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Orofacial Space Infections in Systemically Compromised Individuals: A Case Series and Review of Literature

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Odontogenic Infection; Cellulitis; Septicemia; Surgical Decompression

1. Abstract

1.1. Aim and Objectives: To review the management of head and neck infections due to odontogenic origin in medically compromised and non-compromised patients. Specifically, to observe their severity, response to common first-line broad-spectrum antibiotics, responsible bacterial microflora, and complications thereof.

1.2. Material and Methods: 45 cases of odontogenic oro-facial and cervico-facial cellulitis reporting or referred to the maxillofacial surgery outpatient department (OPD) were included in the study. Out of these, 38 were those diagnosed with some form of systemic debilitating condition and 7 were systemically healthy individuals. All the cases were managed by the same team of surgeons. Presenting cellulitis was surgically decompressed by incision and drainage. Pus specimens were subjected to culture sensitivity testing and antibiotic sensitivity testing (ABST). All patients were postoperatively monitored for wound healing, Lenth of Stay (LOS), complications, and incidence of morbidity and mortality. A prospective cohort study design was employed for the compilation and review of data.

1.3. Result: A high incidence of antibiotic resistance to routine broad-spectrum empirical antibiotics was seen in 57.8% of cases. Polymicrobial growth was seen in 65.7 % of medically compromised individuals. Only 3 out of the sample of 38 systemically compromised patients succumbed to complications like septicemia, while all patients from the immune-competent group recovered uneventfully, thus reporting a mortality rate of 8%.

1.4. Conclusion: All the cases of oro facial and maxillofacial infections due to odontogenic origin should be evaluated to rule out any undiagnosed systemic conditions in order to formulate a comprehensive treatment plan.

2. Introduction

Odontogenic infections presenting in the Emergency Department (ED) have become fairly common. Space infections evolving from the offending tooth have haunted mankind for a long; with signs of dental abscesses and evidence of osteomyelitis found in the remains of early Egyptians (1). Often unaware of their underlying systemic co-morbidities, these patients present to the ED with involvement of multiple facial planes; often complicated with life-threatening emergencies of airway compromise. Although the majority of these infections are managed routinely as out-patients, timely diagnosis with prudent surgical as well as medical management prove to be life-saving. Odontogenic infections, depending on the virulence of micro-organisms and host immunity, can present with an array of signs and symptoms. While the milder ones might limit themselves to alveolus or the jaw, the fulminating infections of the submandibular, sublingual, or parapharyngeal spaces can lead to airway compromise, cavernous sinus thrombosis, mediastinitis, or widespread septicemia. These complications are associated with a high rate of morbidity and mortality especially in medically compromised patients [2]. The systemic illnesses are known to alter the hosts' immunity and hence these patients are more susceptible to microbial infections. Systemic diseases like diabetes mellitus, renal disease, cardiac disorders, radiotherapy,

chemotherapy, and impaired liver function are considered to be an immune-compromised state [2]. Each of these conditions leads to a host environment that is more susceptible to severe pathogenic invasion, presenting either as cellulitis, pan facial abscess, septicemia, necrotizing fasciitis, and osteomyelitis. Management of these infections requires early and accurate diagnosis, aggressive incision and drainage (I&D), culture sensitivity, proper empirical antimicrobial therapy, improved nutritional status, and addressing the underlying systemic condition to achieve resolution and to reduce morbidity and mortality. This article aims to review the severity, response to commonly used empirical antibiotics, microbial flora, complications, and outcome of treatment of head and neck infections due to odontogenic origin in medically compromised and non-compromised patients. The objective of the retrospective cohort study is, to culture the microflora as well as observe antibiotic sensitivity in fulminating fascial space infections of odontogenic origin in the orofacial region.

