



Necrotizing Fasciitis

S. J. Fratesi MD





N. (*Northern*)

O. (*Ontario*)

O. (*Operating*)

R. (*Room*)

I. (*Interest*)

G. (*Group*)



Sponsored by The Sault Areas Hospitals, Sept.20,2003



Hippocrates..5th century BC

“ The erysipelas would quickly spread in all directions.Flesh ,sinews, and bones fell away in great quantities.”



J. Jones, US Confederate army 1871

..”I have seen the skin in the affected spot melt away in twenty-four hours into a grayish and greenish slough.”

British tabloids, Gloucestershire, 1994

“galloping gangrene”, “killer bug”, “flesh –eating bacteria”



“To go where no man
(person) has gone before”

Capt. James T Kirk
(USS Enterprise)



The local heat

HEALTH CARE

Province prepared to help as SAH deals with flesh-eating disease

Local medical officials made no request for provincial assistance — ministry spokesma

Flesh-eating disease cases make Sault residents wary

By JEFFREY OUGLER
The Sault Star





*THE SAULT STARJanuary
24,2001*

....although doctors are much more up to speed on the disease than they were a decade ago, the nature of the symptoms and the fact physicians might see merely one case during the course of their careers more often than not makes necrotizing fasciitis a mystery (Dr.Don Low, chief of microbiology at Mount Sinai Hospital,Toronto)



Group A Streptococcus confirmed isolates(SAH)

06/6/00	scrotal gangrene/septic shock
26/9/00	septic shock/leg ulcer ..died
28/9/00	post-op malaise/infected incision
21/12/00	infected hand
27/12/00	cellulitis arm
30/12/00	hip pain/septicemia ... died
18/01/01/	sepsis/arm(L)/leg®/finger® ... died



Necrotizing fasciitis

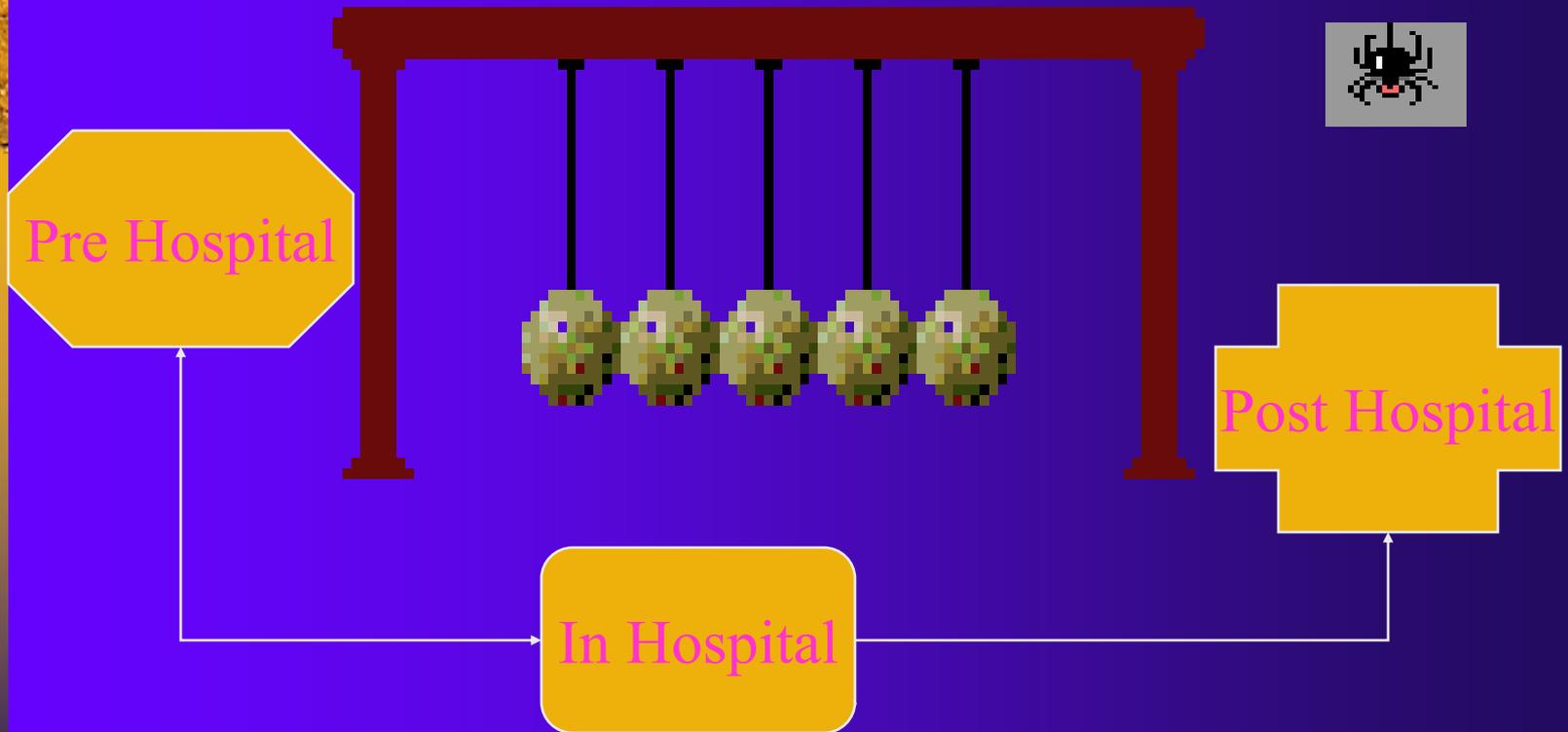
- DELAY in DIAGNOSIS increases mortality and morbidity
- The importance of clinical suspicion
- The importance of early radical surgical debridement
- The importance of specific antibiotic coverage and supportive therapy



Approach to soft Tissue infection (Scientific American 2000)

- 1) Is infection present?
- 2) Is there an underlying condition that favors infection?
- 3) Should antibiotics be given?
- 4) Which antibiotics are most appropriate?
- 5) Is surgical treatment required?

