INTERESTING CASE

Intern Kornkamol Leangrugsa

HISTORY TAKING

Male 50 years
U/D T2DM, HT, OldCVA

CC : ไข้ ซึมลง 1 day PTA

HISTORY TAKING

Present illness

7 days PTA ถูกตอไม้ทิ่มขาบริเวณหน้าแข้งขาซ้าย มีแผลเปิดขนาดประมาณ 1 cm ทำแผลเองโดยใช้เบตาดีน

1 day PTA แผลที่ขาซ้ายแย่ลง มีหนอง ปวดบวมแดงร้อน มีไข้ ญาติให้ประวัติว่ามีไข้สูง ซึมลง ไม่มีไอน้ำมูก ไม่มีเสมหะ ไม่หอบเหนื่อย ไม่ปวดท้องไม่อาเจียน ไม่ถ่ายเหลว ไม่มีประวัติอ่อนแรงมากขึ้น ไม่มีชักเกร็ง ปฏิเสธประวัติอุบัติเหตุบริเวณศีรษะ

HISTORY TAKING

Past history

- ไม่เคยมีอาการเช่นนี้มาก่อน
- ไม่เคยได้รับการผ่าตัด
- **U/D** T2DM , HT ,
  Old CVA (Lt hemiparesis)

Drug & allergy

- **ยาที่ใช้ประจำ**
  ASA(81) 1x1 po pc
  Clopidogrel(75) 1x1 po pc
  Losec(20) 1x1 po ac
  Atorvastatin (40) 1x1 po hs
  MFM(500) 2x2 po pc
- **ปฏิเสธประวัติแพ้ยาแพ้อาหาร**

HISTORY TAKING

At รพช.

- **V/S :** T 38 ℃ ,PR 127 /min, RR 20 /min , BP 82/48 mmHg
- **NS :** E4V5M6 , pupil 3 mm RTLBE ,look drowsiness
- **Left Leg : warm , redness , debris tissue**

Dx : Infected wound Left leg with septic shock

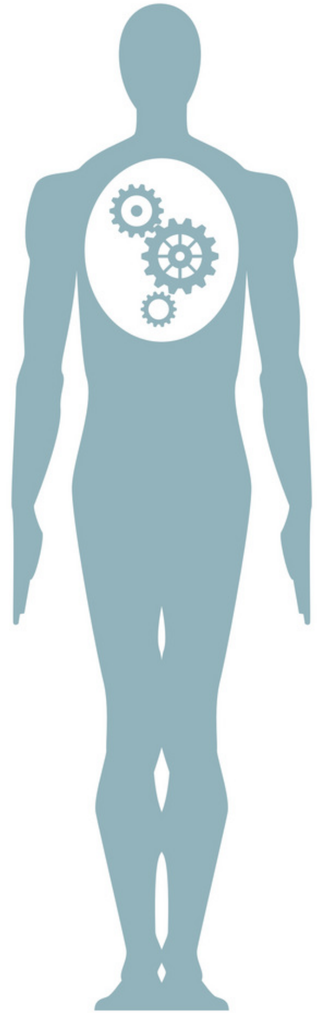
HISTORY TAKING

Management at รพช.

- NSS 1000 ml iv load
- Dopamine (2:1) iv drip 5 mcdrop/min
- Ceftriaxone 2 g iv stat
- Retained foley catheter
- DTX 133 mg%
- Lactate 6

