Cervicofacial necrotizing fasciitis: can we expect a favourable outcome?

NARESH K. PANDA, M.S., D.N.B., F.R.C.S. (ED.), SRIDHAR SIMHADRI, M.S., D.N.B., SURYANARAYANA RAO SRIDHARA, M.S.

Abstract

Necrotizing fasciitis of the head and neck is an uncommon, progressive, destructive soft tissue infection of mixed aerobic and anaerobic organisms, having high mortality if left untreated (22 to 100 per cent). This study makes an attempt to analyse various factors and management methods determining the overall prognosis.

A retrospective analysis of all cases of necrotizing fasciitis involving the head and neck, with exclusion of those involving the eyelid and the scalp, was undertaken. Various parameters such as demography, aetiology, complications, management and outcome were studied.

Males outnumbered the females with the latter having a greater risk of involvement after 60 years. Odontogenic infection was the primary source of infection. Anaerobes were cultured in seven out of 17 cases, with six others showing mixed Gram positive and Gram negative organisms. Anaemia was the most commonly associated illness, with diabetes affecting four out of 17 cases. Aggressive surgical debridement with triple antibiotic therapy was used in the management of necrotizing fasciitis with an overall mortality of 11.8 per cent. Patients having late referral, anaemia and one or other complication had increased duration of total hospital stay.

Better results can be obtained with proper control of infection by early diagnosis, aggressive surgical debridement and triple antibiotic therapy, along with timely control of complications and associated illnesses.

Key words: Fasciitis, Necrotizing, Comorbidities; Antibiotics, Combined; Debridement; Treatment Outcome

Introduction

Necrotizing fasciitis is a severe, synergistic soft tissue bacterial infection causing necrosis of the superficial fascia and subcutaneous tissue, with sparing of skin and muscle. Initially called 'hospital gangrene' by Joseph Jones in 1871,¹ the condition is frequently associated with post-operative wounds of the perineum, abdomen and extremities. Meleney in 1924, considered subcutaneous necrosis as a pathognomonic feature of necrotizing fasciitis.² He noticed that haemolytic streptococci were frequent aetiological agents. Wilson was the first to use the term necrotizing fasciitis.³

Necrotizing fasciitis has a great propensity to involve the trunk, groin and lower limbs and rarely involves the head and neck. Necrotizing fasciitis of the head and neck commonly occurs secondary to dental infection but may follow insect bites, trauma or peritonsillar abscess. Systemic illnesses, such as diabetes and malnutrition, suppress the host immune system thus predisposing it to this rapidly progressive disease.

The condition is associated with extensive tissue necrosis and extreme systemic upset. Left untreated, necrotizing fasciitis is associated with a mortality of between 22 and 100 per cent.4,5,6 The keys to successful management include early diagnosis, broad-spectrum antibiotics, radical surgical debridement and nutritional support with management of complications.

This is a report of 17 cases of necrotizing fasciitis taking into consideration the various patient profiles, sources of infection, bacteriology, associated risk factors and ultimate outcomes along with the possible complications. An attempt was made to determine the factors responsible for a favourable outcome.

Material and methods

This is a retrospective study of all cases of cervicofacial necrotizing fasciitis (i.e. patients with involvement of face, neck and upper chest either in combination or in isolation) treated during the period between 1996 and

From the Department of Otolaryngology Head and Neck Surgery, Postgraduate Institute of Medical Education and Research, Chandigarh, India. Accepted for publication: 24 June 2004.

772



Fig. 1

CT scan taken at level of the mandible showing air within the soft tissue of the cheek (masseter muscle), a pathognomonic sign for necrotizing fasciitis.

2002 in the Department of Otolaryngology PGIMER, Chandigarh. Certain criteria were decided upon and followed in diagnosis of all the cases. The criteria included: 1) Fascial necrosis; 2) Undermining of skin; 3) Presence of gangrenous tissue; 4) Involvement primarily of face and neck; 5) Histopathological evidence of necrotizing inflammation; 6) Crepitus; 7) Characteristic computed tomography (CT) scan findings (Figure 1). Criteria 1, 2, 3 and 4 were considered mandatory for the diagnosis of necrotizing fasciitis. These were noted either by clinical examination or during surgery. The rest of the criteria were recorded and, if present, strengthened the diagnosis.