The chain reaction of a hospital/community crisis



Necrotizing Fasciitis

- ◆ Described for over 100 years
- ◆ Bouchard , the premier of Quebec
- ◆ True world incidence not really known.
- ◆ Male/female 2:1
- ◆ Mortality some series-70%(if associated with myonecrosis)
- ◆ In Canada there are 90-200 cases/year and as of 2000 is a reportable disease (provincially since 1998)
- ◆ Sentinel Health Unit Surveillance System a branch of Canada's Laboratory Centre for Disease Control





Necrotizing Fasciitis

Meleney's synergistic gangrene	Hemolytic streptococcal gangrene	War gangrene	acute dermal gangrene
Hospital acquired gangrene	Fournier's gangrene	Synergistic necrotizing cellulitis	Gas gangrene



Necrotizing Fasciitis

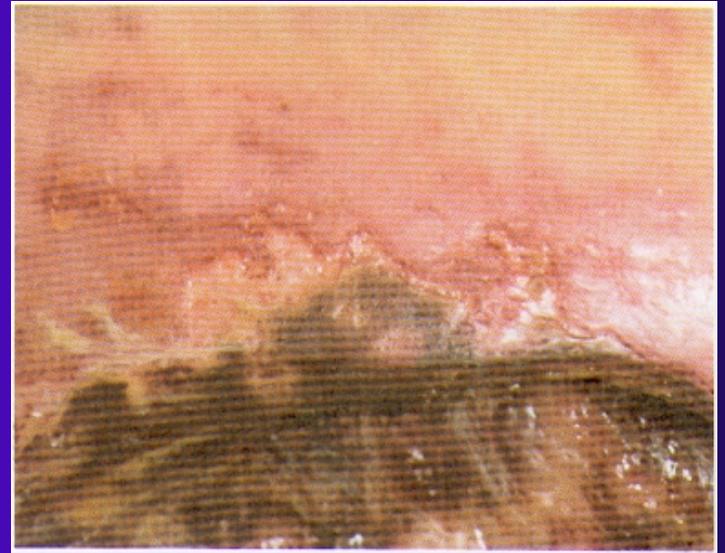
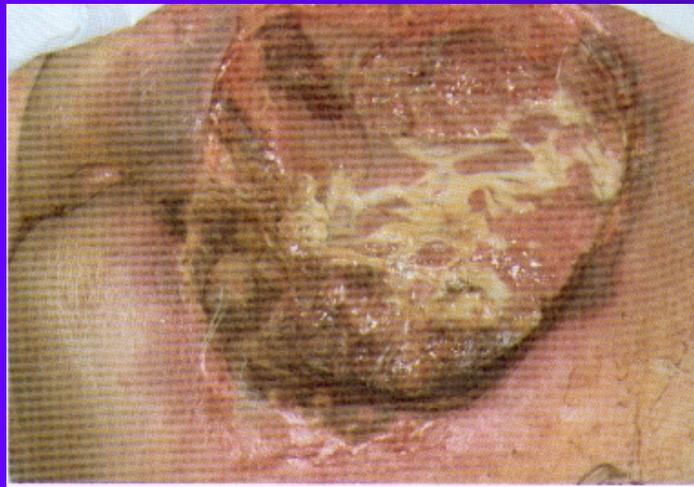
- ◆ Definition: a progressive, rapidly spreading inflammatory infection involving the superficial/deep fascia with secondary necrosis of the subcutaneous tissue/ skin and occasionally underlying muscle(associated myonecrosis)
- ◆ Fever, severe pain and a severe rapidly spreading red swelling +/- dishwater discharge



Necrotizing Fasciitis

- ◆ Type 1.....most common...mixed aerobic and anaerobic bacteria
- ◆ Type 2.....group A strept +/- staph aureus. This form may develop into a streptococcal myonecrosis and toxic shock syndrome
- ◆ There is often confusion with Clostridial myonecrosis(gas gangrene)

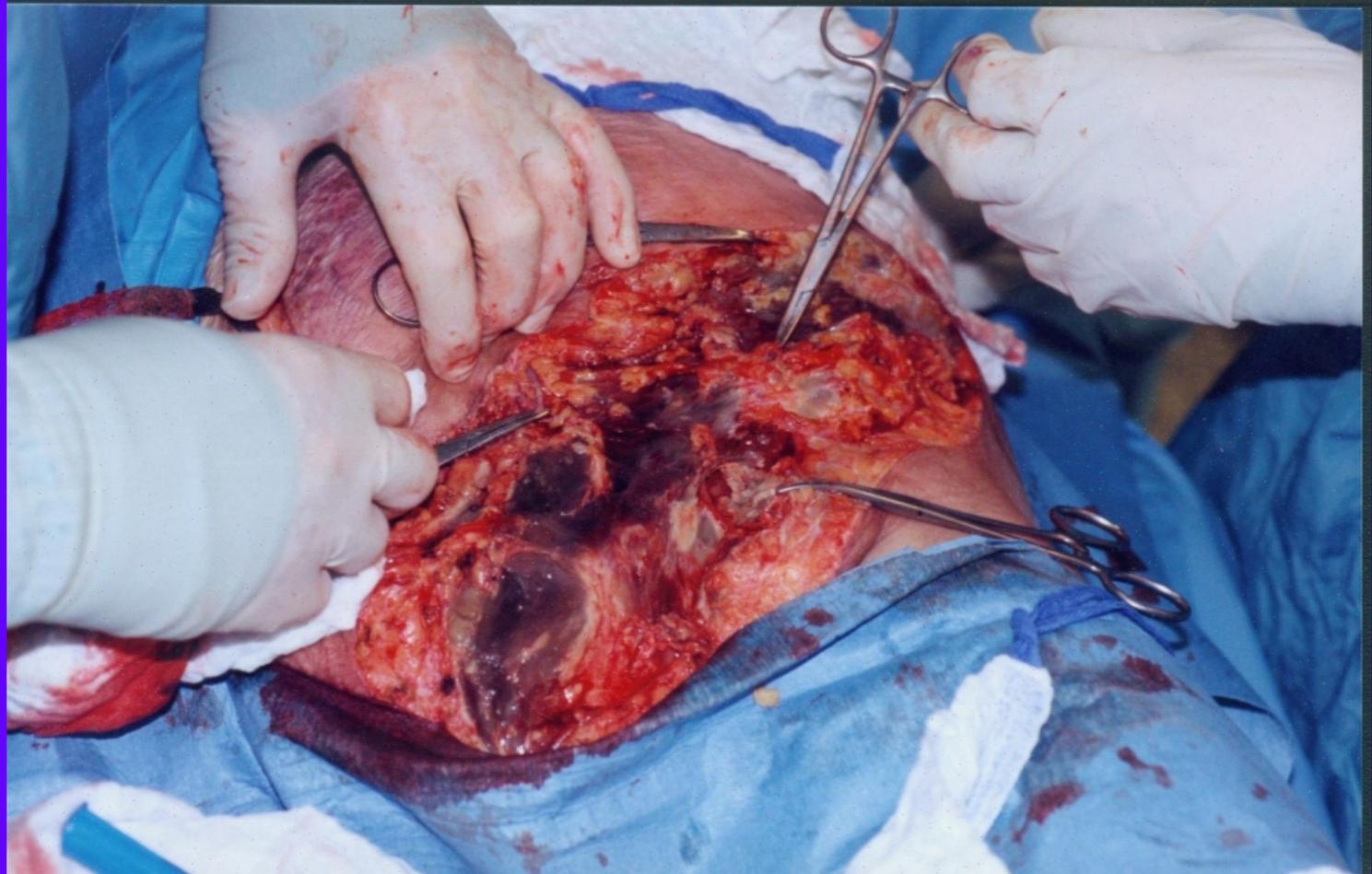
*Synergistic gangrene in
pressure sore(SA)*



Non-surgical necrotizing fasciitis
(gas gangrene due to *Klebsiella*)



Post-op necrotizing fasciitis





There's a lot of stuff to digest.. so let's go !

