posterior thoracotomy with contra lateral VATS exploration, or the clamshell procedure.(23)

Debridement can be accomplished in a one-stage procedure via the clamshell approach, but may not be tolerated in critically ill patients and carries the risk of sternal osteomyelitis and phrenic nerve palsy.(3) Posterolateral thoracotomy is a well-validated approach to treating a posterior mediastinal infection (3, 10), allowing access to all mediastinal spaces, including the pericardial and pleural spaces, and placement of large-bore chest tubes for mediastinal and pleural drainage.(3) Marty-Ane *et al*(3) achieved a relatively low mortality rate of 16.5% in their series of 12 patients by aggressively utilising thoracotomy as a treatment modality. All patients that underwent both cervicotomy and thoracotomy survived. More recently, less invasive approaches successfully employing thoracoscopic or mediastinoscopic drainage have been reported. (24, 25)

5.6 Complications

These include bilateral empyema, purulent pericarditis, pneumonia and acute respiratory distress syndrome.(8) Vascular complications include internal jugular and brachiocephalic venous thrombosis and pseudoaneurysm of the carotid artery.(24) A case of suppurative rupture of the proximal vertebral artery, internal jugular vein and subclavian vein has also been reported.(25) Additional sequelae include septic shock, cardiac tamponade from pericarditis, airway obstruction, pleuromediastinal or pleuro-oesophageal fistula, and erosion into major blood vessels.(24) The oesophageal and aortic hiatuses represent potential routes of spread into the peritoneum and retro peritoneum, and a case of trans-hiatal extension of infection into the retro-peritoneum has been reported.(26)

5.7 Mortality

Despite the introduction of modern antimicrobial therapy and CT imaging, this form of mediastinitis has continued to produce mortality rates between 25% and 49%. Table 1.(27)

Table I. Cumulative mortality of DNM in the literature

	<i>No. of Reports</i>	<i>No. of Patients</i>	<i>Survivors</i>	<i>Mortality %</i>
Pearse (1938)	1	37	18	49
1970-79	8	13	9	31
1980-89	11	26	16	38
1990-98	27	57	43	25

Freeman et al J Thorac Cardiovasc Surg 2000; 119:260-267

6. AIMS AND OBJECTIVES

This is a retrospective study that seeks to identify causative factors, treatment, complications and management outcomes of patients with descending necrotizing mediastinitis of odontogenic origin treated between 01/01/2007 to 01/07/2010 at Charlotte Maxeke and Chris Hani Baragwanath Academic Hospitals for comparison with published international studies.

“The aims are to:”

Identify causative factors and co-morbidities associated with DNM

Record and analyse patient`s vital signs and blood results on admission

Record the CT scan findings

Record the medical and surgical management

Record the length of hospital stay, complications and outcome

Assess the validity of treatment protocols.

7. MATERIALS AND METHODS

The clinical records of patients with DNM secondary to odontogenic infection who were treated at Charlotte Maxeke and Chris Hani Baragwanath Academic Hospitals between June 2007 and August 2010 were reviewed.

In all cases the criteria of Estrera *et al* were fulfilled. These criteria include: (1) clinical manifestation of severe oropharyngeal infection (2) demonstration of characteristic roentgenographic features of mediastinitis (3) documentation of necrotizing mediastinal infection at operation or post-mortem examination or both and (4) establishment of relationship between oropharyngeal infection and development of necrotizing mediastinal infection.

In addition to the patient's demographic data, the following parameters were recorded: co-morbidities, length of time between development of symptoms and hospitalization, vital signs (temperature, blood pressure, respiratory rate and pulse), and blood results on admission (full blood count, urea and electrolytes).

Radiographic findings on routine chest X-ray and CT i.e as pleural effusions, widening of the mediastinum and surgical emphysema were documented.

The empirical antibiotic therapy, microbiology, culture and sensitivity were recorded as were the surgical procedure, post surgical complications, length of hospital stay and mortality.