Details regarding the possible aetiology, presentation, associated illness, organisms involved, complications, management, total hospital stay, final outcomes and various other aspects (demography, source of infection and areas involved) were revised. Information was tabulated and analysed. All cases involving the scalp, and eyelid in isolation, were excluded from the study.

Results

Of the total 152 cases reviewed, only 17 cases were found to fulfil the criteria of necrotizing fasciitis. The average age was 47.71 years (range 30 to 80 years) with 10 cases less than 60 years. The male to female ratio was 1.43:1 (10 males and seven females). Five out of seven females (71.4 per cent) had an average age equal to, or greater than 60 years as compared to one out of 10 males (10 per cent) (Table I).

Source of infection and aetiology

Primary odontogenic infection was the single most common source of infection: 10 out of 17 (58.82 per cent) cases. One patient developed necrotizing fasciitis following peritonsillar abscess and one following an insect bite. Five had no obvious source of infection.

Microbiological examination of infected tissues showed *Staphylococcus aureus* in four cases, mixed aerobic Gram positive and Gram negative cocci in six cases, with five cases being sterile. *Klebsiella* sp. and *Acinetobacter lwoffii* were cultured in one case each. Seven out of 17 cases (41.2 per cent) showed anaerobic bacilli following culture (Table I).

TABLE I PATIENTS' PROFILES

No.	Age (yrs.)	Sex	Source of infection	Bacteriology		Prior to
				Aerobic	Anaerobic	treatment (days)*
1	42	М	Idiopathic	-ve	-ve	12
2	30	Μ	Dental	Staph. aureus	-ve	7
3	33	Μ	Idiopathic	Staph. aureus	-ve	3
4	45	Μ	Dental	Klebsiella	-ve	10
5	44	Μ	Dental	Mixed#		12
6	60	F	Dental	Mixed#		8
7	48	Μ	Dental	-ve	-ve	4
8	60	F	Dental	Acinetobacter lwoffli	-ve	4
9	63	Μ	Idiopathic	-ve	-ve	5
10	35	Μ	Peritonsillar abscess	Staph. aureus	-ve	5
11	80	F	Insect bite	Mixed#		10
12	32	F	Idiopathic	Mixed#		8
13	32	F	Dental	-ve	-ve	7
14	52	М	Dental	Mixed#		7
15	60	F	Dental	Mixed#		6
16	65	F	Idiopathic	Staph. aureus	+ve	7
17	30	Μ	Dental	-ve	-ve	8

* Prior to reaching our institution; M = male; F = female; -ve indicates negative; +ve indicates positive; # includes aerobic and anaerobic organisms.

Tracheostomy, Cases 7, 12 had nasotracheal intubation for 24 hours post surgery, M = muscle; gl = gland.

CERVICOFACIAL NECROTIZING FASCIITIS

TABLE II

SITES OF INFECTION			
Site involved	No. of cases involved	Percentage (%)	
Submandibular trians	gle 14	82.35	
Carotid triangle	13	76.47	
Submental triangle	8	47.06	
Carotid sheath	5	29.41	
Posterior triangle	5	29.41	
Parotid space	5	29.41	
Masseteric space	5	29.41	
Chest wall	5	29.41	
Upper mediastinum	4	23.5	
Para-pharyngeal spac	e 3	17.65	
Retro-pharyngeal spa	ace 2	11.76	
Pre-tracheal space	2	11.76	

Extent of the disease

All cases had involvement of the neck, with the submandibular triangle followed by the carotid triangle being the commonest sites. Spread to the carotid sheath, chest wall and upper mediastinum were noted in five cases each. The sites least involved included the retropharyngeal space and pretracheal area (two cases each). The parotid gland and masseteric space were involved in five cases (Table II).

Associated comorbidities

Of the 17 cases reviewed, 12 cases had an associated systemic condition (76.47 per cent): anaemia was the dominant finding with 10 out of 17 (58.82 per cent) presenting with haemoglobin less than 10 gm per cent: four out of 17 had diabetes mellitus (IDDM) with two cases presenting with ketoacidosis: four had a prior history of pulmonary disease: one each was associated with chronic renal failure, alcoholic addiction, HbsAg positive serology, and Arnold Chiari malformation (Table III).

