الالتهاب التنخري للنسيج الخلوي تحت الجلد والصفق العضلية في منطقة الوجه

ميسون قاسم *

الملخص

خلفية البحث: يعدُ إلتهاب الصفاق العضلي التنخري سريع الانتشار من أخطر أخماج الأنسجة الرخوة خاصة ويمكن تشخيصه خطأ على أنه التهاب نسيج خلوي بسيط، الأمر الذي يؤخر تشخيصه ومن ثَمَ علاجه.

هدف الدراسة: إن الهدف من هذه الدراسة هو مراجعة جميع حالات التهاب النسيج الخلوي التنخري في الوجه التي عولجت في مستشفى مبارك الكبير في دولة الكويت في الفترة ما بين 1989 – 2005 وتحليل مظاهر هذا المرض النادر واستجابته للعلاج وإيضاح مدى خطورته على الحياة ومن ثَمَّ تقليل نسبة الوفيات بالتشخيص المبكر والعلاج السريع.

المواد والطرائق: وتتناول دراسة 4 حالات من التهاب النسيج الخلوي التنخري في منطقة الوجه من أصل 120 حالة خمج خلوي وجهي عولجت في قسم الجراحة العامة في مستشفى مبارك الكبير، وتشكّل متابعة علاج هؤلاء المرضى سريرياً ودراسة ملفاتهم وتحليلها أساس هذا التقرير.

النتائج: لاحظنا أن معظم المرضى كانوا يعانون من وهن وإنهاك عام يرافقه ترفع حروري و انخفاض في الضغط، كما لوحظ أن معظمهم لديه قصة مرض عام موهن يؤهله للإصابة بهذا الخمج.

^{*} مدرسة - قسم جراحة الوجه والفكين- كلية طب الأسنان- جامعة دمشق.

وقد عولجوا بنقل المصول والسوائل وإعطاء المضادات الحيوية واسعة الطيف والمسكنات عن طريق الوريد، كما أزيلت جميع الأسجة المتنفرة وبشكل واسع، أما الأسجة الحية المكشوفة فقد تم غُطِّيت مؤقتاً بضمادات خاصة لتغطى فيما بعد بالطعوم الجلدية المناسبة. هذا وبفضل الكشف المبكر والتدخل الجراحي السريع فقد تم إنقاذ هؤلاء المرضى دون حدوث وفيات.

المناقشة: يمكننا القول إنَّ الكشف المبكر والتدخل الجراحي الواسع والسريع فضلاً عن العلاجات الداعمة بإعطاء الأدوية ونقل السوائل المكثف، تشكل حجر الأساس في علاج هذا المرض.

الكلمات المفتاحية: الالتهاب التنخري للنسيج الخلوي تحت الجلد في الوجه – التشخيص المبكر – التدخل الجراحي الواسع.

Facial Subcutaneous Necrotizing Fasciitis

Maysoun Kassem^{*}

Abstract

Background: Diffuse necrotizing fasciitis is the most treacherous soft tissue infection particularly because it may misdiagnosed as simple cellulitis, thereby delaying diagnosis and treatment.

Purpose: the purpose of this study is to review all cases of facial S. necrotizing infections managed in MKH_kuwait In between 1998-2005. and to analyze the features of this disease and its response to treatment, to supplement the understanding of this relatively rare and life threatening disease, and to minimize its mortality risk by early diagnosis and aggressive treatment.

Methods: 4 patients with facial necrotizing fasciitis between 120 cases of facial infections admitted to MKH were managed in the surgery unit. The hospital records of these patients were reviewed and form the basis of this report.

Results: Most patients presented with fever, hypotension and malaise. All of the patients had pre-morbid state which could have predisposed them to the infection. They were managed with intravenous fluids, antibiotics and analgesics, most patients had extensive debridement with subsequent wound dressing before skin grafting. all patients had survived, with no mortality thanks to early diagnose and correct intervention.

Conclusion: Early diagnosis, aggressive surgical intervention combined with supportive therapy is crucial to the successful treatment of the disease.

Key words: facial necrotizing fasciitis, early diagnosis, aggressive surgical intervention.

^{*}Instructor. Dept. of OMS. Faculty of Dentistry, Damascus University.

Introduction:

Necrotizing fasciitis is a soft tissue infection that causes necrosis of fascia and subcutaneous tissue, but spares skin and muscle initially (1,2,3,4). The groin, abdomen and extremities are the most frequent sites involved by this disease(1,9,10). The eyelids, scalp, face and neck are rarely involved, with only 40 cases reported in the literature (9). In 1952, Wilson first used the term "necrotizing fasciitis," which is the most accurate term to describe this disease.

necrotizing fasciitis of the face is very rare, Dental infections are the most common etiology, followed by trauma, peritonsillar and pharyngeal abscesses, and osteoradionecrosis (8,9,13,16) The bacteriology consists of anaerobes, gram negative rods group A *b-hemolytic strep*, and staph. species (1,4,5,12).