3. Patients and Methods

This prospective cohort study was conducted on 45 patients belonging to all socio-economic groups and presenting to the division of Oral and Maxillofacial Surgery from January 2016 to June 2018, with severe Orofacial and Cervicofacial infection of odontogenic origin. The inclusion criteria involved those without any systemic disease, as well as known and unknown medically compromised patients with severe oro facial and cervicofacial infections, of any age and both the sex. The source of the infection was odontogenic in origin in all the cases. Isolation of microflora in the culture sample was mandatory (Table 1). Exclusion criteria were patients with oro facial cellulitis due to non-odontogenic causes such as furuncle, tonsillar abscess, and trauma and cases in which no microflora could be grown from the samples. All 45 patients were investigated and treated with empirical antimicrobial therapy to start with followed by incision and drainage, debridement, and extraction of the offending tooth/teeth which were the foci of infection. Out of thirty-eight patients having systemic illness, twenty-six were taken up for surgical decompression under local anesthesia and sedation by Hilton's method and the remaining twelve were surgically decompressed by Vazirani's technique under general anesthesia (GA). Of the twelve patients that were operated under general anesthesia, six were operated as planned, non-emergent procedures. The remaining six patients were operated on as medical emergencies and reported with respiratory distress, dysphagia, and signs of septicemia (Figure 1). The surgical technique consisted of first securing the airway either via endotracheal intubation or the use of a laryngeal mask airway (LMA). The surgical site was scrubbed using povidone-iodine solution and draped. Oral irrigation was done with 0.12% Chlorhexidine rinse. A decompressing incision was placed in the most dependent area and blunt dissection was carried out to break the locules. The samples were collected in sterile containers and subjected to culture and antibiotic sensitivity test (ABST). Patients with necrotizing fasciitis and open draining wounds were subjected to debridement and fasciotomy and necrotic skin and fascia samples were sent for culture, ABST, and histopathological examination (Figure 2). The wound was irrigated copiously using hydrogen peroxide and normal saline until saline backflow was free of any debris and clear. Non-irrigating corrugated drain was placed and secured with a silk suture. All 12 patients who had been operated under GA and only 6 cases who were treated under local anesthesia were admitted for post-operative monitoring. A total of 18 medically compromised patients required admission. Six patients who were taken up for emergency surgical decompression were shifted to the ICU for monitoring as per ICU protocol including ventilator support and periodic arterial blood gas analysis. All medically compromised patients were concomitantly treated by the concerned physician for their underlying systemic conditions. Empirical parenteral antimicrobial therapy was instituted with Injection Ampicillin / Augmentin, Cefotaxime, Metronidazole, and Amikacin. Metronidazole was administered for anaerobic spectrum of microbes, Amikacin for gram negative, and similarly for gram-positive cover, Injection Ampicillin or Augmentin was started empirically. Individuals, who had been prescribed penicillin derivative drugs prior to reporting to us and had not shown resolution of infection, were started with Injection Cefotaxime for gram-positive aerobic cover. Injection Amikacin was not administered to those patients with a history of liver and renal impairment. Depending upon the biochemical parameters, culture, and ABST (Table 3), the antimicrobial spectrum was altered for rendering judicious and specific chemotherapeutic agents such as Meropenem, Vancomycin, and Tazobactam. The dietary requirements of the patients having systemic illness were customized. Wound dressing was carried out twice a day, with 2% glacial acetic acid used for dressing in necrotizing fasciitis cases. The injectable antibiotics were continued for 12 to 14 days, with monitoring of biochemical parameters. All patients were followed up for a period of 6 months and underwent treatment for their medical conditions by concerned specialists as outpatients.

