Necrotizing Cervical Fasciitis, Retrospective study of Cases and management

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Abstract:

Total nine patients of Cervicofacial Necrotizing Fasciitis (CNF) were treated at ENT Department Al Ain Hospital between September 2007 and September 2009. The out come of their management studied retrospectively and presented. All patients were evaluated regarding clinical manifestations, etiology and site of infection and details of their management were discussed. Majorities of patients were diabetic male adults. All patients had odontogenic infection as the primary source of infection, except one who had parapharyngeal abscess.. The bacteriology showed streptococci on top of the list, but all of them had multibacterial infections. Gentamycin, metronidazol, benzyle Penicilline and clindamycin represent a combination thearapy covering all gram negative, gram positive and anaerobes for all cases. Histopathology showed extensive tissue necrosis with dermal vascular necrosis. Mortality was 1/9 (11.1%). One patient died from his illness and others recovered after proper surgical and medical management. Details of their presentation, site of infection, bacteriological causes and out come of the management are discussed and results compared with other articles. We emphasize early diagnosis and initiation of both aggressive surgical (repeated debridement) and medical treatment (combinations of antibiotics) and a multidisciplinary approach is essential to minimize mortality.

Keywords: CNF , Fascitis

Introduction:

Cervical Necrotizing fasciitis (CNF) is a serious soft tissue infection generally caused by Group A beta-hemolytic Streptococci (bacteria), but may also be caused by anaerobic bacteria (Peptostreptococcus, Bacteroides). Necrotizing fasciitis is a life threatening infection first described by Meleney with a recognized pattern of subcutaneous necrosis and progression [1].

It is common in immunocompromised patients with diabetes mellitus, cancer, alcoholism, vascular insufficiencies, organ transplants, HIV, or neutropenia [2]. NF may occur as a complication of a variety of surgical procedures, including cardiac catheterization. Infection is often the result of a skin abrasion or puncture that becomes secondarily infected with Streptococcal bacteria. Most cases have a recognized odontogenic or pharyngeal origin.

Subcutaneous spreading infection is infrequent and its development is determined by continuity with fascial planes of adjacent anatomical regions, notably thorax and mediastinum [3,4].

NF tends to begin with constitutional symptoms of fever and chills. After 2-3 days, erythema is noted, and supralesional vesiculation or bullae formation appears. Serosanguineous fluid may drain from the affected area.

In a pediatric NF series, clinical features begin 1 week after the initiating event, beginning with edema and indurations, which is followed in 24-48 hours by erythema or a violaceous

discoloration of skin Pain and occasionally crepitation, was also noted early [5]. The latter indicates the presence of gas produced by aerobic and anaerobic bacteria and is highly suggestive of the diagnosis of NF. The patient usually appears moderately to severely toxic, but early in the disease the patient may look deceptively well. Typically, the infection begins with an area of erythema that quickly spreads over a course of hours to days. The redness quickly spreads, and the margins of infection move out into normal skin without being raised or sharply demarcated. As it progresses, the infection gives way to dusky or purplish skin discoloration near the site of insult (Figures 1,2). Multiple identical patches develop to produce a large area of gangrenous skin, as the erythema continues to spread. The initial necrosis appears as a massive undermining of the skin and subcutaneous layer. If the skin is open, gloved fingers can pass easily between the two layers and may reveal yellowish-green necrotic fascia. The normal skin and subcutaneous tissue are loosened from the rapidly spreading deeper necrotic fascia that is a great distance from the initiating wound. Facial necrosis is typically more advanced than the appearance suggests. (figure 3).

Organisms spread from the subcutaneous tissue along the superficial and deep fascial planes, presumably facilitated by bacterial enzymes and toxins. This deep infection causes vascular occlusion, ischemia, and tissue necrosis. Superficial nerves are damaged, producing the characteristic localized anesthesia [6,7].