Gas gangrene... Clostridial sepsis



A



B



C



Clostridial gas gangrene

A



B



Clostridial gas gangrene

A



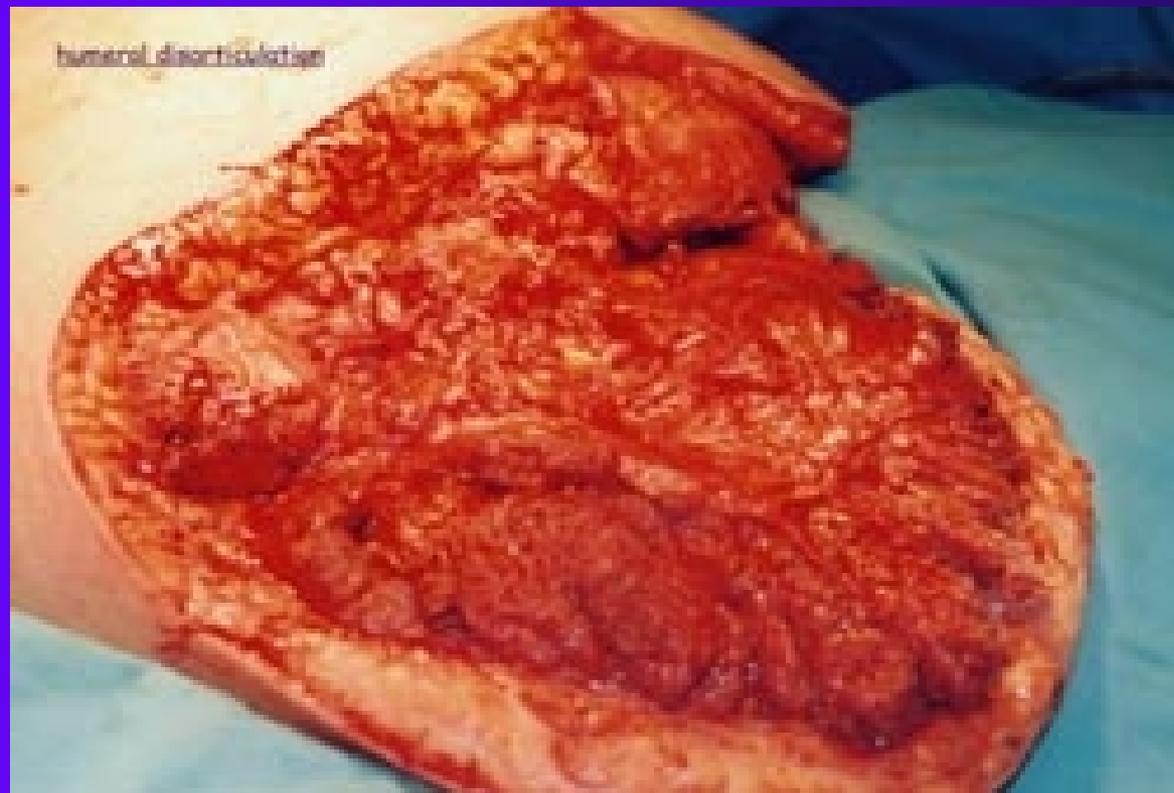
B



C



Clostridial gas gangrene...shoulder disarticulation





Gas Gangrene

- ◆ Damage caused by exotoxin formed by clostridium (large gm+rod, spore forming bacillus)
- ◆ Anaerobic, source gi tract or soil contamination
- ◆ Traumatic-most common, clostridium perfringens (inoculation of contaminated ischemic wound)
- ◆ Spontaneous (cl. septicum), compromised patient (malignancy, alcohol, diabetes, pvd)





Gas Gangrene

- ◆ US-1000 cases /year
- ◆ Traumatic-*Cl.perfringens*-25% mortality
- ◆ Spontaneous-*Cl. septicum*- 65-100%
- ◆ It is the exotoxin not bacteria which causes the spread leading to muscle destruction and anaerobic environment
- ◆ Secondary toxicity due to CPK,myoglobin and K^+

Gas Gangrene

- ◆ Pain, crepitus, warm skin, drainage, bronze skin, bullae, ischemic muscle
- ◆ *C. perfringens* more aerotolerant and can invade normal viable tissue
- ◆ Early diagnosis on gram stain - pleomorphic gm⁺ rods with few leukocytes
- ◆ Work up/Therapy essentially the same as NF



Flesh-eating disease



Flesh-eating disease...initial assessment





Flesh-eating disease



Flesh-eating disease (Group A hemolytic streptococcus)