7.1 Data analysis plan

Non-randomised, purposive sampling was used to select all patients with DNM who attended the Charlotte Maxeke and Chris Hani Baragwanath Academic Hospitals from 2007 to 2010 and who met the inclusion criteria. This sampling method was appropriate due to the low frequencies of outcome under investigation.

7.2 Data extraction and capturing

Data was extracted from the microfiche records in the hospital archives. Data was then captured in Microsoft Excel (Microsoft Corporation Redmond, Washington, USA) spreadsheet then imported to STATA version 10 for data cleaning and for analysis purposes.

7.3 Descriptive analysis

Demographic and clinical characteristics were determined for all the participants. For categorical variables such as gender, frequencies and percentages were calculated, whereas for continuous variables such as age, mean and standard deviation were computed. The results were presented on frequency tables and bar graphs. The median and interquartile ranges were used to summarise non-normal data.

8. RESULTS

8.1 Demographic data

11 patients with a diagnosis of DNM were treated of which 81.82% were males. The mean age of the study participants was 36.5 ± 32 (range 23-52).

8.2 Clinical data

The main clinical symptoms included swelling and dysphagia in 8 patients, associated with fever and malaise. Trismus was present in 7 patients. Four patients had dyspnoea associated with oropharyngeal oedema and pleural effusions. Before hospitalisation all patients had received antibiotics prescribed by the referring primary care practitioners, which had proven inadequate in controlling the infection

The mean length of time in days between the onset of sepsis or symptoms and hospital admission was 7 ± 3 days (range 2-11) (Table 2).

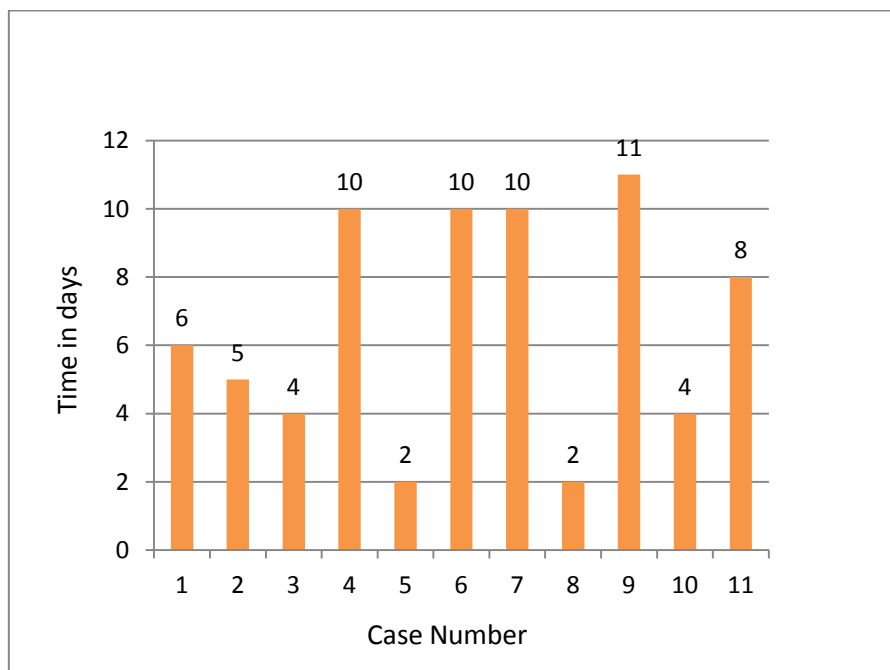


Table 2 Time of first symptom to hospitalisation in days.

The mean length of hospital stay was 44 ± 29 days (range 21- 117) (Table 3).