Without treatment, secondary involvement of deeper muscle layers may occur, resulting in myositis or myonecrosis. Normally, however, the muscular layer remains healthy red with normal bleeding muscle under the yellowish-green fascia. Septicemia is typical and leads to severe systemic toxicity and rapid death unless appropriately treated [8].

figure 1: showing swelling and erythematous change.

figure 2: showing initial violaceous discoloration of skin.

figure 3: showing wide necrosis of soft tissue.

Streptococcal pyrogenic exotoxins (SPEs) A, B, and C are directly toxic and tend to be produced by strains causing NF. These pyrogenic exotoxins, together with streptococcal superantigen (SSA), lead to the release of cytokines and produce clinical signs such as hypotension [9].

This infection results in extensive soft tissue destruction (necrosis) of skin, subcutaneous tissue and muscle. Often repeated surgical intervention (debridement) in combination with antibiotic therapy is required [10].

Mortality rate is high particularly if treatment is delayed. Familiarity with NF may facilitate earlier diagnosis and initiation of appropriate therapy and good outcome .

Materials and Methods:

Medical records of cases of NF which were seen at Al-Ain hospital between September 2007 and September 2009 were borrowed from Medical Record section at Al Ain Hospital, UAE. Criteria for diagnosis of NF adopted by us (see below). Data for age, sex, history of Diabetes, site of infection and presenting symptoms all collected.

Details of radiological and Lab. Investigations, including bacteriological results were recorded. The outcome from detailed surgical and medical therapy, length of stay in hospital, complications occurred and survival of cases all recorded. Data compared with other internationally published cases.

Our treatment plan consisted of operative wound exploration, the fascial planes of the deep cervical fascia exposed through a wide and low transverse incision in the midline. Widespread debridement of all infected and necrotic tissues was carried out until normal tissues were noted and serous or cloudy fluid was no longer seen. The wound was well irrigated with 1% H_2O_2, normal saline and then povidone iodine. Hemostasis was maintained, and the wound was kept open. Repeated operative debridement in either the operating room or at the bedside was carried out to prevent further progression of the disease. Initial empirical broad spectrum antibiotic coverage, starting with gentamycin inj, benz pecicilline, metronidazole and Clindamycin was given, followed by antibiotic adjustments based on culture, and sensitivity results.

Results:

Criteria adopted by us for the Diagnosis of Cervical Necrotizing Fasciitis:

1. Clinical evidence of severe, rapidly progressive infection with +/- history of immunosuppression.
2. Evidence of any oral, dental, oropharyngeal, laryngeal or cervical focus of infections, figure 4.
3. Radiological evidence of subcutaneous air. Figure 6,7.
4. Evidence of necrosis on surgical exploration with sampling of deep tissue for histopathologiacl confirmation of dead necrotic tissues, is the most accurate means of diagnosis.

All patients were males. The age ranged from 35-55 yrs. (mean age-45 yrs). Seven out of nine cases were having uncontrolled diabetes. All patients had odontogenic infection as the primary source of infection, except one who had parapharyngeal abscess (figure 4,5).

Laboratory investigations included full blood counts (WBC>14,000/µL), blood urea nitrogen(>15 mg/ L), serum electrolytes (serum sodium <135 mmol/L), coagulation profile (PT, APTT, INR- deranged), Bl. Sugar (R >8 mmol), C/S of the aspirate and histopathological examination of the underlying tissue. (Table 1).

Plain-Xray neck (figure 6), MRI or CT (figure 7), delineation of the extent of NF and presence of gas in soft tissues and it was very useful in directing rapid surgical debridement.

Follow up CT control at intervals after surgical debridement was a good guide line for further debridement.

Bacteriology: Majority had mixed infections, both aerobic and anaerobic. Commonest pathogen was Staph.aureus and Strept.group C and pseudo aerogenosa, sensitive to Penicillin, Cloxacillin, gentamycin and Clindamycin. Table 1.

Five patients out of nine required ICU care. Three of them required prolonged ICU care because of respiratory and multiple organ failure. While two of them were in ICU for short period of time as they developed Mediastinitis and coagulopathy. Staying in hospital ranged from 10 days to 93 days with a mean of 36 days.