Flesh-eating disease



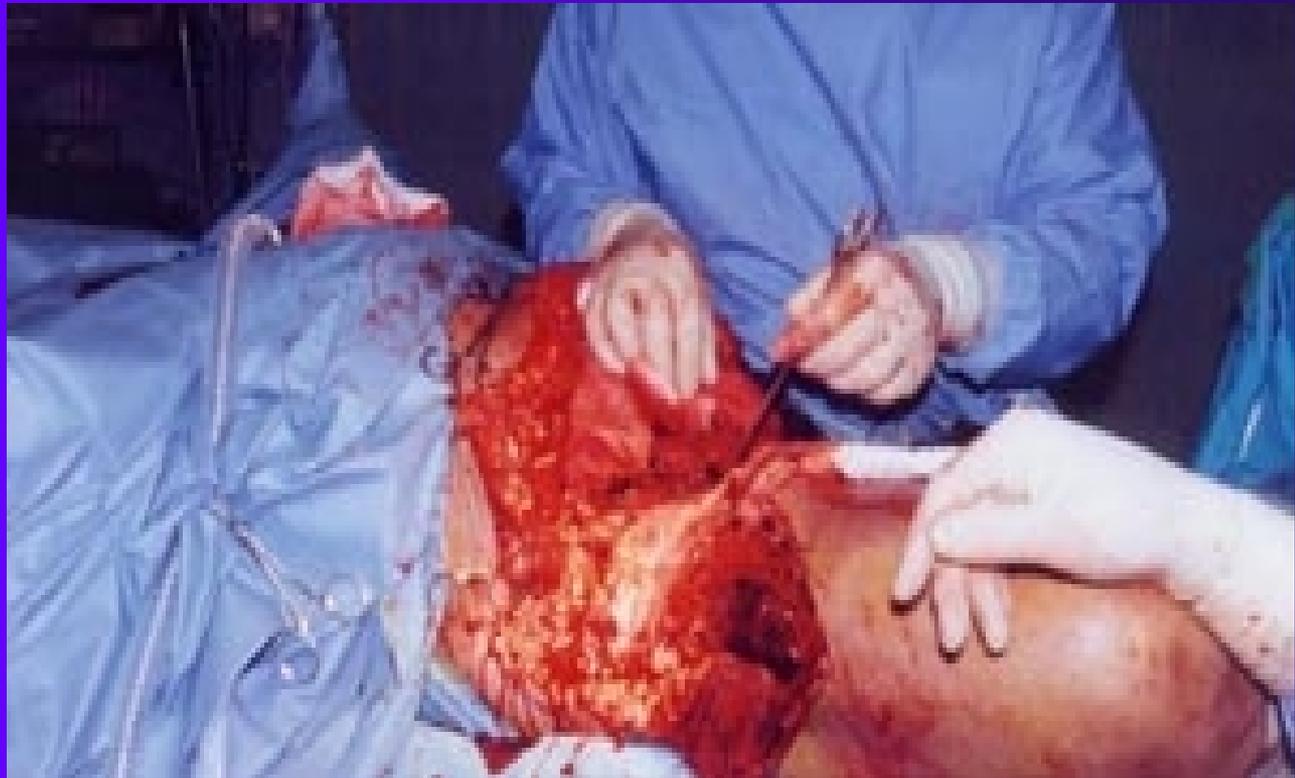
Flesh-eating disease



Vesicles/bullae of hemolytic eruption



Flesh-eating disease..hip disarticulation





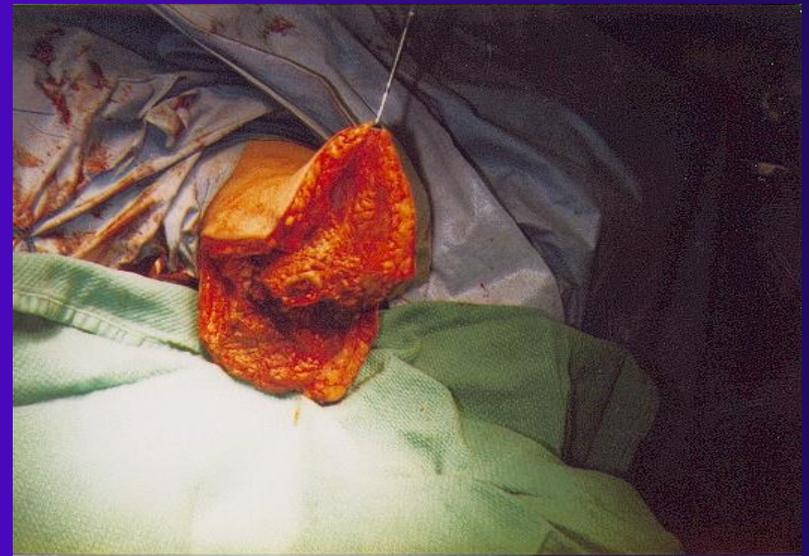
flesh eating disease



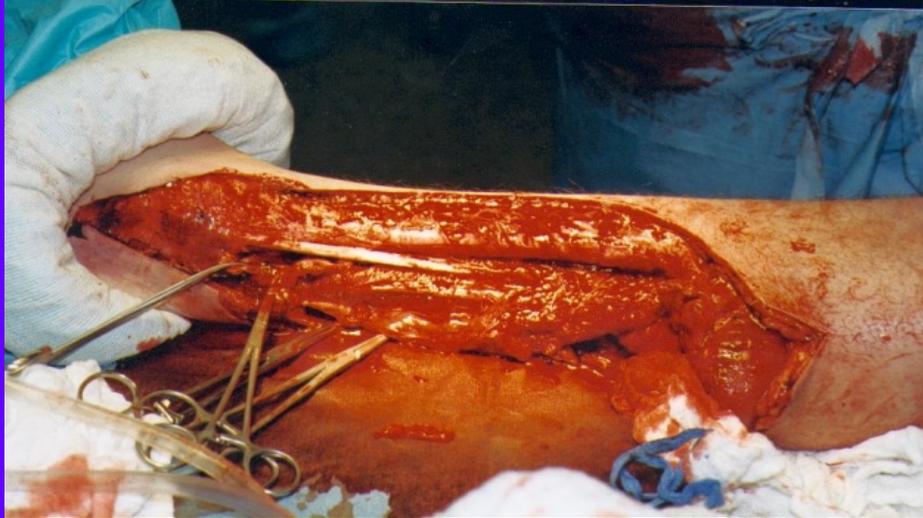
flesh eating disease



flesh eating disease



flesh eating disease







trauma

*Foreign
body*

**Necrotizing
Fasciitis**

*Immune
deficient*

*Idiopathic
(healthy)*

Aerobic , anaerobic, mixed bacterial organisms



What bugs are involved ?

Group A hemolytic

streptococcus

Staph aureus

Bacteroides

Clostridium

E.coli

Proteus

Pseudomonas

Klebsiella

Peptostreptococcus

Enterobacter



*Leukocytes (PMN)
show decreased
function in hypoxic
wound*

*Stimulates growth of
facultative anaerobic
bacteria*

*Decrease in
oxidative/reduction
potential*

*Anaerobic bacterial
proliferation*





NF increase with decrease in immune competence

- ◆ Diabetes
- ◆ Chronic renal failure
- ◆ Organ transplantation
- ◆ HIV patient
- ◆ Cancer
- ◆ Alcoholism
- ◆ Hepatitis
- ◆ Neutropenic syndrome(collagen disease/blood dyscrasia)



Necrotizing fasciitis

- ◆ May present with fever, malaise /flu-like symptoms
- ◆ Tissue necrosis/discharge
- ◆ Vesicles/bullae
- ◆ Severe pain
- ◆ Gas formation
- ◆ Burrowing through fascial planes
- ◆ Lack of classical signs of inflammation
- ◆ If left untreated may advance to myositis/myonecrosis



Necrotizing Fasciitis

- ◆ There may be no antecedent history
- ◆ may have had recent trauma or surgery
- ◆ Pain/ swelling are primary features particularly pain out of proportion to clinical findings.
- ◆ Within hours the are may become totally anaesthetic
- ◆ Presentation may be very subtle or a florid toxic shock syndrome