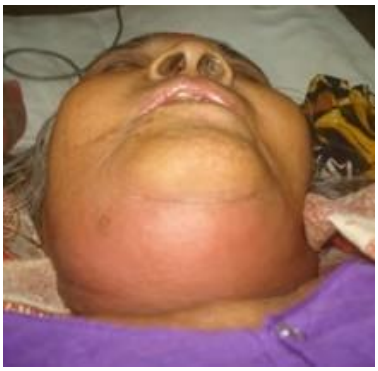

Figure 1: Cellulitis with respiratory distress, and signs of septicaemia

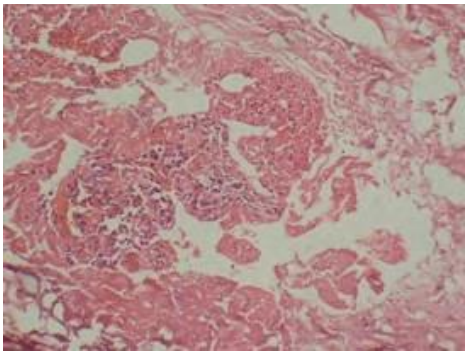

Figure 2: Photomicrograph displaying necrotic fibrous tissue with mixed inflammatory infiltrate with granulation tissue and necrotic debris

Table 1: Culture sensitivity tests of medically compromised patients

Case	Age	Sex	Microbiology	Systemic illness
1	64	F	Pseudomonas , Streptococcus (β-haemolytic),	Diabetes mellitus
2	71	F	Streptococcus(α-haemolytic), Pseudomonas	Diabetes mellitus with hypertension
3	56	M	Streptococcus (α-haemolytic)	Rheumatoid arthritis(prolonged steroid therapy)
4	58	M	Staphylococcus aureus	Diabetes mellitus
5	50	M	Streptococcus (α-haemolytic)	Nephrotic syndrome
6	61	F	Streptococcus (β-haemolytic), Pseudomonas	Diabetes mellitus with IHD
7	42	M	Streptococcus (α-haemolytic)	Alcoholism
8	44	M	Staphylococcus epidermis , klebsiella	Diabetes mellitus with IHD
9	38	F	Streptococcus(α-haemolytic), bacteroides	Chemotherapy
10	52	F	Streptococcus (β-haemolytic), Pseudomonas	Diabetes mellitus
11	51	M	Streptococcus (α-haemolytic) ,klebsiella	Glomerulonephritis
12	36	M	Coagulase negative staphylococci	Alcoholism
13	33	M	Bacteroides , Streptococcus (α-haemolytic)	Diabetes mellitus
14	37	F	Staphylococcus aureus	Diabetes mellitus with hypertension
15	41	F	Streptococcus (β-haemolytic), Pseudomonas	Diabetes mellitus with hypertension
16	38	M	Staphylococcus epidermis , bacteroides	Diabetes mellitus
17	27	M	Streptococcus (α-haemolytic)	Glomerulonephritis
18	33	F	Staphylococcus aureus	Diabetes mellitus
19	43	M	Streptococcus (β-haemolytic), Pseudomonas	Diabetes mellitus with IHD
20	36	M	Coagulase negative staphylococci	Alcoholism
21	45	M	Bacteroides , Streptococcus (α-haemolytic)	Diabetes mellitus
22	43	F	Streptococcus (α-haemolytic), klebsiella	Diabetes mellitus with hypertension
23	65	M	Streptococcus (β-haemolytic)	Diabetes mellitus
24	55	M	Streptococcus (β-haemolytic) , bacteroides	Diabetes mellitus
25	46	F	Streptococcus (β-haemolytic), Pseudomonas	Diabetes mellitus with hypertension
26	41	F	Streptococcus (α-haemolytic)	Glomerulonephritis
27	55	M	Staphylococcus aureus	Diabetes mellitus
28	39	M	Streptococcus (α-haemolytic)	Diabetes mellitus
29	45	M	Streptococcus (β-haemolytic), klebsiella	Diabetes mellitus
30	51	M	Streptococcus (α-haemolytic) ,bacteroides	Diabetes mellitus
31	49	F	Streptococcus (β-haemolytic)	Glomerulonephritis
32	41	F	Streptococcus (α-haemolytic) , bacteroides	SLE (prolonged steroid therapy)
33	33	M	Bacteroides , Streptococcus (α-haemolytic)	Diabetes mellitus
34	37	F	Staphylococcus aureus , pseudomonas	Diabetes mellitus
35	41	F	Streptococcus (β-haemolytic), Pseudomonas	Diabetes mellitus
36	38	M	Staphylococcus epidermis , bacteroides	Diabetes mellitus with hypertension
37	45	M	Staphylococcus aureus , bacteroides	Diabetes mellitus
38	51	M	Streptococcus (β-haemolytic), Pseudomonas	Diabetes mellitus