It is not only associated with a high mortality but can also result in a severe disfigurement of the face, posing challenging reconstructive problems (1,9,16).

The onset of symptoms is usually 2 to 4 days after the insult (1,5,9). The skin is smooth, tense and shiny with no sharp demarcation, and develops a dusky discoloration with poorly defined borders (1,9,16). There is localized necrosis of skin which is secondary to thrombosis of nutrient vessels as they pass through the zone of involved fascia (9). If untreated, this will progress to frank cutaneous gangrene. Clinically there is sudden pain and swelling and the skin becomes warm, erythematous, and edematous and can be mistaken for cellulitis or erysipelas (1,5,10). Three zones of skin are recognized and include a wide peripheral zone of erythema surrounding a tender dusky zone, and a central zone of necrosis that eventually ulcerates. There can be anesthesia of the skin from involvement of the cutaneous nerves as they pass through necrotic subcutaneous tissue (9). Soft tissue crepitance is common from gas formation. These patients often have a low-grade fever and can be anemic and jaundiced from bacterial hemolysis. Massive amounts of fluid can be sequestered with resultant hyponatremia, hypoproteinemia, dehydration. Hypocalcemia can develop from necrosis of subcutaneous fat and subsequent saponification (8,9,16).

Evaluation should consist of routine blood work looking for metabolic abnormalities as mentioned. Cultures of the wound and blood should be obtained and sent for routine and anaerobic cultures. If there is any

question about subcutaneous necrosis, the wound can be probed to ascertain the presence and extent of fascial involvement (9,10,11). Imaging studies that are helpful include plain soft tissue films of the neck looking for gas in soft tissues and retropharyngeal widening; chest x-ray to evaluate the mediastinum for widening and to look for pleural effusions and CT scanning which is probably the single most useful study because it can detect gas in areas inaccessible to palpation, identify areas where infection has spread preoperatively, and can detect vascular thrombosis, erosion of vessels, or mediastinitis (13,14,15).

These patients should be treated with broad spectrum antibiotics after cultures have been obtained (1,5,9,10). Penicillin is the drug of choice for streptococcus-group A, B, C, G, and H, *strep viridans* and most clostridia. Clindamycin is adequate therapy against anaerobes resistant to penicillin, and an aminoglycoside should be used to cover gram negative bacilli (1,3,4,12). Debridement of all necrotic tissue is the most important aspect in the treatment of these patients. Immediate surgical exploration is indicated in the presence of subcutaneous emphysema, rapidly advancing infection despite 24 to 48 hours of medical therapy, obvious fluctuance, or skin necrosis in an area of cellulitis (1,3,7,16). It is important to remember that fascial necrosis usually extends further than cutaneous involvement.

Several factors have been found to influence survival in necrotizing fasciitis. A delay of greater than 24 hours is associated with a much higher mortality rate (70%) than in those patients treated in less than 24 hours (36%). Diabetes mellitus and atherosclerosis are also associated with a much higher mortality rate and chronic renal failure, obesity, immunosuppression, and malnutrition have all been found to influence survival rates adversely (2,3,5,9,10). The complications that have been associated with necrotizing fasciitis of the head and neck include necrosis of the chest wall fascia, mediastinitis, pleural effusion, pericardial effusion, empyema, airway obstruction, arterial erosion, jugular vein thrombophlebitis, septic shock, lung abscess, and carotid artery thrombosis (9).

The most important aspects in the care of these patients are the early recognition and correction of metabolic abnormalities, broad spectrum antibiotic coverage, and early radical debridement of all necrotic tissue (1,3,7,8,9,16,17).

Material and Methods:

In the period between 1998-2005, four patients with diagnosed facial necrotizing fasciitis were managed in MKH in Kuwait three of these patients were initially treated in the general surgery unit, and only one patient referred to the OMF surgery unit and treated there by us.

Based on a comprehensive review of the Facial Necrotizing Fasciitis cases reported in the literature, and on our experience with facial NF and the results of all previous case reports ,the clinical manifestations, pathogenesis, and management of this disease are discussed.

Symptomatology:

The duration of symptoms ranges between one day and one week. all. patients presented with fever, mostly high grade with associated rigor. There was history of pain and swelling in the

region affected by most of the patient, so a diagnosis of cellulitis was made in the majority of cases.

Most of the patients had history of one form of trauma precipitating the infection. These range from a minor trauma or skin injury ,or an odontogenic source of infection. Four of the patients had a pre-morbid state which could have predisposed them to the infection. Our case was a known uncontrolled diabetis mellitus patient.