Fournier's disease

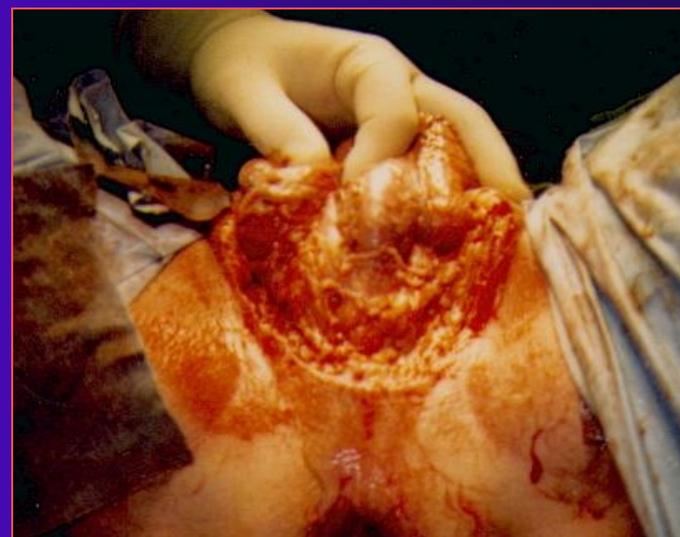
- ◆ Significant association with diabetes
- ◆ Pain , erythema, swelling of scrotal skin
- ◆ Necrosis of scrotal fascia / enlarged scrotal size
- ◆ Crepitus in 50%
- ◆ May be indolent(2-7 days)
- ◆ If advanced beyond scrotum behaves like all other necrotizing fasciitis
- ◆ Female variant is classical necrotizing variant because of thicker/denser tissue involvement



Fournier's disease



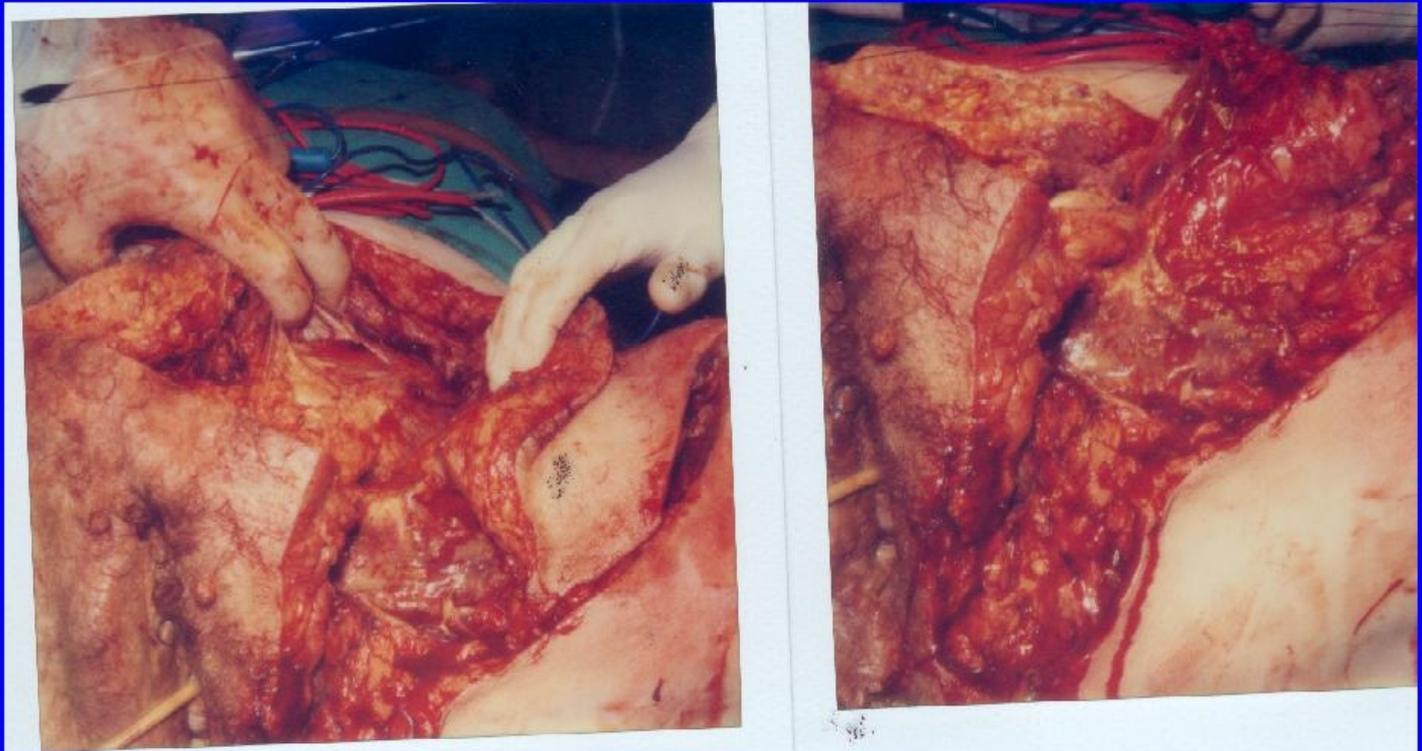
Fournier's disease



Fournier's disease(female)



Fournier's disease(female)



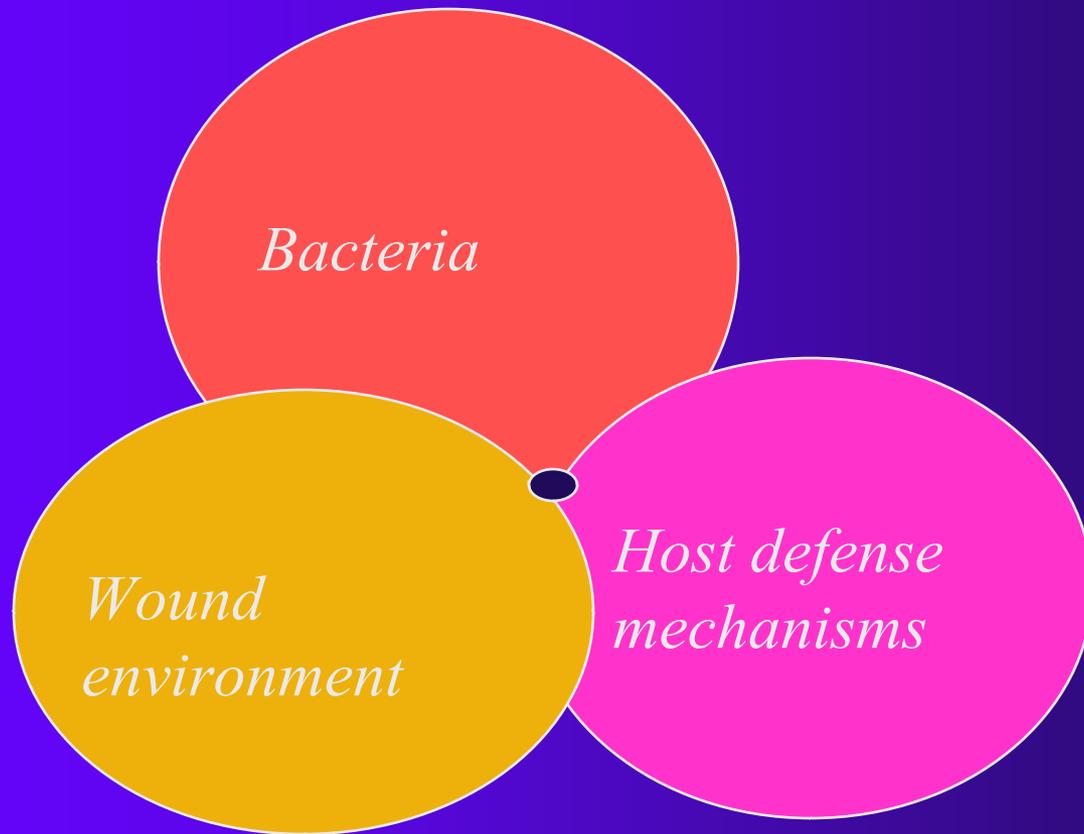


The micro-organism known as Bacteria

- Complex structure with a variety of proteins, nucleic acids, polysaccharides and lipids
- The main activity is reproduction
- The precise needs of the organism depends mainly on the enzymes it possesses