Treatment modalities or therapy

Before reaching our institution. The average number of days that lapsed between the onset of symptoms and the treatment at our institute (a tertiary referral centre) was 7.24 days (range three to 12 days). Five patients received prior treatment either in the form of intravenous antibiotics or incision and drainage. Four patients received a blood transfusion for anaemia (Table I).

A multipronged approach was adopted to deal with this acute life-threatening disease at our institution (Table IV).

Therapy and management

Antibiotic therapy

Our initial drugs of choice were benzyl penicillin (2-4 million units/six hourly) along with a third generation cephalosporin (2 g i.v. twice a day) and metronidazole (500 mg i.v. thrice daily). Four cases with pulmonary complication were given amikacin (375 mg i.v. twice daily) or gentamicin (80 mg i.v. twice a day). The antibiotics were later modified as per the culture and sensitivity results.

Surgical management

All cases underwent aggressive surgical debridement, which included resection of all necrotic material (fascia. skin, muscles, glands, bone) utilizing fasciotomy incisions (one to multiple) parallel to the major blood vessels. Three cases required two or more major debridements, with one of them requiring three. Fourteen out of 17 required only one major debridement. All cases underwent multiple (on average range of two to eight) minor debridements. Five out the 17 patients (Cases 2,5,6,8,13) were tracheostomized to maintain the airway. Two patients (Cases 7,12) had nasotracheal intubations for 24 hours post-surgery after which they were successfully extubated (Table V). Split thickness skin grafting was used to cover the skin deficient areas in five cases. Most wounds (12 cases) healed by secondary intention.

Outcomes

Out of the 17 patients who underwent treatment for necrotizing fasciitis, only two (11.8 per cent) died of the disease. One had septicaemia with multiple organ dysfunction syndrome (MODS) and the other

	ASSOCIATED COMORBIDITIES	
Condition associated	Patient affected (case no.)	Number of cases
Diabetes mellitus	Cases 5,6,7,9	4 (23.53%)
Hypertension	Cases 1,9	2 (11.77%)
Pulmonary disease		4 (23.53%)
Chronic bronchitis	Case 7	1
Bronchial asthma	Case 3	1
Emphysema	Case 5	1
Pulmonary Koch	Case 4	1
Alcoholic addiction	Case 3	1 (5.88%)
Arnold Chiari malformation	Case 1	1 (5.88%)
Dermatological problems		2 (11.77%)
Acne vulgaris	Case 3	1
Vitiligo vulgaris	Case 4	1
Anaemia ($\leq 10 \text{ gm}\%$)	Cases 1,2,4,6,9,11,12,13,15,16	10 (58.82%)
Renal complication (CRF)*	Case 6	1 (5.88%)
Hbs Ag positive serology	Case 13	1 (5.88%)
No association disease	Cases 8,10,14,17	4 (23.53%)

TABLE III

*CRF = chronic renal failure

General assessment	Intravenous antibiotics	Surgery
Correction and control of: Fluid and electrolyte imbalance Protein deficiency Diabetes Hypertension Psychiatric problems <i>Radiology</i> Chest X-ray, CT scan	1st line: polymicrobial coverage Gram positive Gram negative Anaerobes Later on modified as per sensitivity and organism cultured	Extensive debridement Major Performed under G/A Radical in type Included resection of necrosed fascia, skin, glands, muscles and subcutaneous tissues Minor Performed under L/A Included irrigation with 1 per cent hydrogen peroxide Resection of necrosed tissue Thorough irrigation with antibiotic solution and isotonic saline initially 2 to 3 times a day for 5 days later on once daily Tracheostomy Emergency elective

 TABLE IV

 TREATMENT AT THE INSTITUTE OF MEDICAL EDUCATION AND RESEARCH, CHANDIGARH

succumbed to acute renal failure with azotemia following diabetic ketoacidosis. Uncontrolled diabetes mellitus with ketoacidosis was noted in both of these patients (Table V).