PHYSICAL EXAMINATION

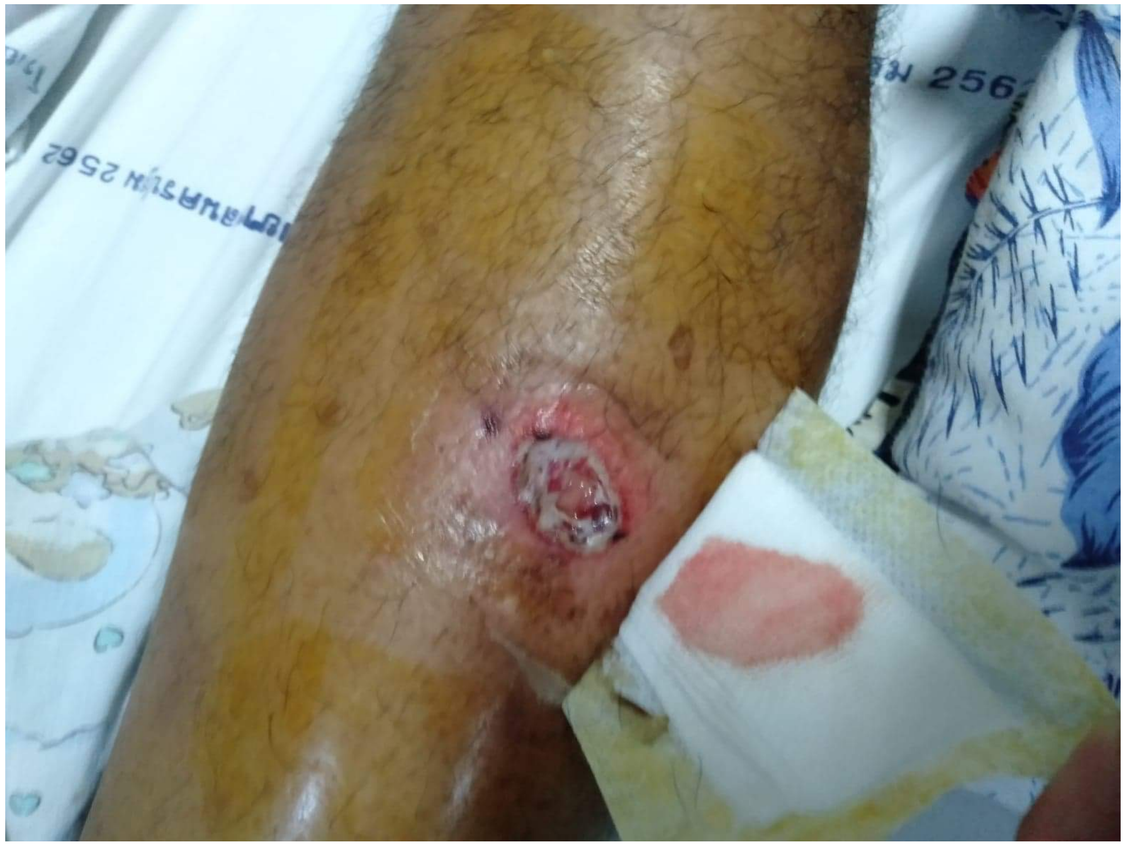
- **V/S :** T 37.1 ℃ ,PR 126 /min, RR 20 /min , BP 91/62mmHg
- **GA :** drowsiness
- **HEENT :** normocephalic head without evidence of trauma, not pale conjunctivae , anicteric sclerae
- **CVS :** normal S1S2 , no murmur , no carotid bruit
- **RS :** normal and equal breath sound , no adventitious sound
- **Abdomen :** soft , not tender , normoactive bowel sound , liver and spleen not palpable
- **GU :** no tenderness at both CVA
- **Extremities :** no edema
- **Skin :** no rash , no petechiae , no ecchymoses
- **Lymphatic system :** no lymphadenopathy
- **NS :** E4V5M6 , pupil 3 mm RTLBE ,No stiff neck

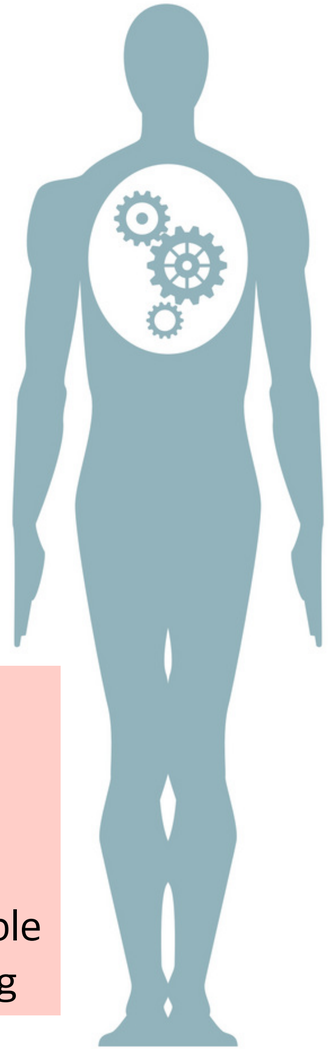


V/V	III/III
V/V	III/III

PHYSICAL EXAMINATION

- **Extremities :** Left leg >> ulcer 2*3 cm with necrotic tissue with pus , swelling , erythema , warmness ,tenderness,capillary refill < 2 sec





Pulse	Rt	Lt
FMA	2+	2+
PPA	2+	2+
PTA	2+	Can not palpable due to swelling
DPA	2+	

PROBLEM LIST

1. Left leg ulcer with swelling with tender 7 days PTA
2. Acute fever with drowsiness 1 day PTA

DEFERENTIAL DIAGNOSIS

1. Infected wound
2. Cellulitis
3. Necrotizing fasciitis

INVESTIGATIONS

CBC

CBC

WBC	**33.70**	CR	10^3 /uL	4.0-11.0	9.70
WBC corrected	**33.70**	H	10^3 /uL	4.0-11.0	9.70
RBC	**3.55**	L	10^6 /uL	4.0-5.5	4.85
HGB	**11.5**	L	g/dL	14-18	14.7
HCT	**30**	L	%	42-52	41
MCV	**83.9**		fL	83-97	83.5
MCH	**32.4**		pg	27.0-33.0	30.3
MCHC	**38.6**	H	g/dL	31.0-35.0	36.3
RDW-CV	**13.6**		%	11.5-14.5	12.5
PLT count	**227**		10^3 /uL	140-440	190
Platelet estimate	**Adequate**				Adequate
NE%	**73**		%	40-75	69
LY%	**20**		%	20-50	21
MO%	**5**		%	2-10	8
EO%	**2**		%	1-6	2
BA%	**0**		%	0-2	0
NBRC	**0**		Cells/100 W		0
RBC morpho	**Normochromic Normocytic**			Normal	Normochromic N

INVESTIGATIONS

Coagulogram

Hematology					
PT	13.6	H	Seconds	9.8-12.1	10.8
INR	1.23				0.96
PTT	33.4	H	Seconds	22.2-31.5	25.1
PTT Ratio	1.25				0.94