Table 2: Antibiotic sensitivity results of immune-compromised patients for first line empirical antibiotic therapy

Patient	Ampicillin	Augmentin	Amikacin	Metronidazole	Cefotaxime
1	R	R	S	S	S
2	R	R	S	S	R
3	S	S	S	S	S
4	R	S	S	S	S
5	S	S	S	S	S
6	R	R	S	S	S
7	S	S	S	S	S
8	R	R	S	S	S
9	S	S	S	S	S
10	R	R	S	S	S
11	S	S	S	S	R
12	S	S	S	S	S
13	S	S	R	S	R
14	S	S	S	S	S
15	R	R	S	S	S
16	R	R	S	S	S
17	S	S	S	S	S
18	S	S	S	S	S
19	R	S	S	S	S
20	S	S	S	S	S
21	R	S	R	S	S
22	R	S	R	S	S
23	S	S	S	S	S
24	S	S	S	S	S
25	R	R	S	S	S
26	R	S	S	S	S
27	S	S	S	S	S
28	S	S	S	S	S
29	R	R	R	S	S
30	S	S	S	S	S
31	S	S	R	S	R
32	S	S	S	S	S
33	R	S	S	S	R
34	R	R	S	S	S
35	R	S	S	S	R
36	R	R	S	R	S
37	R	R	R	S	S
38	S	S	S	S	S

Table 3: Distribution of involved spaces in patients affected

SPACE INVOLVED	NO. OF CASES
Buccal space	11
Submental space	4
Submandibular space	17
Submental, sublingual, and submandibular spaces with Ludwigs angina	4
Para pharyngeal space	2

4. Result

There were 23 males and 15 females with age ranging from 27 to 71 years with an average age of 43.64 years. Thirty-four patients had carious teeth or root stumps, whereas four patients had periodontal problems. Out of the thirty-eight patients with underlying systemic conditions, 18 (47%) were diabetics, 6 (16%) hypertensive co-existing with type I diabetes mellitus, and 3 (7%) were diabetics with CAD (coronary artery disease), 5 (13%) had chronic renal disease (glomerulonephritis), 3 (8%) were chronic alcoholics (impaired liver function), 2 (5%) were on prolonged steroid therapy (auto-immune disorders) and 1 (3%) patient was a operated case of oral malignancy on chemotherapy. Three patients out of these 38 had necrotizing fasciitis of the cervico-facial region spreading over the neck. One case had a history of chronic alcoholism with liver cirrhosis (Figure 3) and the other was a diabetic with a history of CAD. The third case was a female patient having diabetes mellitus who had well-healed extensive scars from a previous incident of severe burn injury of the neck and chest (Figure 4). Microbial culture and existing systemic disease of all thirty-eight medically compromised patients are shown in (Table 1). Polymicrobial growth was observed in 24 out of 38 (63.15%) medically compromised patients. 26 out of 38 patients were those reporting with previous antibiotic administration (long-term antibiotic usage) from other centers. (Table 2), depicts the distribution of involved spaces in patients affected. Submandibular space was most commonly involved with 17 cases (44.7%), followed by buccal space 11 (28.9%) and submental space 4 (10.5%). Classical Ludwig's angina was seen in 4 (10.5%) cases. Parapharyngeal space involvement was the least with only 2 (5.2%) of patients affected. The antibiotic sensitivity testing carried out for all patients is shown in (Table 3). Resistance to one or more antibiotics was observed in 22 out of 38 (57.8%) medically compromised patients. Out of 62 microbial isolates in systemically ill patients, 29 isolates (46.7%) were resistant to one or more group of antibiotics. Thus, polymicrobial growth and antibiotic resistance was substantial in immunocompromised cases. Out of a total of 38 compromised cases, 35 patients (92%) responded well to treatment, and 3 patients (8%), 2 diabetics and 1 chronic alcoholic with liver cirrhosis succumbed to complications of septicemia resulting in multi-organ failure.