Complications

Local complications. Three cases had salivary gland necrosis (one case of the parotid gland; two cases of



Granulation of the wound 15 days following debridement of the necrotic tissue ready for skin grafting.

https://doi.org/10.1258/0022215042450698 Published online by Cambridge University Press

the submandibular gland) after the initial debridement. Four cases showed surrounding myonecrosis (three involving strap muscles and platysma; one masseter) (Figure 2) and one case developed a right-sided lateral pharyngeal wall (LPW) fistula following debridement. Skin necrosis occurred in all but in two cases. (Figure 3). Local haemorrhage was noted in two cases (*Cases 5* and 6), on both occasions originating from a necrosed facial artery that had to be ligated. One case each had external and internal jugular vein thrombosis (*Case 7*).

Thoracic complications. Superior mediastinitis was noted in four patients, with two of them developing pleural effusion. One patient each had pericarditis with effusion and pericardial abscess. Drainage of the pericardial abscess was carried out by pericardectomy. Intercostal drainage was used to treat pleural effusion in both cases.



Fig. 3

Partially granulating wound showing exposed mandible following masseter muscle necrosis and skin loss (four days following debridement).

Sitti	ngs					
No.	Major	Minor	Total Hosp. (days)	Complications	Outcome	Reconstruction
1	1	5	38	Massetric m. necrosis	Alive	STSG
2	2	7	18	Superior mediastinitis	Alive	STSG
3	1	5	9	Nil	Alive	Nil
4	1	8	36	Submandibular gland necrosis	Alive	STSG
5	1	2	1	Sepsis, DKA, MODS, ventilator support, haemorrhage, strap m. necrosis, superior mediastinitis, submand. gl. necrosis	Died after 1 day	Nil
6	1	6	5	Prerenal azotemia, uncontrolled DM, DKA ABE haemorrhage strap m necrosis	Died after 5 days	Nil
7	2	8	40	Mediastinal abscess, pericarditis with effusion, LPW fistula, pleural effusion, EJV, IJV, thrombosis superior mediastinitis	Alive	STSG
8	1	6	18	Nil	Alive	Nil
9	1	7	20	Nil	Alive	Nil
10	3	8	20	Pericardial abscess, pleural effusion, superior mediastinitis	Alive	Nil
11	1	6	27	ARF with ATN, CAD, LVF	Alive	Nil
12	1	8	32	(R) hip effusion, strap m. necrosis	Alive	STSG
13	1	7	22	Parotid gland necrosis	Alive	Nil
14	1	7	22	Epidural abscess	Alive	Nil
15	1	7	15	Nîl	Alive	Nil
16	1	7	10	Nil	Alive	Nil
17	1	5	10	Nil	Alive	Nil

TABLE V MANAGEMENT AND OUTCOME

STSG = Split thickness skin graft; MODS = Multi organ dysfunction syndrome; (R) = Right side;

ARF = Acute renal failure; LVF = Left ventricular failure; ATN = Acute tubular necrosis; CAD = Coronary artery disease; DM = Diabetes mellitus; LPW = Lateral pharyngeal wall; EJV = External jugular vein; IJV= Internal jugular vein thrombosis.*Note-*Cases 2,5,6,8,13* required tracheostomy, *Cases 7,12* had nasotracheal intubation for 24 hours post surgery; m. = Muscle.

Systemic complications. Septicaemia was diagnosed in one out of the 17 cases of necrotizing fasciitis. Acute renal failure was recorded in two cases, of which one had prerenal azotemia. One patient had a myocardial infarction with left ventricular failure while another developed epidural abscess. Diagnosis of right-sided hip effusion (probably of rheumatic origin) was made in one patient. The cases presenting later than six days of symptom onset had increased local and systemic complications (Table V).

Total hospital stay

Total admission days ranged from one to 40 days with an average of 20.18 days. Patients presenting after six days of initial disease onset had an average stay of 23.89 days (excluding two deaths) compared to those presenting earlier (20.33 days). Two deaths (*Cases 5* and 6) that occurred reported on eight and 12 days after the onset of symptoms. Cases having one or other complications had a longer stay (mean 23.6 days) compared to those without (16.9 days). Patients presenting with anaemia at the initial stage had a longer stay than those without it (five days more) (Table V).