INVESTIGATIONS

BS , BUN,Cr ,E'lye, lactate

Item					
Chemistry					
Sugar(NaF)	244	H	mg/dL	74-106	327
BUN	42	H	mg/dL	8-20	14
Creatinine	2.33	H	mg/dL	0.72-1.18	0.92
eGFR	31	L	ml/min/1.73	> 90	97
Sodium(Na+)	130	L	mmol/L	136-146	138
Potassium(K+)	3.3	L	mmol/L	3.5-5.1	3.5
Chloride(Cl-)	96	L	mmol/L	101-109	99
CO2	19	L	mmol/L	21-31	32
AnGap	18		mmol/L	10-20	11
Calcium	7.6	L	mg/dL	8.8-10.6	
Magnesium	1.0	CR	mg/dL	1.8-2.6	
Phosphorus	1.8	L	mg/dL	2.5-4.5	
Lactate	5.0	CR	mmol/L	0.5-2.0	

INVESTIGATIONS

LFT

Chemistry				
Total Protein	5.4	L	g/dL	6.6-8.3
Albumin	2.6	L	g/dL	3.5-5.2
Globulin	2.8		g/dL	2.5-3.5
Total Bilirubin	1.16		mg/dL	0.30-1.20
Direct Bilirubin	0.52	H	mg/dL	< 0.20
SGOT/AST	21		U/L	Male < 40
SGPT/ALT	14		U/L	Male < 40
Alkaline Phosphatase	96		U/L	30-120

INVESTIGATIONS

ABG

Blood Gases

Temp	37.1		Celsius	36.5-37.7
FIO2	20.9		%	
pH	7.16	L		7.400-7.500
pCO2	18	L	mm.Hg	35.0-48.0
pO2	72	L	mm.Hg	83.0-100.0
HCO3	6.4	L	mmol/L	22.0-26.0
HCO3 std	9.2	L	mmol/L	35.0-45.0
TCO2	7.0	L	mmol/L	35.0-45.0
BE (ecf)	-22.3	L	mmol/L	35.0-48.0
BE(B)	-20.2	L	mmol/L	-2.5 - 2.5
O2 Sat	95.3		%	95.0-99.0
lactate(BG)	1.4		mmol/L	
Glucose(BG)	>685		mg/dL	
Sodium(BG)	100		mmol/L	
Potassium(BG)	1.2		mmol/L	
Chloride(BG)	40		mmol/L	
Calcium(BG)	-		mmol/L	
Hct(BG)	21		%	

INVESTIGATIONS

UA

Urine Analysis

Color	**Yellow**		Yellow
Clarity	**Slightly Turbid**		Slightly Turbid
Sp.Gr.	**1.026**	1.003-1.030	1.037
pH	**5.0**	5.0-7.0	5.5
Protein	**2+**	Negative	3+
Sugar	**2+**	Negative	2+
Blood	**Negative**	Negative	1+
Leukocyte	**Negative**	Negative	Negative
Nitrite	**Negative**	Negative	Negative
Ketone	**Trace**	Negative	Negative
Urobilinogen	**1+**	Negative	1+
Bilirubin	**1+**	Negative	Negative
RBC	**0-1**	0-1 cell/HPF	3-5
WBC	**0-1**	0-1 cell/HPF	1-2
Squa Epi	**0-1**	0-1 cell/HPF	1-2
Bacteria	**Many**		Few
Amorphous	**Trace**		...
Calcium Oxalate	**1-2**		...

INVESTIGATIONS

PUS C/S

* Aerobic Culture *

1. Numerous Streptococcus pyogenes (Strep. group A)

	1-MIC	SIR	1-MIC	SIR	1-MIC	SIR
AMPICILLIN.....		S				
CEFTRIAXONE....	<=0.12	S				
CLINDAMYCIN....	<=0.12	S				
ERYTHROMYCIN...	<=0.25	S				
LEVOFLOXACIN...	1	S				
PENICILLIN.....	<=0.03	S				
VANCOMYCIN.....	<=0.5	S				

LAB NO. : 6-0065 (ราคารวม 200 บาท)

REPORTED BY ติณณา ฐิติพันธ์รังสฤต (MT.18452) / APPROVED BY ติณณา ฐิติพันธ์รังสฤต (MT.18452)

RELEASE : 11-10-2020 11:45

S=Susceptible I=Intermediate R=Resistant N=Nonsusceptible
U=No Interpretive Categories and MIC Breakpoints

รายงานนี้ รับรองผลเฉพาะตัวอย่างที่ทดสอบนี้เท่านั้น ห้ามคัดถ่ายใบรายงานผล โดยไม่ได้รับอนุญาต

INVESTIGATIONS

H/C

* Aerobic Culture *

No Growth after 2 days

(ขวด Hemoculture จะถูกบ่มเพาะเชื้อต่ออีก 3 วัน จนครบ 5 วัน หากมีเชื้อขึ้น จะรายงานให้ทราบทันที)

LAB NO. : 1-0631 (ราคารวม 290 บาท)

REPORTED BY กาญจนา ใช้เจริญ (MT. 16336) / APPROVED BY กาญจนา ใช้เจริญ (MT. 16336)

RELEASE : 11-10-2020 07:59

รายงานนี้ รับรองผลเฉพาะตัวอย่างที่ทดสอบนี้เท่านั้น ห้ามคัดถ่ายใบรายงานผล โดยไม่ได้รับอนุญาต