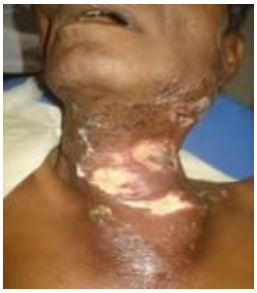

Figure 3: Medically compromised patient with chronic alcoholism with liver cirrhosis

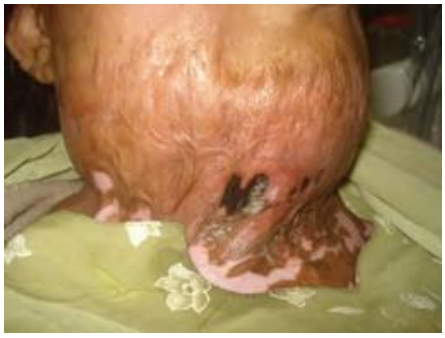

Figure 4: Female patient having diabetes mellitus and severe burn injury of neck and chest

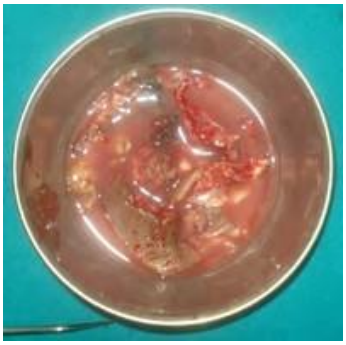

Figure 5: Sample of necrotic tissue for culture

5. Discussion

Frequently underestimated in terms of mortality and morbidity, an odontogenic infection usually occurs secondary to dental caries, trauma, or unsuccessful root canal treatment. [3] The propensity for these infections to spread and cause severe sepsis and death has been known since antiquity. The potential lethal complications of an odontogenic infection include deep neck and mediastinal abscess, sepsis, and multi-organ failure. Nevertheless, the role of bacteria in this pathological process was not realized until the turn of the 20th century.[3] Previous prospective studies revealed a 2:1 ratio of anaerobes to aerobes with a predominance of Prevotella, Anaerobic and Anginosus group streptococci and Fusobacterium. [4] However, recent researches confirm the polymicrobial nature of Odontogenic infections that includes aerobic, anaerobic, and facultative anaerobic bacteria, [5][6] that is consistent with our study as per the culture reports (Table 1). In our study, 65% of the positive cultures of the purulent exudate were polymicrobial.

No single species however has been consistently implicated in all odontogenic infections. The virulence factor of individual bacteria coupled with the synergistic relationship with other members of pathogenic flora explains the pathogenic potential of these organisms. [7][8] There are three crucial virulence factors determining the pathogenicity of anaerobic bacteria- the innate capability to survive through the oxygen tension of host tissues, the cell surface antigens/endotoxins in the form of capsular polysaccharides in gram-positive or lipopolysaccharides (LPS) in gram-negative, and the elaboration of toxins, enzymes or other substances that are lethal to living tissues. [7][8] The capsule of the Bacteroid group