Homeostatic state



The determinants of any infectious process





Oxygen requirements of bacteria

- ◆ Aerobes
- ◆ Obligatory aerobes
- ◆ Facultative anaerobes
- ◆ Obligatory anaerobes
- ◆ Micro-aerophiles



Pathogens

- Relatively newHIV
- Old ,but recently identified as important...H.pylori
- Old but” smarter “leading to increased virulence and antibiotic resistance.....penicillin resistant pneumococcus
 -emerging/resistant TB
 - ...methicillin resistant s. aureus..
 - Vancomycin resistant enterococcus
 - ... Virulent group A streptococcus



GAS (group A Streptococcus)

- ◆ 10/20 cases /100,000(Canada 2-3/1,000,000)
- ◆ Heightened public interest.?epidemic
- ◆ 1600's,Italy ,Spain,N. Europe
- ◆ 1700's,American colonies
- ◆ 1940's,rheumatic fever
- ◆ 1950's post strept glomerulonephritis

- ◆ Before the era of antibiotics,breakouts due to change in socioeconomic conditions and variation in the organism virulence

G.A.S

- ◆ 80 M types
- ◆ 5 separate toxins-scarlatinic/ pyrogenic
- ◆ Changes in antigens and virulence account for cyclical resurgence
- ◆ STS syndrome(streptococcal toxic shock) affects all ages with no predisposing factors unlike the GAS bacteremia. There is 20-70% mortality with associated soft tissue infection, ARDS, renal / hepatic failure
- ◆ ACQUISITION of Strept A-50% unknown
- ◆ STREPT A PHARYNGITIS rarely if ever a cause



G.A.S.

- ◆ Group A streptococcus major clinical isolates include M types 1, 3, 12 and 28
- ◆ M type 1 has aggressive pyrogenic toxins A & B (most common)
- ◆ M type 3 recently described in “superantigen” reactions



G.A.S.

- ◆ NF....type 1..polymicrobial ,skin ,rectum and urethra
- ◆ ...type 2..GAS
- ◆ GAS..... Ontario 1991-95. Increase from .085/100,000 to .40/100,000
- ◆ Virulence from pyrogenic exotoxins which stimulate Tumor Necrosis factor and interleukin plus impede phagocytosis and promote”superantigen” response



The Toronto Star, Feb 12, 1995

Where the bugs struck in Ontario

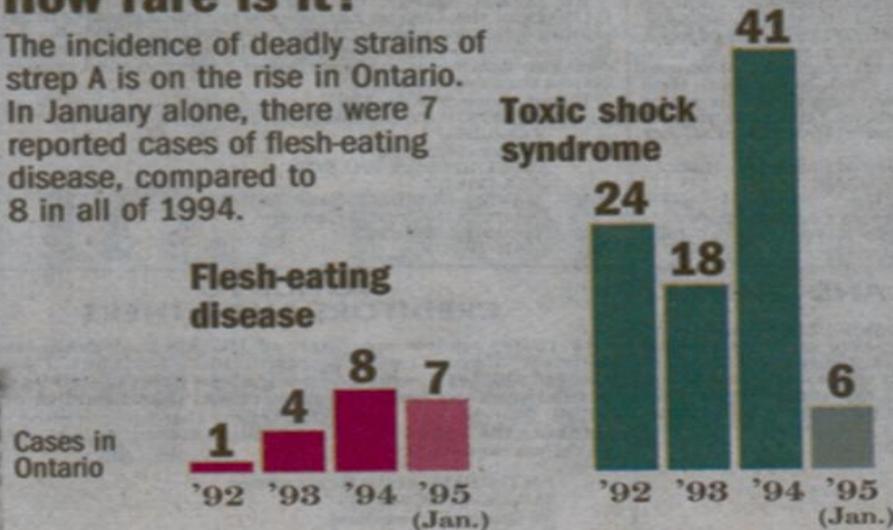
From Oct. 30 to the end of January, Ontario has seen 25 cases of group A streptococcus: 12 had flesh-eating disease, 10 had toxic shock and 3 had both. Nine people died.



Date	Place	Male/ female	Age	Flesh eating	Toxic shock	Died
Oct.30	St. Catharines	M	81		●	■
Nov. 6	Toronto	M	n/a	●	●	n/a
7	Stratford	M	81		●	■
20	North Bay	F	6	●		
29	Scarborough	M	17		●	■
30	Scarborough	M	38	●		
Dec. 2	Newmarket	M	49	●		
11	Sault Ste. Marie	F	55		●	
12	Toronto	M	34	●		
17	Kitchener	M	5	●		
18	Stratford	F	87		●	■
20	Orillia	M	38	●	●	
26	Belleville	M	58	●		
28	Oshawa	M	30	●		
28	St. Catharines	M	37		●	■
30	Midland	M	79		●	■
30	Kitchener	M	7	●		
Jan. 2	Oakville	F	95	●	●	
3	Kenora	M	32	●		
3	North Bay	F	76		●	■
4	London	F	84	●		
11	Hamilton	M	31	●		
25	Oshawa	M	72		●	■
26	Toronto	M	69	●		
31	Toronto	M	80		●	■

How rare is it?

The incidence of deadly strains of strep A is on the rise in Ontario. In January alone, there were 7 reported cases of flesh-eating disease, compared to 8 in all of 1994.





G.A.S.

- ◆ Streptococcus is a gram+ bacteria found in chains
It is a fermentative (anaerobic) organism which is oxygen tolerant. It is classified by its cell wall carbohydrate.
- ◆ Strept Aenzymes that break down tissue host
- ◆ Streptokinases which dissolve clot
- ◆ Cytolysins which kill leukocytes
- ◆ Hyaluronic capsule and M proteins which resist phagocytosis



G.A.S .