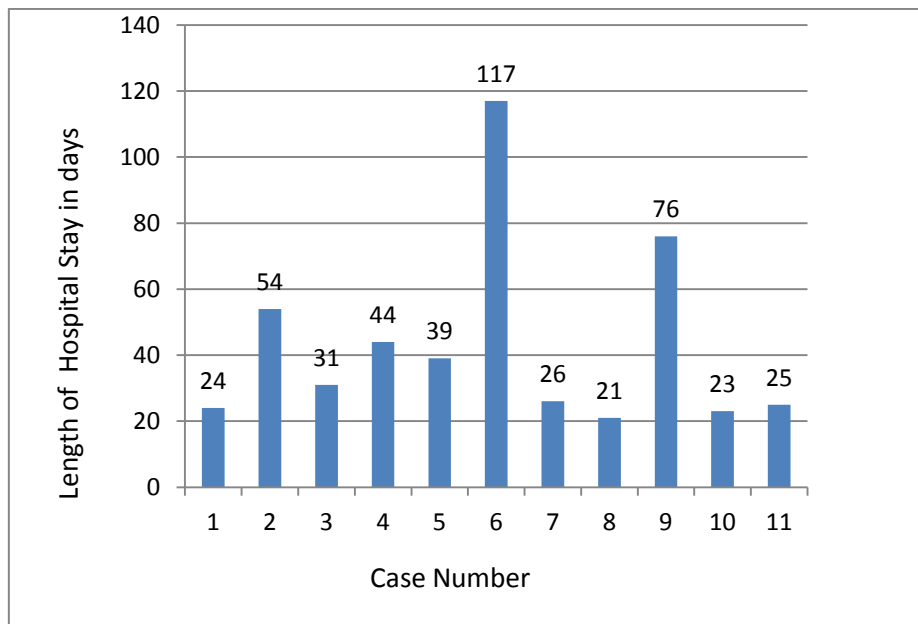


Table 3 Length of hospital stay in days.

8.3 Haematological indices

White Cell Count on admission ranged from 2 to $25.12 \times 10^9/l$ and was elevated, normal and reduced in 5, 4 and 2 cases respectively with a mean of $12.92 \times 10^9/l \pm 8.35 \times 10^9/l$ (Table 4).

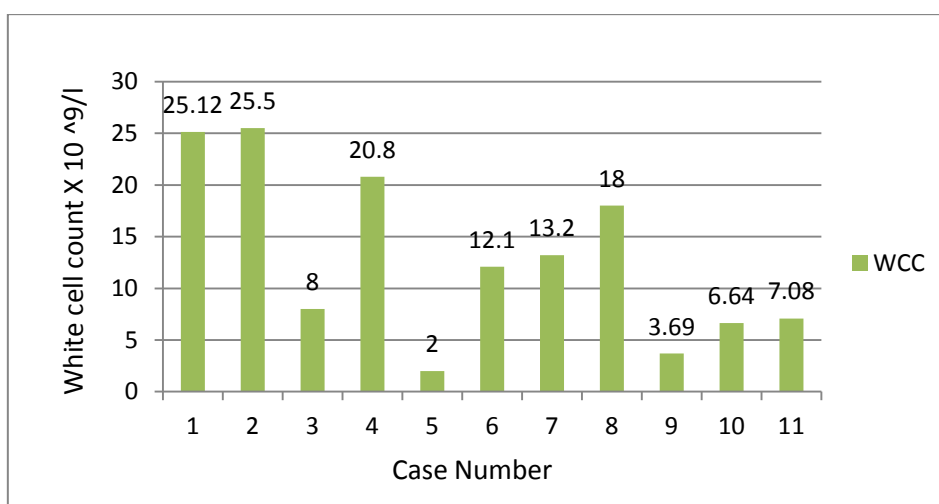


Table 4 Patient’s white cell count on admission

Haemoglobin levels on admission ranged from 9.2 to 15.2 g/dl with a mean of 12.18 g/dl \pm 2.17 g/dl (Table 5).