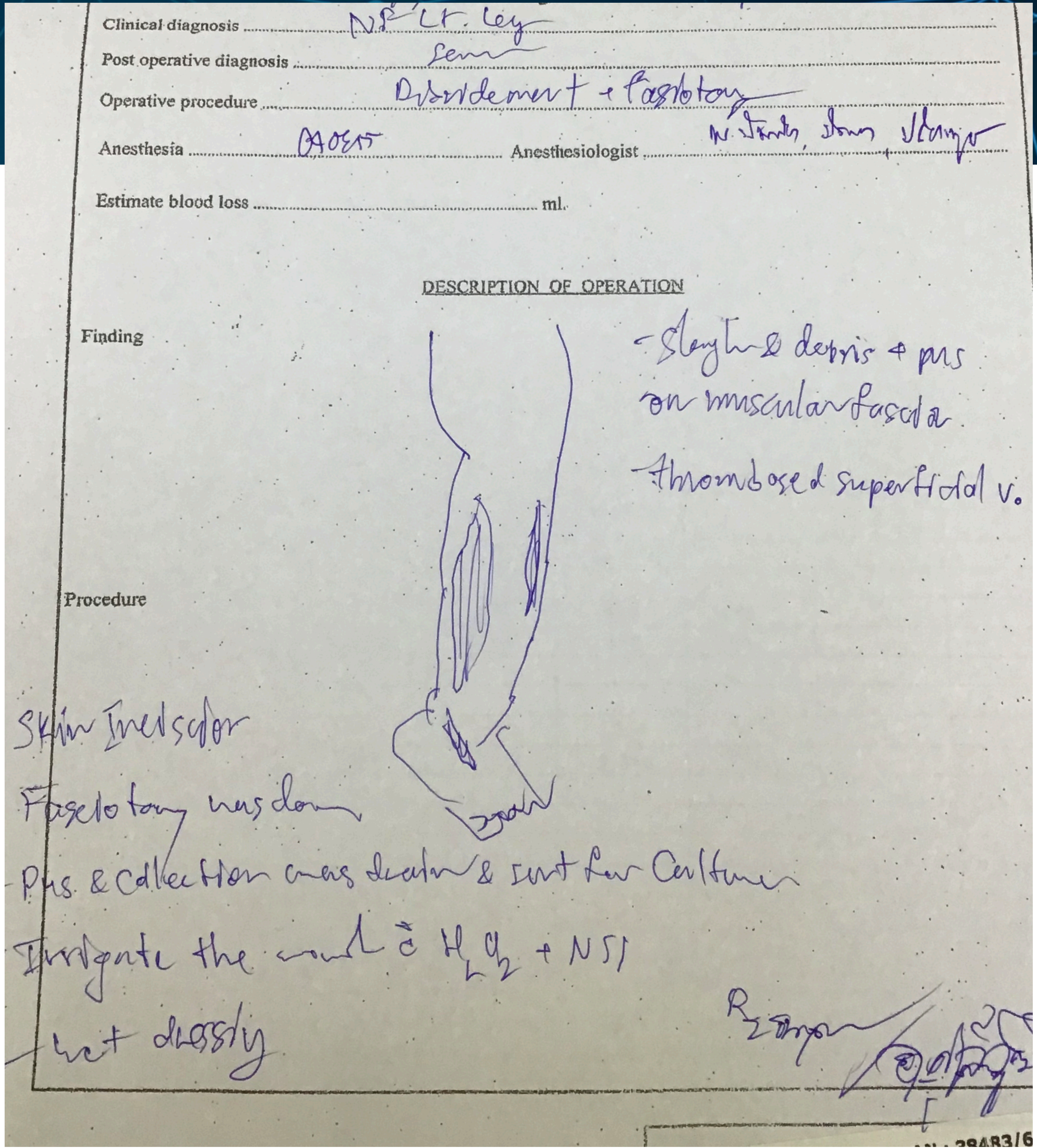
MANAGEMENT

- Resuscitation + Sepsis protocol

- IV fluid resuscitation
- Inotropic drug >> NE
- Hydrocortisone
- IV ATB > ceftriaxone 2 g v OD / clindamycin 900 mg v q 8 hr
- **Set OR for debridement**

MANAGEMENT

- Set OR for debridement



Clinical diagnosis NF Lt. leg

Post operative diagnosis Same

Operative procedure Debridement + fasciotomy

Anesthesia [illegible] Anesthesiologist [illegible]

Estimate blood loss ml.

DESCRIPTION OF OPERATION

Finding

- Slough & debris + pus on muscular fascia
- thrombosed superficial v.

Procedure

Skin incision

Fasciotomy was done

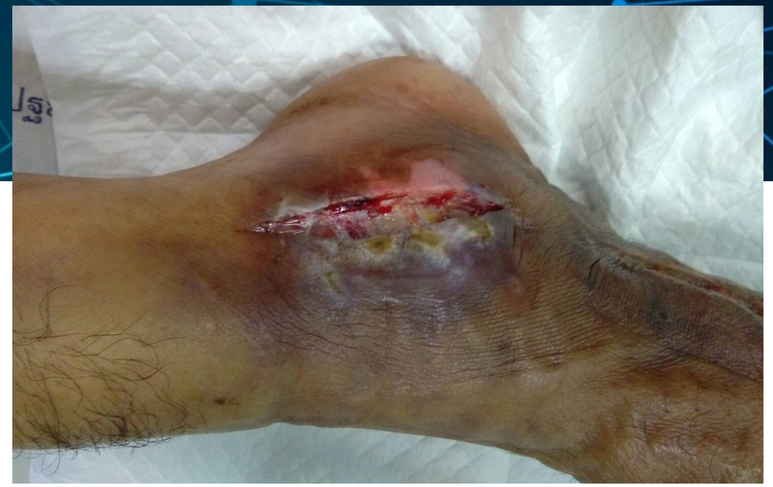
Pus & collection was drain & sent for culture