- ◆ Cutaneous/subcutaneous skin infection
- ◆ Scarlet fever
- ◆ Rheumatic fever
- ◆ Pneumonia (Jim Henson's disease)
- ◆ Post partum fever
- ◆ Necrotizing fasciitis
- ◆ Toxic shock syndrome
- ◆ Acute glomerulonephritis
- ◆ Meningitis

GAS

- ◆ 20% asymptomatic carriers in pharynx especially in winter Also found on the skin. Worst scenario usually “strept throat” or impetigo
- ◆ Clearly a problem arises when the immune system unable to keep in check
- ◆ THE ORGANISM enters the host through mucous membranes or open wound
- ◆ Virulence dependent on M-protein, hyaluronic acid capsule and C-5 peptidase
- ◆ Common theory held that bacteria makes proteins that causes the immune system to destroy both the bacteria and body as well as destroying the tissue directly





GAS

- ◆ Health Canada works with both local/provincial public health offices.
- ◆ If requested will investigate clusters of outbreaks
- ◆ National Streptococcus Centre in Edmonton
- ◆ US...National Necrotizing Fasciitis Foundation

Necrotizing fasciitis

*Redness/skin
breakdown(painful)*

*Rapid advancement to
normal skin(+/- edema)*

*Dusky skin with
vesicles/bullae*

*Ischemic
skin/subcutaneous
tissue*

*Thrombosis of vessels ,nervesanaesthetic
necrosis*





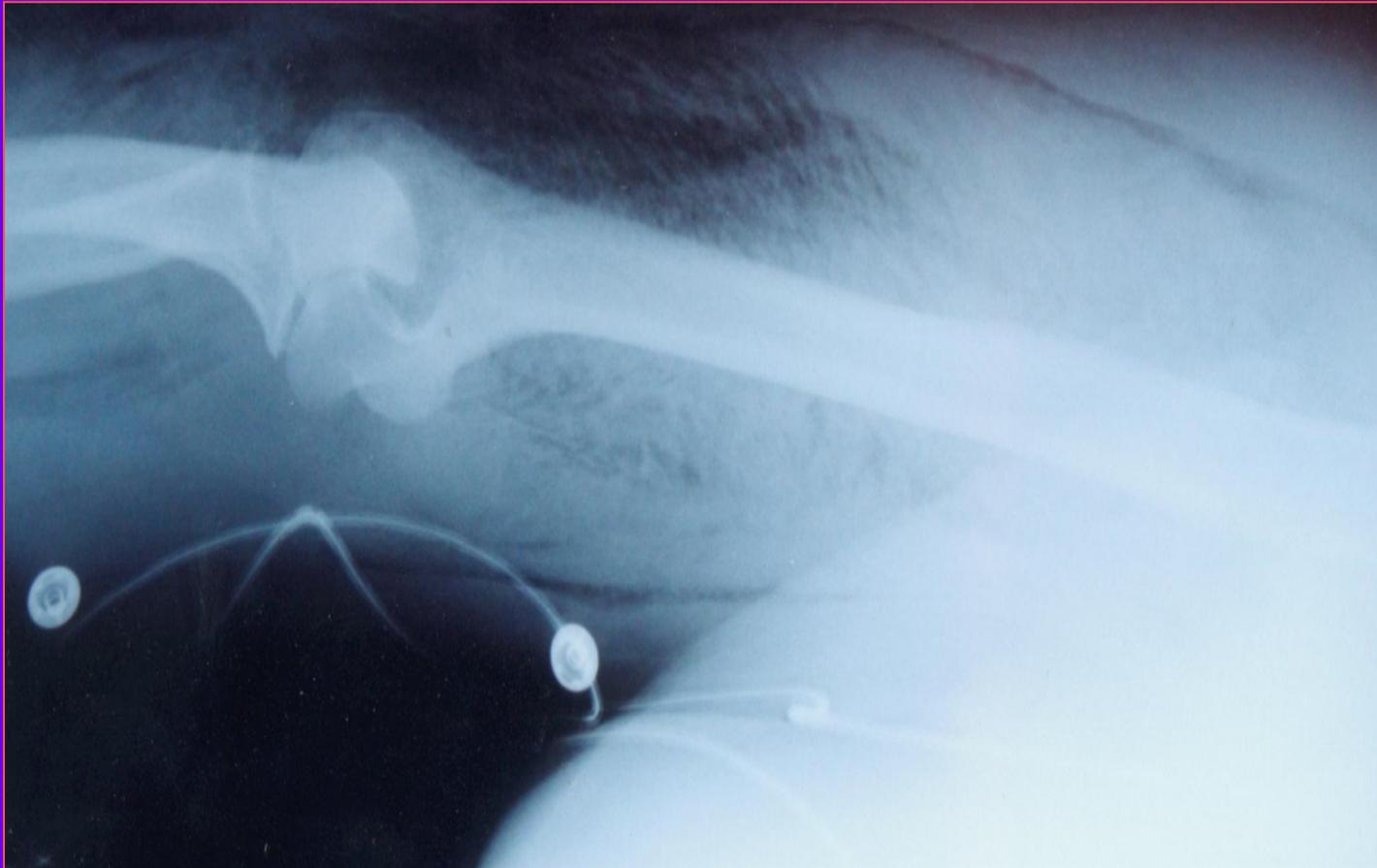
Diagnostic imaging for diagnosis

- ◆ Flat plate
- ◆ CT
- ◆ MRI

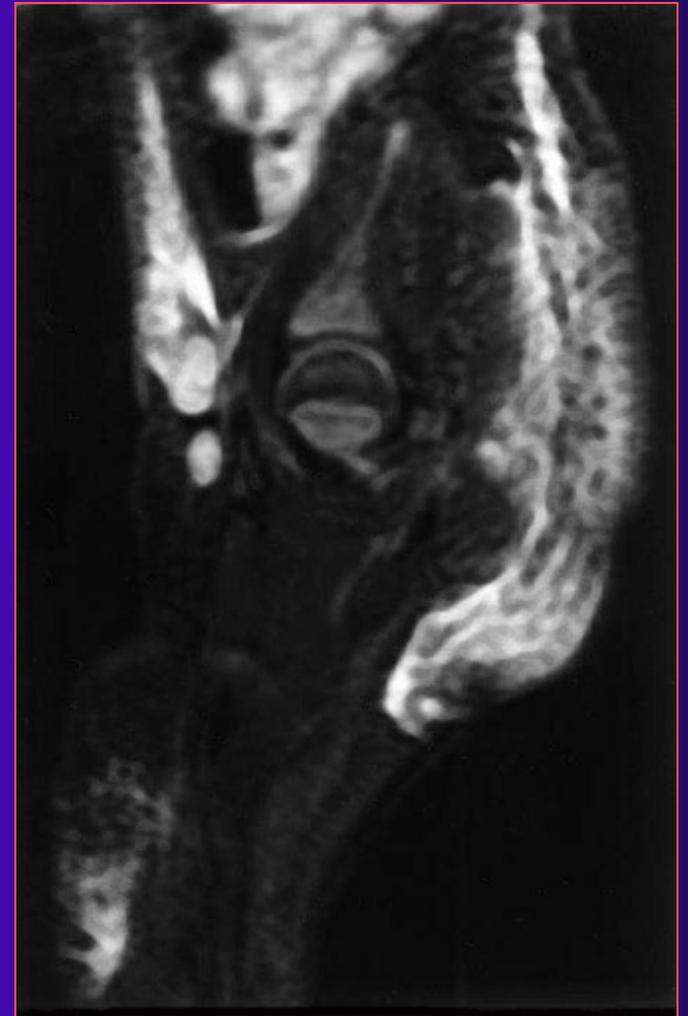
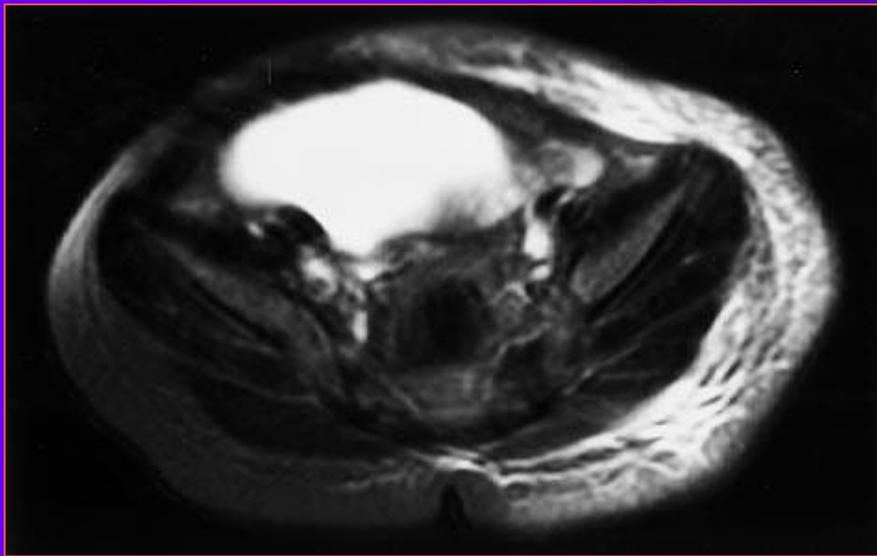
Culture/sensitivity

- Gram stain
- Tissue biopsy (peripheral or deep reached by debridement)

Crepitus/gas in tissue



MRI necrotizing faciitis





Antibiotic Regime

- ◆ Merem/Primaxin
- ◆ Pen G (Na⁺)(Eagle effect)
- ◆ Gentamycin/Clindamycin/Ampicillin

- ◆ Flagyl/Cephalosporins

- ◆ Vancomycin/Chloramphenicol



Who treats ?

- ◆ surgeons/subspecialists(co-operative effort)
- ◆ HBO specialists
- ◆ Infectious disease specialist
- ◆ Medical officer of health(reference person)



The use of human immunoglobulin in necrotizing fasciitis

Based on the theory that streptococcal exotoxin acts as a SUPERANTIGEN

Some series show a decrease in mortality by 40 %

Dosage of 2 grams/kg/day X 2 days

Product in short supply/no double blind prospective study to support



Hyperbaric Oxygen(HBO)

- Use is controversial
- Useful only after debridement
- 3 series x90 minutes of 3 atmospheres of 100% oxygen then b.i.d x 10 treatments
- Decrease in mortality by 20%
- Increases tissue oxygen by 1000%,increases wound healing,decreases edema,has a bactericidal effect,improves PMN function



The basis of treatment is **SURGICAL debridement** of all necrotic tissue including skin , subcutaneous tissue and fascia +/- muscle under a GA with a return to the operating room within 24 hours. Any limb which may have compromised viability should have a **fasciotomy**

It is **NOT** antibiotics or HBO which determines success although they are important