Complications were seen in the form of: Airway obstruction (AO), Pleural effusion (PE), pneumonia (Pneu.), respiratory failure (RF), Pericardial effusion (PcE), heart failure (HF), Empyema, septicemia (Sep), Arterial erosion, coagulopathy (Cog.) and gastrointestinal bleeding (GIT B), disseminated intravascular coagulation (DIC), Jugular vein thrombophlebitis, Septic shock (S sh), and systemic multiple organ failure (SMOF) Table 1.

Four patients out of nine had mediastinal spread of the infection (Med). Two of our patients developed multiple organ failure (MOF).

Four patients developed severe thrombocytopaenia (TP), which was aggressively managed by Peripheral smear, PLT function test monitoring, PLT transfusion and Oprelvekin (Neumega)- was also used in two patients for the prevention of severe thrombocytopenia and the reduction of the need for platelets Transfusion (figure 8).

All patients were started with IV Antibiotic in the form of Benz. Penicillin, Gentamycin, clindamycin and Metronidazole, Later modified according to culture and sensitivity reports.

Early wound exploration was done in all nine patients and surgical debridement of necrotic tissue was done through wide transverse incisions in the neck, the fascial planes of deep cervical fascia was exposed. All infected and necrotic tissue was removed until normal tissue was noted. All patients were kept under close

care to closely monitor hemodynamic parameters.

All patients except two required multiple surgeries for surgical debridement, either in operative room or bed side. Three patients out of nine required tracheotomy after prolonged intubation.

Eight patients recovered well after extensive medical and surgical treatment, one patient died from septicemia and SMOF.

Figure 4: showing odontogenic infection dental root infection and necrosis of lower jaw.

Figure 5 : unhygenic Mouth cavity.

Figure 6:Plain X-ray, showing gas in the soft tissue neck.

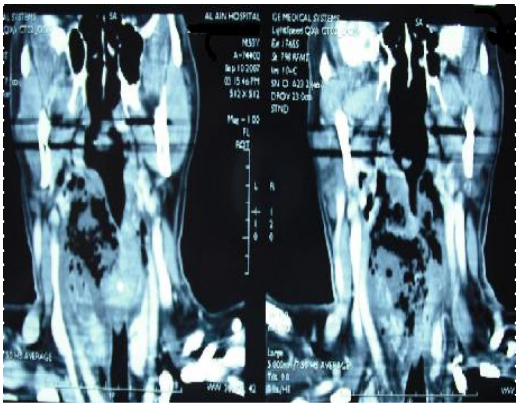

Figure 7: CT (contrast)- same patient, showing detail of gas spread in the neck mediastinium.

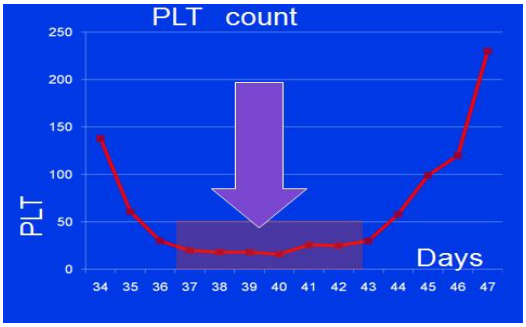

Figure 8: showing changes in platelet counts

Table 1: Short summary of data of patients with Deep Cervical Necrotising Fascitis (CNF).