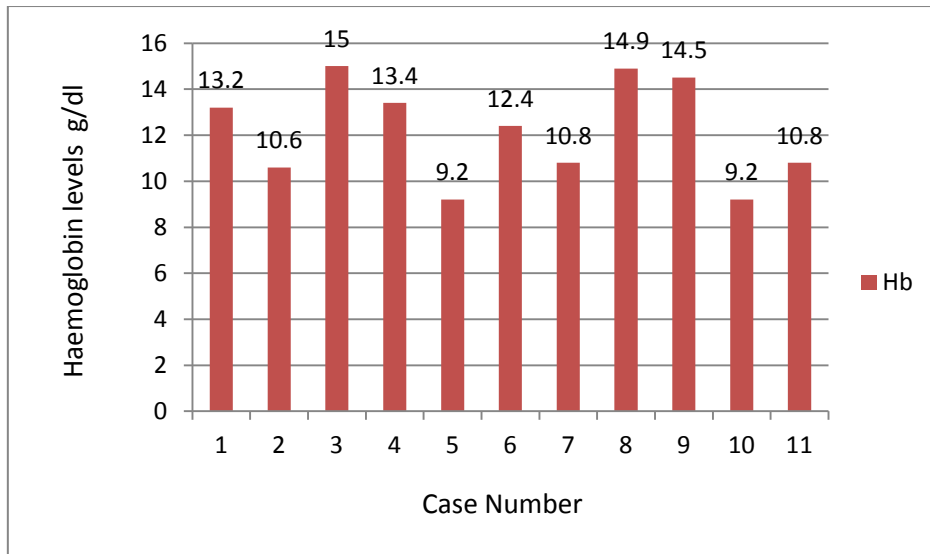


Table 5 Patient`s Haemoglobin levels on admission.

Platelet levels on admission ranged from 29 to 440 x 10⁹/l with a mean of 212.6 x 10⁹/l \pm 134.2 x 10⁹/l (Table 6).

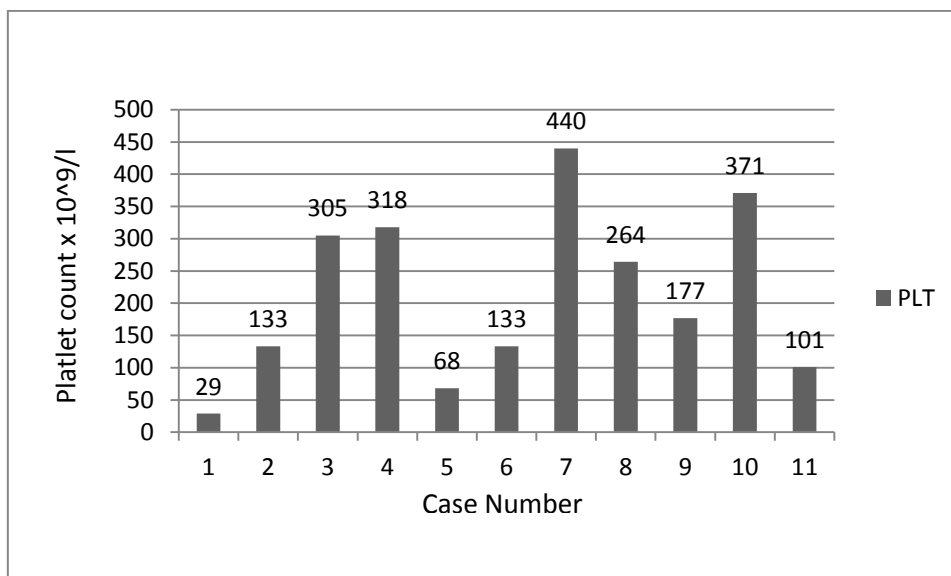


Table 6 Patient`s platelet count on admission.

8.4 Associated co-morbidity

Four patients were seropositive for Human Immunodeficiency Virus, the only co-morbid condition identified (1 was not tested) and 1 patient smoked 20 cigarettes per day.

8.5 Diagnostic investigation and radiographic studies

A clinical suspicion of DNM was followed by a chest x- ray, findings included, tracheal deviation (2 patients), pleural effusions (4 patients), widening of the mediastinum and surgical emphysema (Figure 1).

Contrast-enhanced CT was the method of choice for confirming the diagnosis, directing surgical drainage and postoperative progression.

The most common findings were swelling and infiltration of the cervical soft tissues in all patients, signs of mediastinal infection (encapsulated fluid collections or an abscess with gas formation) and in 4 patients pleural effusion (one bilateral). 5 cases had abscess formation limited to the superior mediastinum and in 7 patients both superior and inferior mediastinum were affected.