Discussion

Necrotizing fasciitis is a progressive, rapidly spreading soft tissue infection, eventually resulting in septicaemia, multiorgan dysfunction and ultimately death if inadequately treated. Involvement of the head and neck is fortunately rare. Rarity of the condition can be reflected by the fact that only 67

https://doi.org/10.1258/0022215042450698 Published online by Cambridge University Press

cases were reported between 1945 and 1990.³ After 1990, increased awareness of the condition resulted in more reports of necrotizing fasciitis appearing in literature. Stone and Martin in 1972⁷ reviewed 63 cases of necrotizing soft tissue infection and found only two involving the neck. Lin *et al.* identified 47 cases involving the head and neck region during their 12 year analysis.⁸ Bahu *et al.*⁹ identified and treated 10 out of 250 cases reviewed over an 11-year period. In this study, we analysed 17 cases primarily involving the face and cervical region while excluding those of eyelids and scalp.

The average mean age noted in this series was 51 years with a median age of 45, which was in similarity to the mean age of NF involving other areas of the body (50.8 years for non-survivors and 43.8 for the survivors).¹⁰ Although males outnumbered the females as a whole (1.4:1), women were predominantly affected after the age of 60 with five out of six cases involved.

In agreement with other studies,^{11–15} primary dental infection remains a major cause of the condition affecting 59 per cent of cases (10 out of 17 cases). Five cases were considered idiopathic. Haematogenous spread or bacterial invasion through small unnoticed breaks in epidermis were considered the probable mode of transmission in these cases.¹⁶ Less common aetiologies such as insect bites,^{17,18} hypodermic needle infection,¹⁹ trauma and peritonsillar abscess²⁰ have occasionally been described as a source of the severe soft tissue infection.

Necrotizing fasciitis was originally believed to be secondary to group A streptococcal and staphylococcal infections. With the advent of more sophisticated anaerobic culture techniques, the concept of a synergistic combination of aerobic and anaerobic infection is considered more appropriate.²¹⁻²⁴ In this study only seven out of 17 cultures showed more than two organisms. Pure cultures were noted in five cases, of which three grew Staphylococcus aureus and one Klebsiella sp. and one Acinetobacter sp. Interestingly, none of the cultures grew group A streptococci, which were considered major organisms by Meleney.¹ Many factors ranging from technique of collection to the identification of organisms and prior antibiotic therapy could explain the sterile cultures noted in five out of 17 cases (29.41 per cent).

- Necrotizing fasciitis in the head and neck has been reported previously in this and other journals
- This paper is a retrospective study of 17 cases
- In this paper the authors discuss their surgical approach to debridement and the factors that they felt were important in the pathogenesis and management of the disease in their patients

Necrotizing fasciitis is often confused with cellulitis and erysipelas in its early stage. Pain, swelling, warm erythematous and oedematous skin often confuse the diagnosis, thus delaying active management. The infection spreads by necrosis of subcutaneous tissue and superficial fascia sparing skin and muscle in the initial stages. To determine the distribution and areas commonly involved, we sectored the face and neck into respective spaces and triangles. Submandibular and carotid triangles were most commonly involved and pretracheal and paratracheal spaces were the least commonly involved. The explanation for this may be that the former are the major areas of lymphatic drainage from the commonest source. Despite the involvement of the carotid triangle in three quarters of the cases, a breach into the carotid sheath was noted in only five cases, which confirmed the protective barrier provided by the carotid sheath.

In a study involving 47 cases of necrotizing fasciitis, Lin *et al.*⁸ observed diabetes mellitus as the most commonly associated disease affecting 72.3 per cent of cases. Hypertension, alcoholism and obesity were the other common systemic disorders considered to compromise the body's immunity.^{4,25} Out of the 17 patients studied, only four had type 1 diabetes, and two hypertension, with no case of obesity. Besides these, a significant number of cases (10 out of 17) had anaemia at the time of presentation. Its importance is reflected by the fact that anaemia results in poor wound healing and decreased body resistance to the disease thus increasing the total duration of the hospital stay by five days.

Aggressive surgical debridement, triple antibiotic therapy covering the Gram positive, Gram negative and anaerobic organisms, along with improvement in general status remain a cornerstone in the management of necrotizing fasciitis (Figure 3). Benzyl penicillin is still the first drug of choice along with third generation cephalosporins and metronidazole. This is in agreement with several reports of the success of penicillin in the treatment of the condition.^{17,18} Aminoglycosides, for better Gram negative coverage, were considered only in patients with associated pulmonary diseases.