Fig.1 Patient by admission



Fig.2 The same patient few hours later



Fig.3 View intraorally

Microbiology:

Wound swabs for microscopy, aerobic culture and sensitivity was obtained from our patients. Organisms cultured were mainly gram negative aerobic bacilli. Most of the wound grew Klebsiella species, Proteus species, Escherichia coli and pseudomonas. Staphylococcus aureus was cultured in one of the patient.

Treatment:

Most of the patients presented with acute clinical conditions like fever, chills, rigor and pain. They were managed with intravenous fluids, antibiotics (mostly penicillin, clindamycin, and metronidazole) and analgesics. Most patients had extensive debridement with subsequent wound dressing before skin grafting. Examples are as shown in the clinical photographs.



Fig.4



Fig.5



Fig.6

Fig.4.5.6 The same case during surgery



Fig.7



Fig.8 Fig.7.8 Wound coverage with sufratuel

Hospital stay:

the duration of hospital stay ranges from four to six weeks. After surgical debridement, wound dressings were done until the wound has granulated and clean enough for skin grafting.

Outcome:

all of our four cases had survived the disease, showing good and acceptable, aesthetic and functional results.



Fig.9



Fig.10 Fig.9.10 Good final result

Discussion:

Most of our patients presented late to the hospital (between 1-3 weeks) and even at presentation, the diagnoses were missed in most cases (3,8). Diagnoses of cellulitis were freely made by the casualty officers and patients were commenced on antibiotics and bed rest only (9). Most of our patients had history of one form of trauma precipitating the infection. This tallies

with the study conducted by (8,16). Necrotizing fascitis is said to be common in the debilitated patient and is associated with high morbidity and mortality (1,3,7,8).

Two of our patients had diabetes mellitus, and another one had protein energy malnutrition. Several reports of necrotizing fascitis has implicated -haemolytic group A streptococci as the primary pathogens. The described group A streptococcal necrotizing fascitis with associated toxic shock is said to occur typically in healthy young subjects However, majority of cases represent a mixed synergistic infection involving both aerobes and obligate anaerobes (1,3,4,9,10,12). Organisms cultured in our patients were mainly gram negative aerobic bacilli such as Klebsiella species, Proteus species, Escherichia coli and Pseudomonas. Most of our patients presented with acute clinical conditions such as high grade fever, chills and rigors, tachycardia and pain as by other investigators (1,5,9,10). They were managed with medical therapy, none had intensive therapy support. Extensive debridement resulted in large surface wound in most cases. These wounds were dressed for some days before they were clean enough for skin grafting (8,16). Delay before a correct diagnosis was made as well as late presentation to the hospital contributed to high mortality. It is said that for necrotizing fascitis, if a regimen of early debridement and skin coverage is followed, and then mortality can be as low as 4%. If therapy is delayed or the debridement is inadequate, then mortality can be as high as 38%.

The earliest clinical clues to recognition of diffuse necrotizing infections are oedema out of proportion to skin erythema, gas in the subcutaneous tissues that may be identified as clinical crepitus and the presence of vesicles (1,5,7). If the early signs are missed, local skin anaesthesia and necrosis occur and systemic progression may present as fever resistant to antibiotic therapy and or hypotension. These findings should provoke prompt surgical exploration and administration of broad

spectrum antibiotic therapy. Doctors that first see these patients must take note of these diffuse clinical presentations. Early diagnosis, aggressive surgical intervention combined with supportive therapy is crucial to the successful treatment of the disease (1,2,3,4,8,9,10,16). Supportive treatment should be aimed at correcting hypovolaemia and systemic toxicity. The underling systemic condition must also be adequately treated. Skin grafting is often required after resuscitation and debridement.

Conclusion:

Subcutaneous Necrotizing Fasciitis is a rapidly progressive soft tissue infection with high morbidity and mortality, its occurrence in the head and neck region is uncommon, the majority of the reported cases being limited to involvement of the neck, usually from infections of dental or pharyngeal origin. invovement of the face from Necrotizing Fasciitis is rare. It is not only associated with a high mortality but can also result in severe disfigurement of the Face ,posing challenging reconstructive problems.

Differentiating NF of the face from benign inflammatory conditions such as cellulitis my be Impossible at an early stage. The differentiation is crucial so that treatment is not delayed ,and a high degree of suspicion may be the most important aid in early diagnosis.

Diagnosis of NF is clinical, patients are very ill with disproportionate pain and only minor skin changes in the early stages. also microbiological investigations are helpful to determine the Causative organisms, MRI or CT delineation of the extent of infection is very useful in directing rapid surgical debridement.

A prompt diagnosis, immediate administration of broad spectrum antibiotic and emergent aggressive surgical debridement of all compromised tissues are critical to reduce the high rate of mortality which is often greater than 25% if late or badly treated.

The role of immunoglobolins, and hyperparic oxygen is controversial, but have been shown to reduce the necessity for repeated debridements and improve survival.

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