potentiates virulence by preventing phagocytosis and killing through polymorphonuclear leukocytes (PMNs) [9] by preventing opsonins deposition on the bacterial cell surface, thereby surpassing the host response. [10][11] Furthermore, the capsular material induces abscess formation even when the viable bacteria are absent. [11][12] The lipopolysaccharide (LPS) endotoxins stimulate the production of inflammatory cytokines, thereby playing a pivotal role in the initiation and magnification of abscess formation. [13] The putrid smell of pus in anaerobic infections is due to the production of volatile sulfur compounds such as hydrogen sulfide and methyl mercaptan. [14][15] The basic therapeutic modality for the management of orofacial odontogenic infections includes surgical drainage, extraction of the offending tooth, and empirical antibiotic therapy to begin with. While the routine use of penicillin did not begin until the 1940s, the treatment of odontogenic infections changed substantially after Howard Florey and Ernst chain developed a powdery form of this antibiotic. [16] However, the injudicious or irrational use of penicillins has witnessed the emergence of resistance against it and the most common mechanism appears to be the drug inactivation through the production of b-lactamases. [17][18] In our study, the bacteria isolated were Streptococci group, Staphylococci, Bacteroids, Pseudomonas, and Klebsiella. The optimal approach in the selection and prescription of antibiotics revolves around choosing the narrowest spectrum of antibiotics that can effectively cover all potential offending organisms. [19] although penicillin still remains the empirical drug of choice for odontogenic infections because of its effectiveness, minimal side effects, low cost, patient tolerability, and ready availability, the rising resistance to these warrants combination with beta-lactamase inhibitors for empirical coverage in the majority of hospitalized patients. [20] In our study, 22 out of 38 (57.8%) medically compromised patients were resistant to commonly used antibiotics. This incidence of marked 'in-vitro resistance' among immune-compromised patients can be attributed to the diminished bacterial clearance due to altered WBC activity. Hyperglycemia and deranged renal/liver function are known to cause reduced leukocyte phagocytic activity and neutrophil chemotaxis as well as decreased humoral immunity. However, the most disturbing trend noticed was the high incidence of antibiotic resistance (57.8%) to one or more antibiotics, most often Ampicillin and Augmentin seen in the group of immunocompromised patients. In penicillin-allergic patients, clindamycin is the main antibiotic prescribed. However, current studies reveal higher rates of treatment failure (up to 14.0%) when clindamycin is used as a monotherapy. [21] Therefore, the preferred antibiotic of choice in penicillin-allergic patients has changed from clindamycin to cefazolin or ceftriaxone. [22] In more severe infections, such as those due to Streptococcus anginosus, combination therapy with metronidazole must be considered. [21] Many factors initiate or potentiate the spread of odontogenic infections. Of these, older age, [23][24] diabetes mellitus, [24-26] organ failure, and drugs are included as causes of immune suppression along with malignancy and acquired or congenital immunodeficiency [27] . Odontogenic infection in these medically compromised cases can have serious complications as it spreads faster to the surrounding tissues along the fascial spaces. In the present retrospective study, 38 out of 45 cases were medically compromised. The causes of medically compromised conditions were diabetes mellitus, coronary artery disease, glomerulonephritis, nephrotic syndrome, prolonged steroid therapy, chemotherapy, and alcoholism. Humoral immunity forms an integral part of host defense. B-lymphocytes produce antibodies against specific antigens produced by the invading microorganism. The host response in most infectious diseases depends on how these antigens are processed by the macrophage cells. Cell-mediated immunity is caused by sensitized T lymphocytes which have been activated by antigens of the invading microorganism [27]. Carey and Dodson in their study reported no evidence of increased incidence of severe odontogenic infection in HIV-positive patients [28]. They did however display a greater level of oral health care required in these cases [28]. Huang et al in their retrospective series of 185 cases, found a statistically significant correlation between medically compromising diseases (diabetes, liver cirrhosis, renal insufficiency, chemotherapy, and myeloproliferative disorders) and old age, complications, tracheostomy, and death [29] . Chen et al, in a retrospective analysis of 214 cases, found a significant association between immunocompromising systemic diseases, such as diabetes, renal failure, malignancy, and complications of infection such as septicemia, shock, mediastinitis, necrotizing fasciitis, and death. [30] Diabetes mellitus is recognized as the most common associated systemic disease in face and neck infections. In diabetics, the host's immune functions are disturbed by short or long-term hyperglycemia, impaired neutrophil bactericidal function, altered cellular immunity, and complement activation. All the major cell types involved in the immune defense are affected. Cellular elements of the innate immune system, including neutrophils and monocytes/macrophages, have altered function. In the neutrophils, functions such as adherence, chemotaxis, and phagocytosis are compromised. This results in a less effective defense against a microbial challenge. The neutrophils from diabetic patients also produce less free oxygen radicals, which reduce their ability to make toxic metabolites for release against microbes.[2] Monocytes and macrophages may have up-regulated catabolism of pro-inflammatory cytokines as well as increased production of matrix metalloproteases, such as collagenase. [2] This creates an imbalance that is detrimental to the containment of head and neck infections. The hyperglycemic state may also lead to a decrease in fibroblast proliferation and synthesis of collagen, impairing tissue turnover and wound repair. [31] These defects in the immune system, along