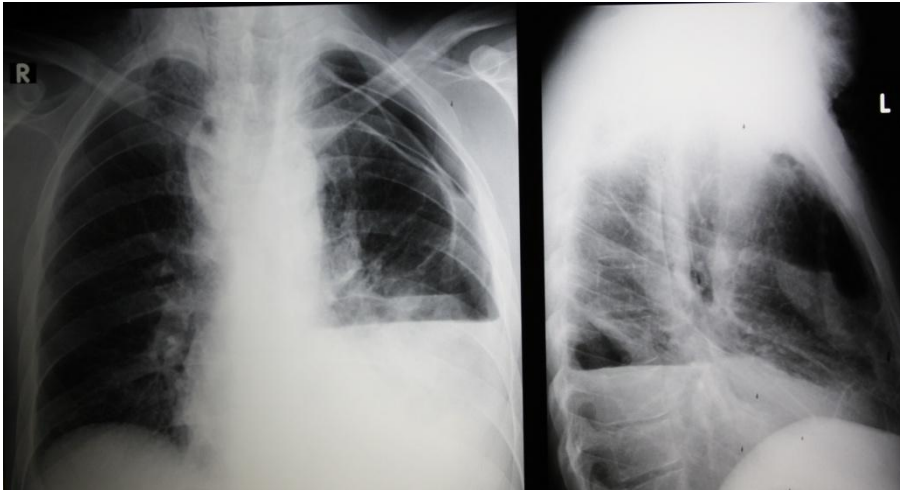


Figure 1 Anterior-posterior and lateral chest X-rays of patient number 2 showing a left pleural effusion with widening of superior mediastinum with multiple focal mottled appearance consistent with pneumomediastinitis.

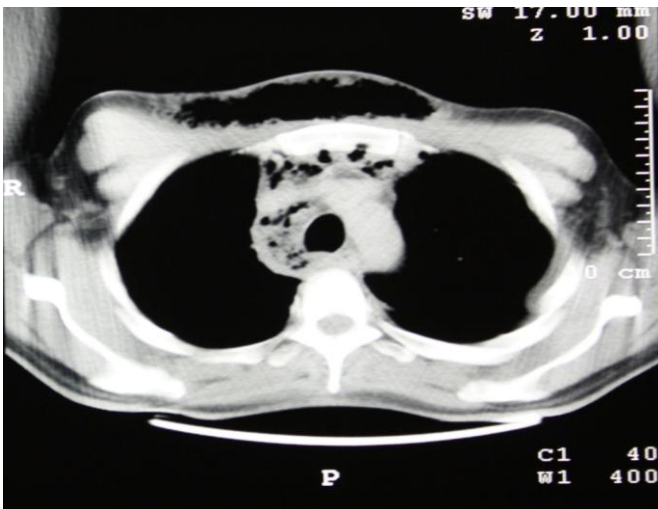


Figure 2 Post-contrast CT scan of the chest (axial view) of patient no 2 at the level of the aortic arch showing multiple air locules and rim enhancing collection with a fluid-fluid level and massive air collection in the anterior chest wall soft tissues.

All patients had postoperative CT scan for evaluation of infection progression or remission and position of drains.

8.5 Microbiology

Microscopy, Culture and Sensitivity of pus samples from the neck (patients 1 and 6), mediastinum (patients 2,5,7,9 and 10) showed aerobic bacteria including *Enterobacter* species (2), *Staphylococcus Aureus*, *Pseudomonas Aeruginosa* (2), and *Pantoea* species. Patients 2 and 10 had Pan Resistant *Actinobacter Baumannii* and were only susceptible to colistin. *Streptococcus Milleri* was cultured in 2 patients, (1 and 7), and both were susceptible to penicillin and ampicillin. Patient 7 developed Methicillin Resistant *Staphylococcus Aureus* which was susceptible to Vancomycin. In 4 patients no organisms were isolated (**Table 7**).