Excision and aggressive debridement of fascia, skin subcutaneous tissues along with involved glands and muscles remain an important step in the management. Stock *et al.*,⁴ Bahu *et al.*⁹ and others^{8,22,25–27} advocated multiple debridements for a better outcome. In contrast to these, in our institute, much stress is laid on single major debridement followed by multiple minor debridements. In our study only three out 17 cases required more than one major debridement. Minor debridements were done under local anaesthesia, which were repeated twice a day in the initial period and later once daily, yielding equally good results and avoiding repeated general anaesthesia exposure.

Literature quotes an overall mortality of between 22 per cent and 100 per cent.^{4,5,6} Bahu et al.⁹ put the figure at 10 per cent in uncomplicated cases and 20 per cent following thoracic complications. Ndukwe et al.²⁴ in their series considered multiple factors such as pre-existing systemic illness, late surgical intervention, septicaemia within 24 hours, mediastinal and thoracic extension of the infection as responsible for an increase in the overall mortality. Wenig et al.²⁰ speculated that diabetes mellitus and impaired immune competence were important factors in decreasing the survival rate. In our study only two out of the 17 cases studied, died of the disease placing the overall mortality at 11.8 per cent. Uncontrolled diabetes with ketoacidosis was noted in two out four (50 per cent). This might have aggravated the primary disease by causing suppression of the immune system thus leading to septicaemia and multiorgan failure, strengthening the speculations of Wenig et al.²⁰ All four patients diagnosed of having one or other thoracic complication (mediastinitis, pleural effusion, pericarditis or pericardial effusion) recovered without sequelae, thus contradicting the findings and theories of Bahu et al.9 and Ndukwe et al.24 Anticipation of the problem, early recognition and active intervention play a vital role in management of thoracic complications.

Complications range from simple skin and muscle necrosis (Figure 2) to septicaemia and multi-organ failure. Vascular complications such as facial artery necrosis and jugular vein thrombosis were rarely reported in the literature. Catastrophes related to the carotid artery such as aneurysm, rupture, thrombosis with hemiplegia²⁸ were not recorded in our series. The longer the period that elapsed between onset and surgical intervention, the higher the complication rate. This fact was strengthened by our study, which noted that a delay of more than six days after the onset of symptoms, increased the local and systemic complications. Tracheostomy may be avoided in most of the cases but could be considered in cases with mediastinal or thoracic complications. Early ambulation along with proper chest physiotherapy helps minimize pulmonary complications.

Conclusion

Favourable outcome can be anticipated in necrotizing fasciitis by using an aggressive medical and surgical approach. Old age, female sex (especially age > 60 years), uncontrolled diabetes mellitus, anaemia, coexistent pulmonary diseases, delayed referral (greater than six days) and late surgical intervention are some of the factors, that determine the ultimate prognosis. Thoracic involvement is not as important a factor as was believed, although it may prolong the hospital stay. identification and Early prompt surgical intervention in the form of major and minor debridements, along with anticipation and early recognition of possible complications and comorbidities help decrease mortality and morbidity thereby providing a better prognosis.

References

- 1 Jones J. Investigation upon the nature, causes and treatment of hospital gangrene as it prevailed in the confederate armies 1861–1865. In: Hamilton FH, ed. *Surgical Memoirs of the War of Rebellion*. Riverside, N.Y: Hurd and Houghton, 1870–1871;146–70
- 2 Meleney FL. Hemolytic streptococcal gangrene. *Arch Surg* 1924;**9**:317
- 3 Banerjee AR, Murty GE, Moir AA. Cervical necrotizing fasciitis: A distinct clinicopathological entity? *J Larynol Otol* 1996;**110**:81
- 4 Stock CR, Winstead W, Marinez SA. Oodontogenic necrotizing fasciitis in Ile-Ife, Nigeria. Br J Oral Maxillofac Surg 2002;40:64–7
- 5 Spankus EM, Flint PW, Smith RJ, Miller RH. Craniocervical necrotizing fasciitis. Otolaryngol Head Neck Surg 1984;92:261–5
- 6 Rouse TM, Malangoni MA, Schulte WJ. Necrotizing fasciitis: a preventable disaster. Surgery 1982;92:765–70
- 7 Stone HH, Martin JD. Synergistic necrotizing cellulites. Ann Surg 1972;**175**:702–10
- 8 Lin C, Yeh FL, Lin JT, Ma H, Hwang Ch, Shen BH, *et al.* Necrotizing fasciitis of the head and neck: An analysis of 47 cases. *Plast Reconstructr Surg* 2001;**107**:1684–93
- 9 Bahu SJ, Shibuya TY, Meleca RJ, Mathog RH, Yoo GH, Stachler RJ, et al. Craniocervical necrotizing fasciitis: An 11year experience. Otolaryngol Head Neck Surg 2001; 125:245–52
- 10 Francis KR, Lamaute HR, Davis JM, Pizzi WF. Implications of risk factors in necrotizing faciitis. Am J Surg 1993;9:304–8
- 11 Skorina J, Kaufman D. Necrotizing fasciitis originating from pinna perichondritis. *Otolaryngol Head Neck Surg* 1995;**113**:445–9