with vascular insufficiency, render diabetic patients at higher risk for a variety of severe or invasive infections, such as pyogenic bacterial infections and necrotizing infections. This inability to contain the infection leads to a high frequency of complications, including tracheostomy and prolonged length of hospital stay.[32] Alcoholism has been proven to have a detrimental effect on the immune status of an individual. It has been demonstrated that ingestion of large amounts of ethanol leads to a relatively broad impairment of host defense mechanisms. Ethanol impairs the function of phagocytic cells, including neutrophils, monocytes, and macrophages [33]. Patients who have chronic renal failure experience poor wound healing because of impairment of the de- layedtype hypersensitivity reaction. These patients are at in- creased risk for developing serious infections caused by Listeria monocytogenes, Klebsiella, and Yersinia. [27] Patients taking therapeutic doses of glucocorticosteroids may experience impaired resistance to infections. This impairment is manifested by defective phagocytic and cell-mediated functions.[27] Management of head and neck infections due to odontogenic origin remains a challenging task, more so when the immunological status of the patient is not known, either by the patient himself or the treating surgeon. Since the problem starts with a toothache, the patients are routinely advised a course of antibiotics before active dental treatment. Repeated courses of antibiotics in non-responsive cases worsen the condition further. A thorough history coupled with a detailed physical examination should be carried out for all patients. This provides information about onset, course, systemic conditions, and medications. Such indicators include a positive history of HIV, a previous diagnosis of diabetes, or signs and symptoms of the disease, alcohol or illicit drug use, renal dialysis, and a recent history of recurrent infections.[33] When performing the physical examination, one should keep in mind that immunocompromised individuals on steroid therapy and those reporting with repeated antibiotic administration may have an attenuated immune response resulting in decreased signs and symptoms of inflammation. In acute and severe infection, the most important consideration is the assessment of the airway. If the airway is compromised, the first course of action is either oral or nasal endotracheal intubation. In instances where edema of the oropharyngeal airway is severe, it may be necessary to perform a tracheostomy to establish a competent airway.[34] None of our cases required emergency tracheostomy. Of the six patients who had been taken up for emergency surgical decompression, all underwent fiberoptic intubation to secure the airway. Various laboratory and imaging studies are done to establish the diagnosis and determine the extent of the infection. Routine laboratory studies such as a total and differential white blood cell count, hemoglobin and hematocrit determination, platelet count, measurement of electrolytes, blood urea nitrogen, creatinine, and glucose should be performed. A high percentage of immature neutrophils would indicate that the immune system is struggling to produce cells to fight the infection.[2] For any odontogenic infection, the acute phase response is a complex of systemic and metabolic reactions, and the concentration of C- reactive protein (CRP). However, CRP is not a prognostic factor in assessing the extent of odontogenic infection; instead, clinical evaluation, assessment, and severity scoring are of the greatest prognostic value. [35] Imaging studies may include plain films, CT scans with or without contrast, and MRI. However, recent studies highlight that there has been a rising trend towards overuse of CT for the workup of odontogenic infections in the emergency department which can lead to increased risk of malignancies and poor coat-effectiveness for patients. [36] Rather, the use of ‘red flag sign’ can aid in making better clinical decisions as to whether to demand a CT or not. [36] Samples of necrotic tissue or discharge should be collected for culture and ABST (Figure 5). The primary treatment of head and neck fascial space infection with suppuration is surgery. Incision and drainage to surgically decompress the fascial spaces is the cornerstone of surgical treatment. Decompression permits evacuation of the pus and necrotic debris, decreases hydrostatic pressure, and provides an aerobic medium within the tissue spaces. If necessary, repeated surgical intervention to remove necrotic debris, carry out thorough debridement, and irrigation may be necessary if fulminating infection persists. This was done in two of our cases of widespread necrotizing fasciitis who were taken up for surgical debridement a second time, three days after the first surgery. Although the initial prescription of antimicrobial therapy is always empirical, over the last decade we have observed a change in practice with the use of third-generation cephalosporins, in conjunction with metronidazole, replacing benzylpenicillin and metronidazole. More recently, evidence has emerged suggesting that antimicrobial resistance in nosocomial infections could be related to the widespread use of second and third-generation cephalosporins.[34] The ‘in vitro’ resistance to the empirical antibiotics employed translates in-vivo into improvement in clinical conditions. If the infection responds well to the empiric use of an antibiotic, the regimen should be continued even if the culture and antibiotic sensitivity test indicate a change may be appropriate. However, in the absence of clinical improvement, the culture and antibiotic sensitivity test results should form the basis for continued antimicrobial therapy.[37] In our study, 22 out of 38 (57.8%) medically compromised patients displayed ‘in-vitro’ resistance to commonly used antibiotics. As no improvement in clinical condition was observed in those displaying resistance in-vitro, the antibiotic regime in the said patients was modified and tailor-made as per the antibiotic sensitivity results, resulting in rapid improvement in their clinical condition. Thus effectively, 22 out of 38 (57.8%) medically compromised patients displaying ‘in-vitro’ resistance to commonly used empirical broad-spectrum antibiotics required revision of their antibiotic regimen in accordance with their sensitivity results for in-vivo correction of clinical