	Age/ Gender	Aetiology	Past history	Radiological evidenc Subcut air	Repeated surgeries / No.	I.C.U.	Tracheostomy	Complications	Culture	Hospital Stay days	Out come
1	55/ M	odontogenic	DM	Plain + CT +	Yes 7	Prolong	Yes	Med., AF, Pneu, HF,RF, Sep, GIT B, Coag, DIC, TP, MOF	Staph.aur., Pseud.aer. Strep.gr.C Kleib..pne	60	Died
2	49/ M	odontogenic	DM	Plain + CT +	Yes 6	Prolong	Yes	Med, Pneu HF,RF,GIT B Cog, DIC,PE, PcE, TP MOF	Stap.aurus Strep.gr.C Psd.aerug. Haemp.inf Asper.fum Bacteroids	93	Dischar ged
3	44/ M	odontogenic	DM	Plain + CT +	Yes 5	Prolong	Yes	Med., Sep., RF, PE., TP	Stap.aure Bacteroids Strep.virid	48	Dischar ged
4	42/ M	Paraphary. abscess	DM	Plain + CT +	Yes 2	short	No	Med.,Coag.	Stap.aure. Strep.gr.C Pseud.aer.	12	Improv ed LAMA
5	45/ M	odontogenic	DM	Plain – CT +	No (1)	No	No	Coag,TP	Stap.aur. Strep.gr.C	38	Dischar ged
6	38/ M	odontogenic	DM	CT +	Yes 2	No	No	Coag	Stap.aure. Kleb.pneum . Bacteroids	10	Dischar ged
7	50/ M	odontogenic	DM	CT+	Yes 3	Short	No	Med., Coag., RF	Strep.viri. Strep.gr.C Bhaem. Strep Peptostrepto	27	Dischar ged
8	36/ M	odontogenic	Nil	CT +	No (1)	No	No	Nil	Strep.virid. Stap.aure.	14	Dischar ged
9	35/ M	odontogenic	Nil	CT +	Yes 3	No	No	Nil	Stap.aureus Pseud.aerug	12	Dischar ged

Discussion:

Approximately one half of the cases of streptococcal NF occur in young and previously healthy people. The mean age of a patient with necrotizing fasciitis is 38-44 years. However cases were reported in pediatric age group also [11]. The male-to-female ratio is 2-3:1.

Most of the odontogenic and oropharyngeal lesions are relieved with proper antibiotics and anti inflammatory drugs. But in rare cases, especially in patients with poor health or immunocompromised, this life threatening infection may spread through deep neck spaces and hence producing soft tissue necrosis namely Cervicofascial Necrotizing Fasciitis.

The overall reported annual incidence of Necrotizing Fasciitis in human body is 2-3 cases per year. Jacob Ndas et.al. reported 56 cases of NF in four years [12]. But reported incidence of Necrotizing Cervical Fasciitis is quite low. N. Vaid reported only 68 cases of Necrotizing Cervical Fasciitis in world literature till 2002 [13]. Helmy A S et.al. reported only eight cases of Cervical Necrotizing Fasciitis in the period of 53 months [14]. In our study the incidence of nine cases in the span of twenty four months.

All of our patients were males in the adult age group and majority were having premorbid immunosuppresive condition of diabetes. Odontogenic infection was the primary source of infection in all of them except one in which it was a parapharyngeal abscess.

This life-threatening infection tends to be caused by mixture of gram-negative and gram-positive organisms. Group A beta hemolytic streptococci and *Staphylococcus aureus,* alone or in synergism, are frequently the initiating infecting bacteria. However, other aerobic and anaerobic pathogens may be present, including *Bacteroides, Clostridium, Peptostreptococcus,* Enterobacteriaceae, coliforms, *Proteus, Pseudomonas,* and *Klebsiella*. *Bacteroides fragilis* is usually noted as part of a mixed flora in combination with *Escherichia coli* [15]. The polyculture of bacteria often seen may create bacterial synergism and causing increasing virulence [16].

In our study also the infection was polymicrobial. Commonest pathogen were gram posive Staph.aureus. and Strept.group C sensitive to Penicillin, Cloxacillin, anaerobics sensitive to Clindamycin and Metronidazole and gram negative Pseudmonuos aerogenosa sensitive to gentamycin, see Table 1.

Based on our sensitivity results, all patients were given IV Antibiotics, IV Benzyl penicillin, Gentamycin clindamycin and Metronidazole. Majority of patients responded well to this regime.

Ultrasonography is a useful investigation for early diagnosis [17].