Table 7 SPECIMEN SITE, TYPE, CULTURE AND SENSITIVITY

Cases	Specimen and site	Bacteria Isolated	Sensitivity
1	Pus(Neck)	Streptococcus Milleri++	Penicillin/ampicillin
2	Pus(Mediastinum)	Actinobacter Baumannii Pseudomonas Aeruginosa	Colistin Tazocin
3	Pus	No bacterial growth	No records
4	Pus	No bacterial growth	No records
5	Pus(Mediastinum)	Enterobacter species	Imipenem, Meropenem and Ertapenem
6	Pus(Neck)	Staphylococcus Aureus Pseudomonas Aeruginosa	Imipenem and Meropenem
7	Pus(Mediastinum)	Streptococcus Milleri Methicillin Resistant Staphylococcus Aureus (MRSA)	Penicillin Vancomycin Linezolid
8	None	No bacterial growth	
9	Pus(Mediastinum)	Escherichia Coli Pseudomonas Aeruginosa	Ertapenem
10	Pus(Mediastinum)	Actinobacter Baumannii	Colistin
11	Pus	No bacterial growth	No records

8.6 Antimicrobial and Surgical Therapy

The empirical first line antibiotic regimen was Amoxicillin with Clavulanic acid 1.2g eight hourly and Metronidazole 500mg eight hourly intravenously. Airway protection was effected with fiber-optic intubation in one patient, tracheostomy in 5 patients (emergent) and no records of airway management were found on the other five patients. All 11 patients had drainage of the relevant floor of the mouth (submental, submandibular and sublingual spaces) and neck spaces (lateral pharyngeal and retropharyngeal), whilst mediastinal surgical drainage was effected via Chamberlain procedure in 1 patient and posterolateral thoracotomy (3 bilateral), in 10 patients. Four patients required revision surgery twice, 2 patients required 3 revision surgeries and one patient had four surgeries. Two patients needed skin graft to the anterior neck and chest wall due to sloughing obliging surgical resection.

8.7 Complications

Patient 8 developed empyema and Patient 11 bled from the internal jugular vein. Patient 9 developed septic shock, necrotizing pneumonia and meningitis. Patients 2 and 6 had anterior neck and chest soft tissue loss.

8.8 Outcomes and mortality

Ten patients were discharged and one (patient 9) died of septic shock 4 weeks after admission.

9. DISCUSSION

The incidence of DNM is quite low, and thus many of the publications are case reports or review of patient groups collected over extended periods of time. During the past 3 years, we have treated 11 patients who developed DNM. DNM, a potentially life-threatening condition, is a feared complication of severe soft tissue infections of the head and neck.

The length of time between the onset of symptoms and hospital admission was 7 ± 3 days (range 2 - 11), which is longer than that reported in other studies (mean of 2.5 days). (4)

The length of hospital stay was 44 ± 29 days (range 21 to 117) these were comparable to other studies which had a mean of 48 days (10)

The importance of the patient's general health and pre-existing pathologies have infrequently been discussed as additional morbidity and mortality factors in previous studies. (15) Diabetes mellitus and alcoholism are the most frequently cited predisposing systemic conditions for Descending Necrotizing Infections and DNM. In diabetic patients, defects of the immune system, along with vascular insufficiency, are supposed to facilitate the spread of odontogenic or pharyngeal infections to the mediastinum.(28) In this study none of the patients were diabetic, it was also difficult to prove if some of the patients were alcoholics as most of them said they drank alcohol occasionally. HIV did not affect the patient's treatment outcome as all the HIV positive patients had an unremarkable recovery. Most of the patients had no known co-morbidities, therefore the long held conviction that DNM follows cervical infections in patients with co-morbidities cannot be supported.