symptoms. Higher-end antibiotics such as Imipenem, Meropenem, Piperacillin, Tazobactam, and Teicoplanin were employed in accordance with the antibiotic sensitivity results. This conveys the reliability of in vitro determination of antibiotic sensitivity. The lack of vascularity can result in failure of immune effectors and antibiotics to reach the infected sites. Proper placement of drains and periodic irrigation through them is essential for the continuous removal of necrotic debris and enhancement of vascularization. Specimens for culture should be taken at the time of surgical debridement or incision and drainage. Both aerobic and anaerobic cultures should be done and antibiotic sensitivity testing should be performed to provide guidance in selecting the correct antimicrobial treatment. Finally, close post-operative monitoring, including nutritional supplementation, addressing overall systemic status, additional surgical interventions if required, and follow-up is mandatory for resolution of the infection. Immune-compromised patients with deep and widespread fascial space infections reporting late with signs of septicemia are difficult to save. The incidence of mortality in medically compromised cases in our study group is 8 %. Three patients who had reported with severe septicemia and SIRS (systemic inflammatory response syndrome) due to extensive cervico facial cellulitis and underlying uncontrolled diabetes, developed MODS (multiple organ dysfunction syndrome) and eventually succumbed to cardiac arrest.

6. Conclusion

Broad-spectrum bactericidal agents are the first choice of empirical antibiotics to be administered until ABST and culture reports are obtained and early surgical intervention is a must to increase the efficacy of supportive therapy.

In cases of systemically compromised patients, a higher-end antibiotic with a broader spectrum of action should be started on an empirical basis. All cases with fulminating and extensive odontogenic infection should be thoroughly evaluated for systemic illness. Antibiotic resistance and polymicrobial infection are common in immunocompromised cases. Unless the underlying systemic condition is addressed and remedial measures are taken to improve or stabilize the immune status, the infective process will not resolve irrespective of the nature of medication administered or surgery carried out.

7. Acknowledgement

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8. Conflict of Interest

We have no conflict of interest.

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