CT scan was a tool of great help in our study to provide constant features characteristic of CNF and helped in discovering unsuspected extensions of the inflammatory process and provided excellent information of the extent of the disease into mediastinium and confirming the presence or absence of the gas. (figure 9). This is in contradiction with the studies of Deganello A et.al. [11], who failed to get any useful information from CT scan in a case of CNF with Lemierre syndrome.

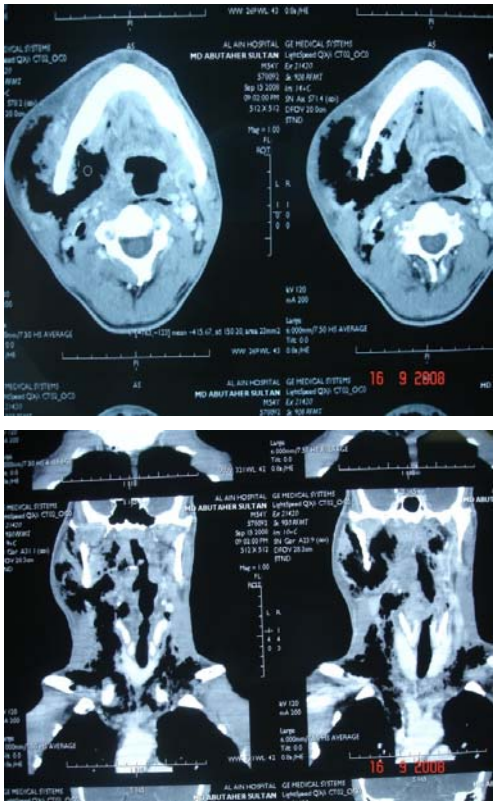

Figure 9: showing extensive soft tissue necrosis and gas in the soft tissue.

Once the diagnosis of NF is confirmed, initiate treatment without delay. Because of the complexity of this disease, a team approach is the best. A team including a surgeon, an infectious disease specialist, a pathologist, and a microbiologist, an E N T surgeon and an intensivist is the best method of treating this complicated disorder.

Combination antibiotic therapy involving the use of 2 or 3 antibiotics has to be initiated. To cover aerobes (usually gram-negative organisms), Ampicillin and Gentamicin are useful. For anaerobes, clindamycin **or** metronidazole has been used. In group A streptococcal infections, clindamycin has been used, specifically in combination with beta-lactam antibiotics.

In severe streptococcal infections associated with NF, the use of intravenous immunoglobulin (IVIG) may be a useful adjunct treatment.

All of our patients needed surgical drainage and debridement of the necrotic tissue. Majority of our patients needed aggressive repeated surgical debridements (more than once, table 1) with wide exposure of all fascial planes and safe removal of all necrotic tissue to ensure survival. Repeated surgical exploration and debridement was necessary until there was no evidence of necrosis. These findings augers well with the previous studies [18,19,20].

The entire necrotic area should be excised. The wound should be well irrigated with 1% H_2O_2, normal saline and then povidone iodine. Hemostasis should be maintained, and the wound should be kept open. Surgical debridement and evaluations should be repeated almost on a daily basis and the wound should be inspected in the operating room.

Follow up CT control at intervals after surgical debridement is a good guide line for farther debridement, (Figure 10).

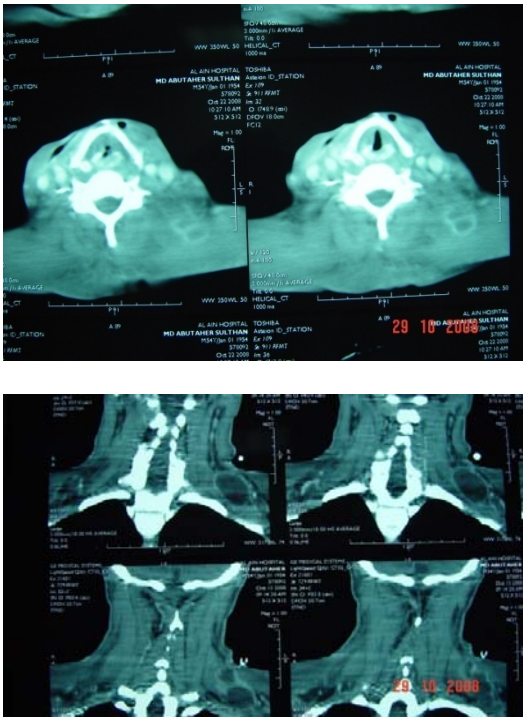

Figure 10: shows pus collection supraclaviculare area after debridment.