The majority of DNI and DNM are mixed polymicrobial aerobic and anaerobic infections reflecting its pharyngeal or odontogenic nature. The causative pathogens are mainly bacteria of the microflora of the superior aero-digestive tract that become virulent in certain conditions and may vary depending upon the infection's origin.(9, 13)

Helped by gravity, respiration and intrathoracic pressure in the mediastinum, pus in the orocervical spaces rapidly reaches the mediastinum. Deficient vascularisation and rarity of cell defences are features of cervicomediastinal spaces.(9)

In this series only aerobic organisms were cultured, *Streptococcus milleri*, *Staphylococcus Aureus*, *Pseudomonas Aeruginosa*, *Escherichia coli*, *Enterobacter species*, pan resistant *Acinetobacter Baumannii* and Methicillin Resistant *Staphylococcus Aureus*, which were comparable to other studies.(3,7)

Both cases 2 and 11 cultured pan resistant *Acinetobacter Baumannii* which was susceptible to Colistin (Polymyxin antibiotic produced by certain strains of *Bacillus Polymyxa*, effective against gram negative bacilli and it is used as a last resort antibiotic for multidrug resistant *Pseudomonas Aeruginosa* and *Acinetobacter*).

Methicillin Resistant *Staphylococcus Aureus* was cultured in patient no 7 which was sensitive to Linezolid and Vancomycin. Linezolid is an oxazolidinone antibacterial and has a broad spectrum activity against Gram-positive organisms. It is active against both Cloxacillin resistant *Staphylococci* and Vancomycin-Resistant *Enterococci*.

3 patients had cultured organisms sensitive to Carbapenems (*Meropenem* and *Ertapenem*).

In 4 patients pus samples failed to culture organisms. Similar experiences have been reported by others. (6) No anaerobes were cultured possibly due to lack of adherence to strict anaerobic culture methods.

Prompt recognition of DNM can be challenging because there may not be clear symptoms and signs. Chest pain, jugular distension, high fever and crepitation on palpation of the neck and chest have been cited as typical signs of DNM.(28)

In this study the main symptoms and signs were dysphagia, neck swelling and erythema on the anterior chest. High fever was only present in three patients, and one patient complained of chest pain and dyspnoea.

Several authors have previously highlighted the value of contrast-enhanced CT as the method of choice for establishing the diagnosis, directing the surgical drainage procedure and monitoring the postoperative progression of DNM.(8, 12) In this study all patients had preoperative CT scans to assess the level of infection and the severity of the soft tissue swelling around the airway. This contributed to the early surgical interventions by all the disciplines involved. Postoperative CT scans were only taken when there was no improvement in the general patient's condition.

The role of tracheostomy in DNM is controversial. Tracheostomy is a pathway for downward spread and subsequent mediastinal infection. In this study the tracheostomy was performed whenever airway compromise was a concern. The protection of the airway must of necessity be prioritized over the justifiable afore mentioned concerns.

A number of surgical approaches have been reported for optimal mediastinal drainage, including a transcervical approach and several transthoracic approaches such as standard postero-lateral thoracotomy, median sternotomy, subxiphoid approach, clamshell incision and image-guided thoracoscopic approaches.(10, 12, 28)

More recently, many surgeons have condemned transcervical drainage of the mediastinum and advocated more aggressive management of DNM with mandatory transthoracic

approaches regardless of the level of mediastinal involvement. These authors contend that the mediastinum cannot be adequately drained via conservative surgical access and have reported statistically significant survival advantage for patients undergoing early thoracotomy. (4, 9, 11, 12)

Midline sternotomy carries the risk of sternal osteomyelitis. Clamshell incision, although exposing the entire mediastinum and both chest cavities, is particularly invasive in these critically ill patients. Therefore, cervicotomy along with a posterolateral thoracotomy incision as a transthoracic approach is currently recommended as the standard care of DNM.(12)

In this study transthoracic drainage involved postero-lateral thoracotomies in 10 patients and one had a Chamberlain procedure also known as an anterior mediastinotomy which was first described by McNeill and Chamberlain.(29) The one patient who had a Chamberlain procedure had collections in the superior anterior mediastinum.

Of the 10 patients who had postero-lateral thoracotomies, 5 had collections extending below the *carina* or into the posterior mediastinum and 4 had collections initially in the superior mediastinum which later spread to the lower mediastinum.

Only one patient died in this study from complications of septic shock, necrotizing pneumonia and meningitis representing a 9,09% mortality.