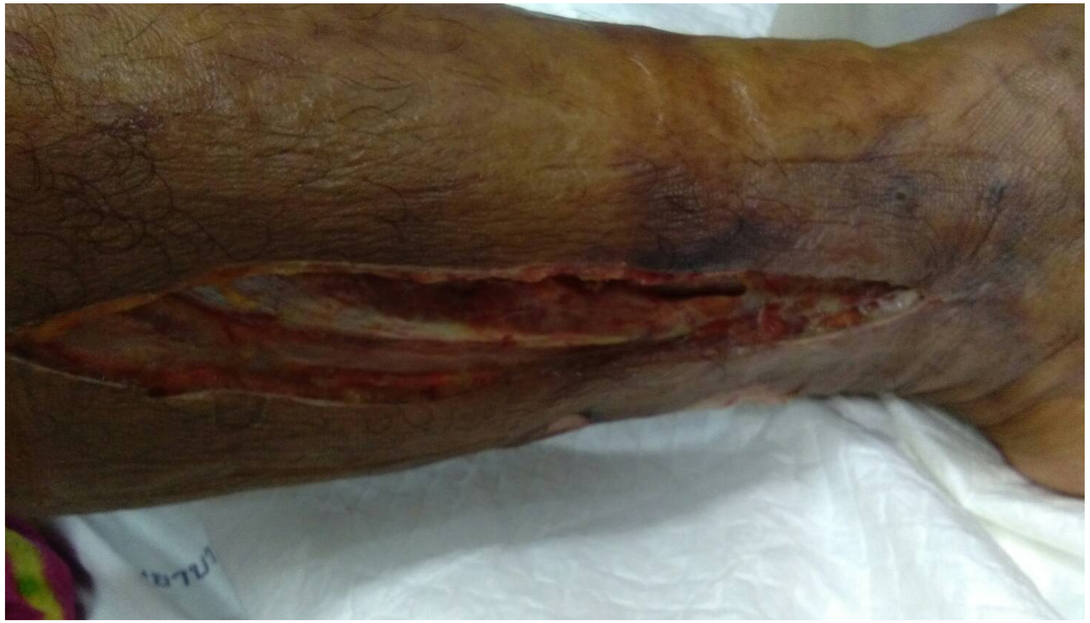
Irrigate the wound c̄ H_2O_2 + NSS

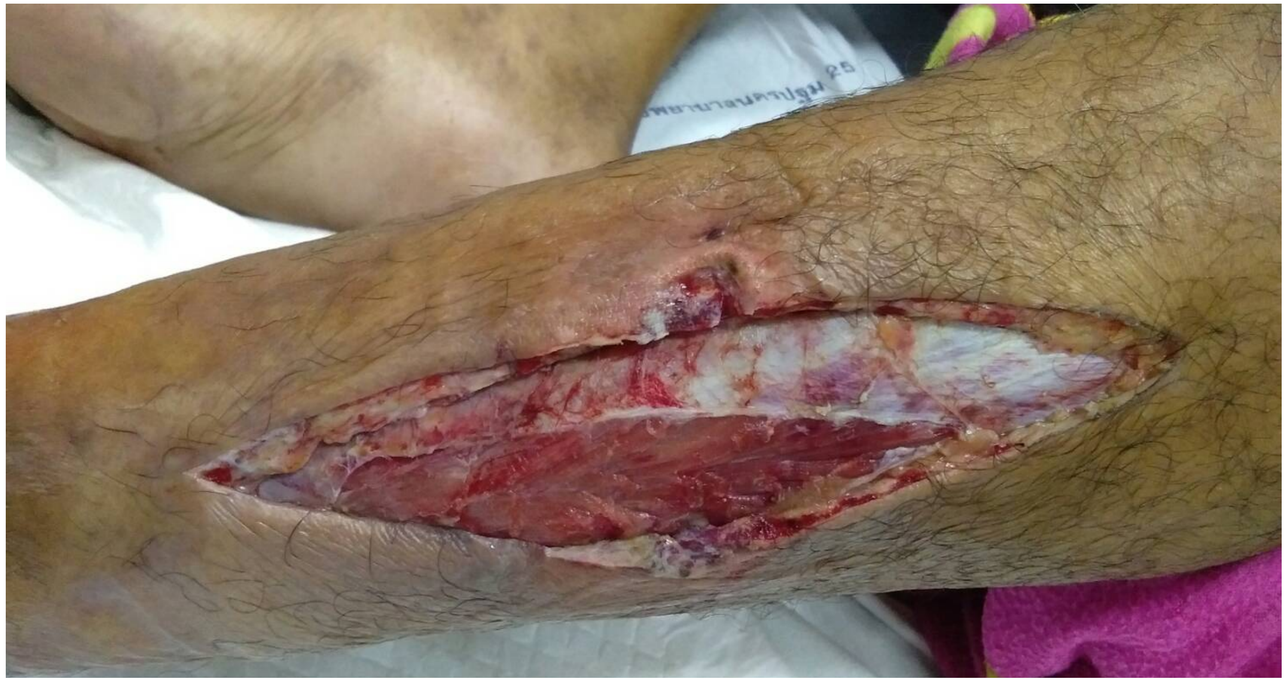