Cellulitis vs Necrotizing Fasciitis

- ◆ Cellulitis: dermis/subcutaneous tissue
 - : red , swollen , tender, warm
 - : borders not usually raised
 - : s.aureus, hemolytic strept
 - : blood borne, medical disorder
 - : skin disruption





Why necrotizing infection Vs simple nonoperative cellulitis

- 1) An anaerobic wound environment
- 2) The presence of toxic lytic enzymes
- 3) Bacterial synergy (you scratch my back..I'll scratch..)
- 4) Thrombosis of nutrient vessels to skin and subcutaneous tissue



Cellulitis vs Necrotizing fasciitis

- ◆ Dermis, subcutaneous tissue AND fascia
- ◆ Vasculitis, thrombosis, tissue necrosis
- ◆ Extremity, abdomen, perineal involvement
- ◆ Compromised/normal population
- ◆ If not treated early, reported 75% mortality
- ◆ Early on may mimic a cellulitis





Cellulitis vs Necrotizing Fasciitis

- ◆ Presentation may be trivial...?
musculoskeletal strain
- ◆ Initially skin may be normal with
subcutaneous tenderness and edema
- ◆ Skin then becomes tense, swollen, shiny and
indurated with maroon, violaceous blebs and
bullae
- ◆ Pain out of proportion to findings followed
by anaesthesia



GAS

- ◆ GAS is found on the skin , nose and throat of healthy people and is passed on usually with personal close contact with an infected individual(kissing ,cigarettes, sharing utensils.At risk are those living in the same household ,share the same bed or have direct contact with mouth or body secretions of the affected individual
- ◆ It is the above group that benefit from prophylactic antibiotic therapy



What are my chances of getting it??

- ◆ In Canada, 2-3/1,000,000
- ◆ You **DO NOT** catch necrotizing fasciitis from someone who has it!
- ◆ Risk increased after GAS exposure if:
 - Illicit drug use
 - Have a skin wound(burn,trauma,surgery)
 - Have immunosuppression
 - Have a chronic disease(lung/heart/liver disease, alcoholism)
 - Have chickenpox

PREVENTION



- ◆ Vaccine..working on it
- ◆ Wash hands
- ◆ Do not share personal items
- ◆ Avoid close contact with “sore throats”MD
assessment of all unexplained redness,
swelling and pain particularly if lingering
- ◆ Clean all cuts and abrasions promptly; seek
attention for a sore throat that doesn’t go
away





GAS

- ◆ GAS normal pathogen on skin,nose and throat of healthy people.Spread by close personal contact with an infected individual
- ◆ At risk persons in the same house, sharing the same bed or in direct contact with mouth or secretions
- ◆ WHILE STREPT A IS PASSED FROM PERSON TO PERSON , NECROTIZING FASCIITIS IS NOT!!!!



If left untreated a true necrotizing fasciitis can develop into a toxic shock syndrome within 24 hours with associated delirium, hypotension, hepatic dysfunction and acute renal failure .Most survivors have surgical treatment within 24 hours

BEWARE of unexplained soft tissue pain and swelling particularly in a compromised patient





Timing is everything