A vexing question is why does DNM occur in a small proportion of patients who present with odontogenic infections?

Co-morbid conditions i.e diabetes mellitus, chronic steroid use, alcoholism, immune deficiency and bacterial virulence are commonly cited as contributing factors to developing DNM. None of the co-morbid conditions have been positively linked to the development of

DNM, which was confirmed by this study. For instance most of the patients in this study had no known co-morbidities. Only four patients were seropositive to HIV and all four had an uneventful recovery.

Therefore we have come to believe that DNM may be the result of either these patients having a specific unidentified deficiency in immune function that renders them susceptible to a rapid and fulminating spread of infection, or a unique bacterial synergy and enzyme production that increases bacterial virulence facilitating the rapid breakdown of tissue planes and thus spread. A combination of the two above mentioned factors is also possible.

10. CONCLUSION

Within a period of 3 years, 11 patients with DNM of odontogenic origin were treated at Charlotte Maxeke and Chris Hani Baragwanath Academic Hospitals with management and treatment outcomes that are comparable to international studies. Early recognition with the aid of a CT scan and aggressive medical and surgical treatments were the mainstay of DNM management in this study and resulted in a mortality rate of 9.10%. HIV did not have any bearing on treatment outcome. Future studies are needed to monitor the trends and incidence of DNM in order to elucidate why some patients are more susceptible to developing this severe infection than others. Perhaps a better understanding of the microbiology may provide insight into this vexing question.

11. Appendix 1

DATA SHEET

AGE/GENDER:

CLINICAL/ MEDICAL HISTORY AND FINDINGS:

Vital Signs

Blood pressure

Temperature

Respiratory rate

Pulse

BLOOD RESULTS: WCC

Hb

Platelets

H.I.V

Microscopy Culture and Sensitivity

CO-MORBIDITIES

TIME PERIOD FROM INITIATING FACTOR TO THE DEVELOPMENT OF SEPSIS:

RADIOGRAPHIC IMAGING AND FINDINGS

TYPE OF MEDICAL/SURGICAL INTERVENTIONS, AIRWAY MANAGEMENT, MECHANICAL VENTILATION AND SURGICAL DRAINAGE:

MEDICAL:

SURGICAL:

AIRWAY:

OUTCOME:

12. Appendix 2

Patient Summary

Case no.	Age (years)	Sex	Co-morbidities	Time from symptoms to hospitalization (days)	Treatment	Length of hospital stay (days)	Outcome
1	42	M	None	6	Tracheostomy Chamberlain Right thoracotomy	24	Discharge
2	34	M	None	5	Tracheostomy Thoracotomy Skin graft	54	Discharge
3	37	M	HIV Positive	4	Tracheostomy Thoracotomy	31	Discharge
4	28	M	None	10	Thoracotomy	44	Discharge
5	46	F	HIV Positive	2	Tracheostomy Thoracotomy	39	Discharge
6	34	M	HIV Positive	10	Thoracotomy Skin graft	117	Discharge
7	23	M	None	10	Chamberlain	26	Discharge
8	32	M	None	2	Thoracotomy	21	Discharge
9	44	M	20 cigarettes per day	11	Bilateral thoracotomy, Laparotomy, jejunostomy	76	Died
10	52	M	HIV Positive	4	Thoracotomy	23	Discharge
11	30	M	None	8	Tracheostomy thoracotomy	25	Discharge

13. Appendix 3

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG
Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49 Dr ET Mabaso

CLEARANCE CERTIFICATE

M10304

PROJECT

A Retrospective Study of Descending
Necrotizing Mediastinitis of Odontogenic Origin

INVESTIGATORS

Dr ET Mabaso.

DEPARTMENT

Department of Surgery

DATE CONSIDERED


26/03/2010

DECISION OF THE COMMITTEE*

Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE 29/03/2010

CHAIRPERSON 
(Professor PE Cleaton-Jones)

*Guidelines for written 'informed consent' attached where applicable
cc: Supervisor : Prof M Lownie

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10004, 10th Floor, Senate House, University.

I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. **I agree to a completion of a yearly progress report.**

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES...

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