wet dressing

MANAGEMENT

- POD 1

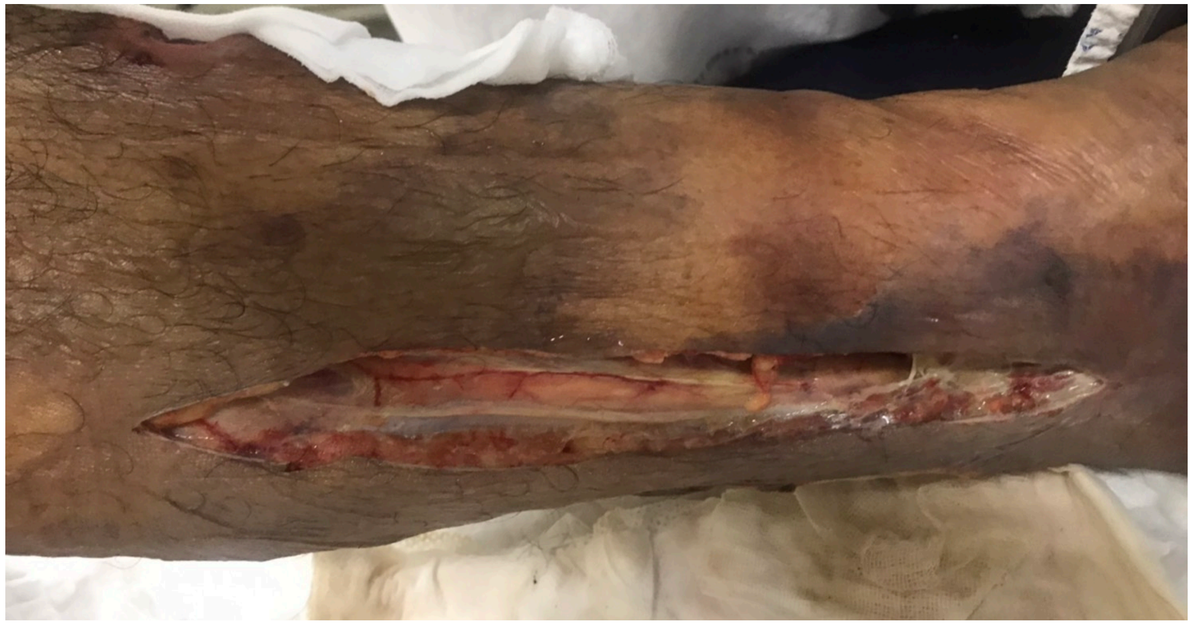


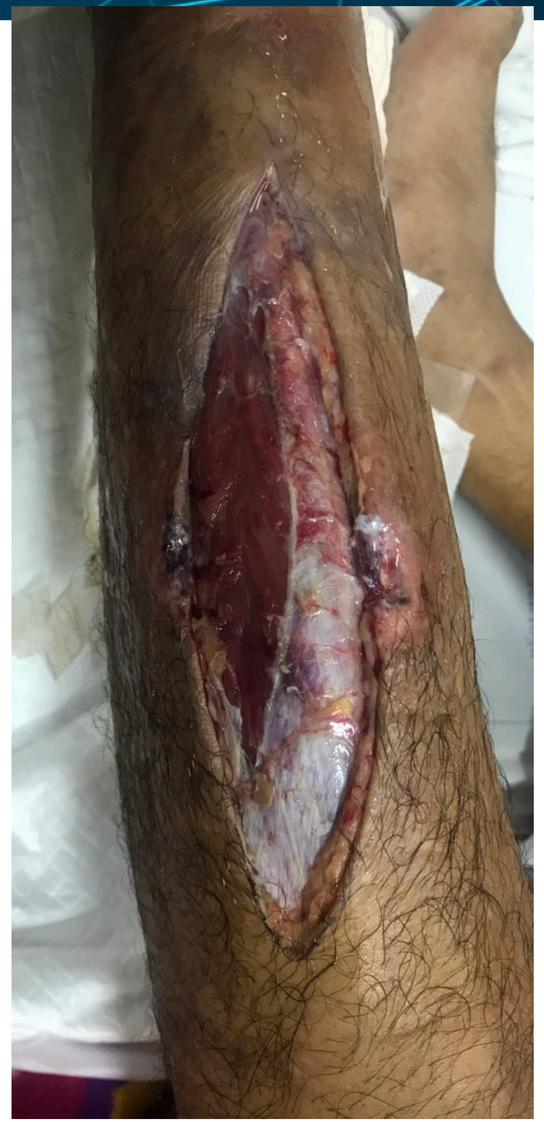