Dunn C et.al and Preuss SF et.al used maggots to clean the necrotic wounds in head and neck Necrotizing fasciitis [21,22]. While Jacob N L used fresh honey to clean and dress the wounds in necrotizing fasciitis [12]. Kamulegeya A [23] used 0.3% hydrogen peroxide to clean the wound while John D Edwards et.al. [17] used 0.5% povidone iodine solution for irrigating the necrotic wound.

We found fresh hydrogen peroxide irrigation on daily bases in the necrotic wound after through surgical debridement and followed by irrigation of the wound with normal saline and povidone iodine 0.5% is the ideal method for the wound care.

Complications were seen in the form of mediastinitis, airway obstruction, pleural effusion, pericardial effusion, empyema, arterial erosion, thrombocytopaenia, coagulopathy, jugular vein

thrombophlebitis, septic shock and systemic multiple organ failure (SMOF).

Three of our patients developed mediastinitis (33.33%) because of descending CNF (table 1).

Infection from neck to mediastinum can occur by three primary routes- via retropharyngeal space, the perivascular space and the pretracheal space. Retropharyngeal space is the most commonest route [19].

Generally these patients need long stay in the hospital with multiple lab. and radiological investigations, series of surgical intervention, intensive medical care, heavy doses of IV antibiotics, multiple transfusions of blood and blood products.

In eight patients study by Helmy et.al. [14], the mean hospital stay was approx. twenty days, while Roccia F [6] reported mean hospital stay of thirty two days. In our study the mean hospital stay was thirty six days (range 10-93).

In cases of CNF mortality is still high despite advances in antimicrobial drug regimes, modern investigating tools, surgical advancement and advancement in care of critically ill patients. The poor prognosis in NF has been linked to infection with certain streptococcal strains. The mortality rate can be as high as 25%. Cases of NF with sepsis and renal failure have a mortality rate as high as 70%. The overall morbidity and mortality is 70-80%.

Reported mortality rates are Helmy A S et.al[14] – 37.5%, F. Roccia et.al.[6] - 30.4%, P. Singhal et.al. [20] - 25-40%, Sumi Y et.al. [24] - 25-40%.

In our present series mortality rate was 11.11%. One of our patients died from systemic multiple organ failure after 2 Months stay in patient care.

Conclusion:

Cervical necrotizing fasciitis is an aggressive bacterial infection and the diagnosis is primarily based on the clinical history, physical findings and histopathological evidences of dead necrotic tissue.

-Based on our study we recommend necessity of early diagnosis of Cervical Necrotizing Fasciitis by high index of suspicion (specially in immuno - compromised patients), based on clinical features, rapid progression, radiological investigations and surgical exploration.

-CT scan is one of the most important tool to diagnose the presence of disease and later on to know the progressive improvement or deterioration of the disease.

- For a good outcome, medical and surgical management of such patients must be immediate and aggressive, specially in those with uncontrolled diabetes, who have compromised immune system.