- 13 Henrich De, Smith TL, Shockley WW. Fatal craniocervical necrotizing fasciitis in an immunocompetent patient: a case report and literature review. *Head Neck* 1995;17:351–7
- 14 Mathieu D, Neviere R, Teillon C, Chagnon JL, Lebleu N, Wattel F. Cervical necrotizing fasciitis, clinical manifestation and management. *Clin Infect Dis* 1995; 21:51–6
- 15 Zbaren P, Rothen HU, Lang H, Becker M. Necrotizing fasciitis of the soft tissues of the face and neck [in German]. *HNO* 1995; **43**:619–23
- 16 Helmy AS, Salah MA, Nawara HA, Khatab H, Khalaf HA, Abd el-Magiud N. Life threatening cervical necrotizing fasciitis. J R Coll Surg Edinb 1997;42:410–13
- 17 Balina M, Canalis RF. Necrotizing fasciitis (streptococcal gangrene) of the face. *Arch Otolaryngol* 1980;**106**:648
- 18 Javenicus RV, Hann SE, Batt MD. Necrotizing fasciitis. Surg Gynecol Obstet 1982;154:97
- 19 Gallia LJ, Johnson JT. Cervical necrotizing fasciitis. Otolaryngol Head Neck Surg 1981;89:935
- 20 Wenig BL, Shikowitz MJ, Abramson AL. Necrotizing fasciitis as a lethal complication of peritonsillar abscess. *Laryngoscope* 1984;94:1576–9
- 21 Moss RM, Kunpittaya S, Sorasuchant A. Cervical necrotizing fasciitis: An uncommon sequela to dental infection. *Ann Otol Rhinol Laryngol* 1990;**99**:643–6
- 22 Stephens BJ, Lathrop JC, Rice WT, Gruenberg JC. Fournier's gangrene: historic (1764–1978) versus contemporary (1979–1988) differences in etiology and clinical importance. Am Surg 1993;59:149–54
- 23 Elliot D, Kufera JA, Myers RAM. The microbiology of necrotizing soft tissue infections. Am J Surg 2000; 179:361-6
- 24 Ndukwe KC, Fatusi OA, Ugboko VI. Craniocervical necrotizing fasciitis in Ile-Ife, Nigeria. Br J Oral Maxillofac Surg 2002;40:64–7
- 25 Krespi YP, Lawson W, Blaugrund SM, Biller HF. Massive necrotizing infection of the neck. *Head Neck Surg* 1981; 3:475
- 26 Nallathambi MN, Ivatury RR, Rao PM. Craniocervical necrotizing fasciitis: critical factors in management. *Can J* Surg 1987;**30**:61–3
- 27 Majeski JA, Alexander JW. Early diagnosis, nutritional support and immediate extensive debridement improve survival in necrotizing fasciitis. Am J Surg 1983;145:784–7
- 28 Bush JK, Givner LB, Whitaker SH, Anderson DC, Percy AK. Necrotizing fasciitis of the parapharyngeal space with carotid artery occlusion and acute hemiplegia. *Pediatrics* 1984;73:343–7

Address for correspondence: Dr Naresh Panda, M.S., D.N.B., F.R.C.S. (Ed.) Department of Otolaryngology, PGIMER, Chandigarh 160012, India.

E-mail: npanda59@yahoo.co.in

Dr N. Panda takes responsibility for the integrity of the content of the paper. Competing interests: None declared