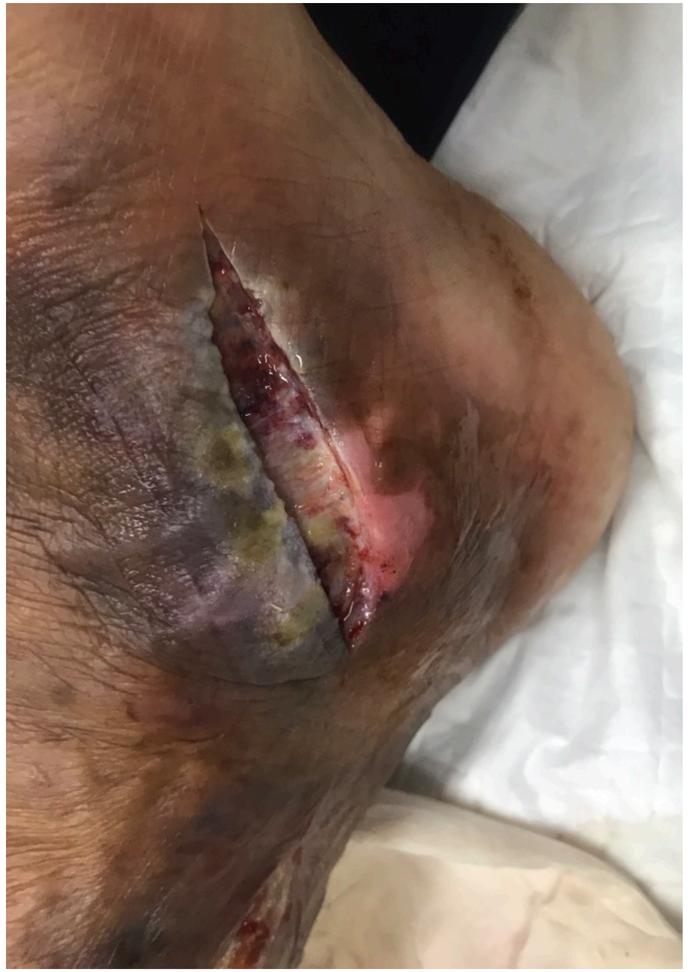


MANAGEMENT

- POD 2

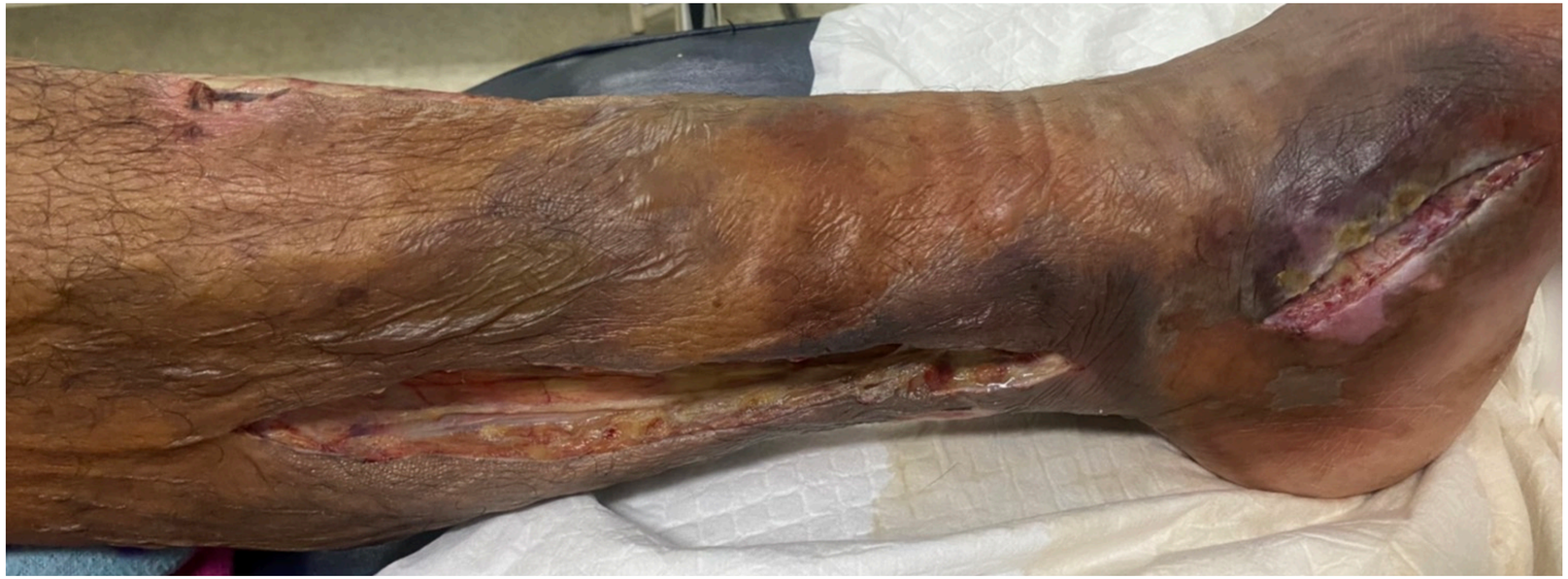