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توێژینەوە لەسەر حالەتی ئەو نەخۆشانەی کە تووشی هەوکردنی ماسولکە و نەرمایی یەکانی مل دەبن

پوختە

نۆ نەخۆش کە تووشی ئەم هەوکردنە ترسناکە بوون لە ملیان چارەسەر کران لە ماوەی نێوان 2007-2009 (24 مانگ) لە نەخۆشخانەی (العین) لە ابوظبی. ئەم حالەتانە زۆربەیان نێر بوون و نەخۆشی شەکرەیان هەبوو. هەوکردنی ملیان لە هەندێ نەخۆشی دڕان و شەویلگەوە بلاو ببوو بۆ ملیان، بە درێژی باسی ئەم نەخۆشیە کرا کە چۆن خۆی نیشان دەدات، چیە نارەحەتی نەخۆش، چۆن دەبێت چارەسەربکرێت. میکرۆبی ئەم نەخۆشیە بریتی بوو لە کۆمەڵێك میکرۆب بە تایبەتی لە چەشنی (ستریپتوکوکس). نەخۆشەکان چارەسەرکران بە دەرمانی (جنتامایسمین)، پەنسلین وە مترونیدازول لەگەڵ کلیندامایسین لەگەڵ رۆژانە پاك کردنەوەی برینەکە باش بە نەشتەرگەری. یەکێك لەم نۆ نەخۆشانە مرد و هەشتیان چاك بوونەوە، هۆی مردنی ئەم نەخۆشە ئەوە بوو کە زۆر لە بەشەکانی لەشی لە کار وەستا بەهۆی هەوکردنەکەوە. لەم توێژینەوەیە زانرا کە ئەم نەخۆشیە زۆر ترسناکە وە ئەگەر بە زووترین کات نەخۆشیەکە دیاری نەکرێت و چارەسەری نەکرێت بە تۆندی بەهۆی دەرمان و نەشتەرگەری وە نەخۆشەکە رزگار نەکرێت لەم هەوکرنەوە ئەوا زۆربەیان دەمرن. ئەم نەخۆشیە بە شێوەیەکی گشتی تووشی ئەوانە دەبن کە بەرگری لەشیان کەم دەبێتەوە بە هۆی نەخۆشی شـــەکرە و دەرمانی چارەســـەرکردنی شێرپە نجە و هتر...

بحث ذو اثر رجعي لحالات الالتهابات أغشية الرقبة النخرية مع معالجة هذه الحالات

الخلاصة

عولج تسعة حالات للمصابين بألتهابات الاغشية الرقبة النخرية من فترة بين أيلول 2007 الى أيلول 2009 في مستشفى العين في ابو ظبي. نوقش معالجة و متابعة هذه الحالات من حيث اعراض مرضهم و معالجتهم والنتيجة الحاصلة من متابعتهم وما حدث لهم. اغلبية المرضى كانوا مصابون بداء السكر وكلهم ذكور. وكان مصدر هذه الألتهابات من الاسنان و الفك في 8 حالات وفي حالة واحدة مصدر الألتهاب كان من البلعوم. الجرثومة المسببة للمرض كان جراثيم مختلفة و على رأسهم جرثومة (ستريبتوكوكس). عولج الألتهابات هذه بعقافير جنتامايسمين، مترونايدازول، ثنسلين مع كليندامايسين. و اعطيت هذه التركيبة بنتائج جيدة. الفحص النسيجي للعينات المأخوذة من هذه الألتهابات بين وجود تلف و نخر من الانسجة الرغوة من الرقبة. توفى احد المرضى من بين تسعة مرضى الذين عولجو، وكان سبب الموت عجز الكلي لمختلف اعضاء جسمه بسبب تسمم بهذه الألتهابات. بينما أشفيت الباقون. في هذا البحث نقدم تفاصيل اعراض هذا المرض و كيفية تشخيص الحالات و معالجتهم بواسطة عقافير طبية و أجراء عمليات تنظيف الألتهابات بشكل يومي و متواصل لحين شفاء المريض. من هذا البحث وصلنا الى نتيجة ان هذه الألتهابات من نوع خطير و تحدث لدى المرضى المصابون بضعف المقاومة الجسمية العامة (مصابون بداء السكر). و نوصى بالتشخيص المبكرللحالة و البدأ بالعلاج الدوائى بشكل مركز وأجراء عمليات جراحية متكررة لهم. بهذه الطرق يمكن انقاذ هذه الحالات من الموت المحقق.