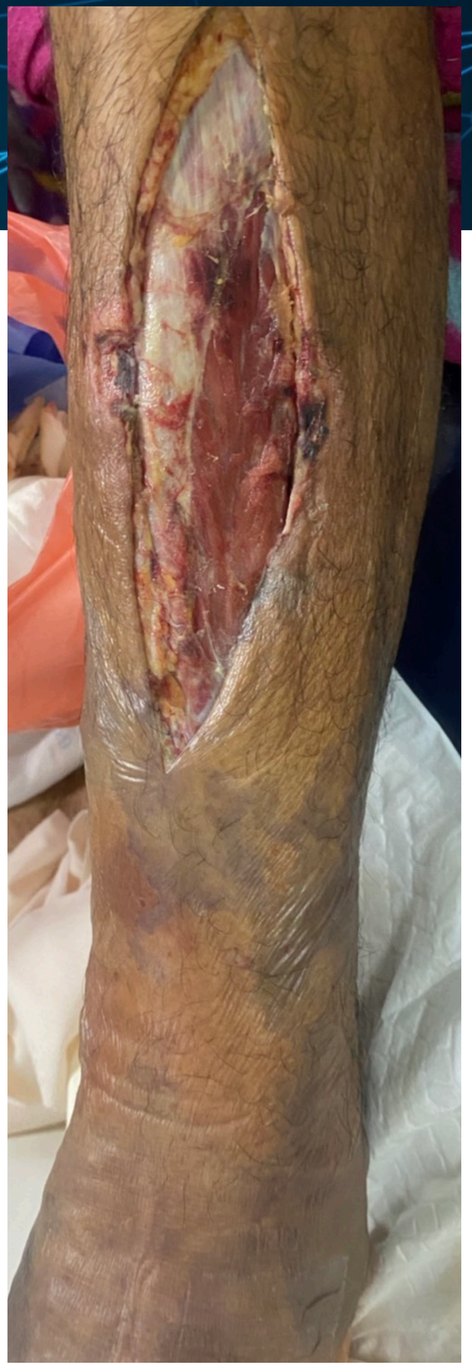




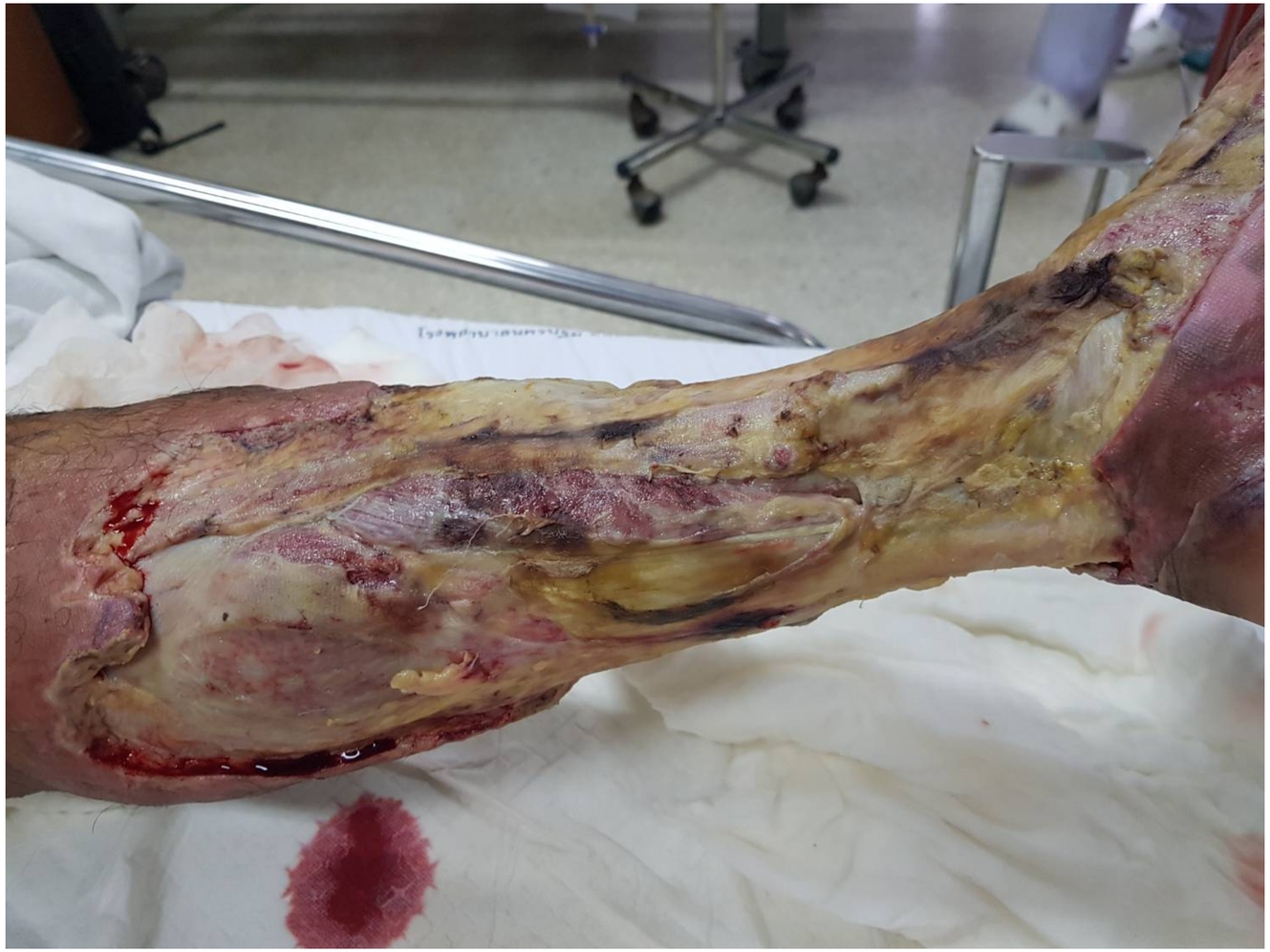
MANAGEMENT

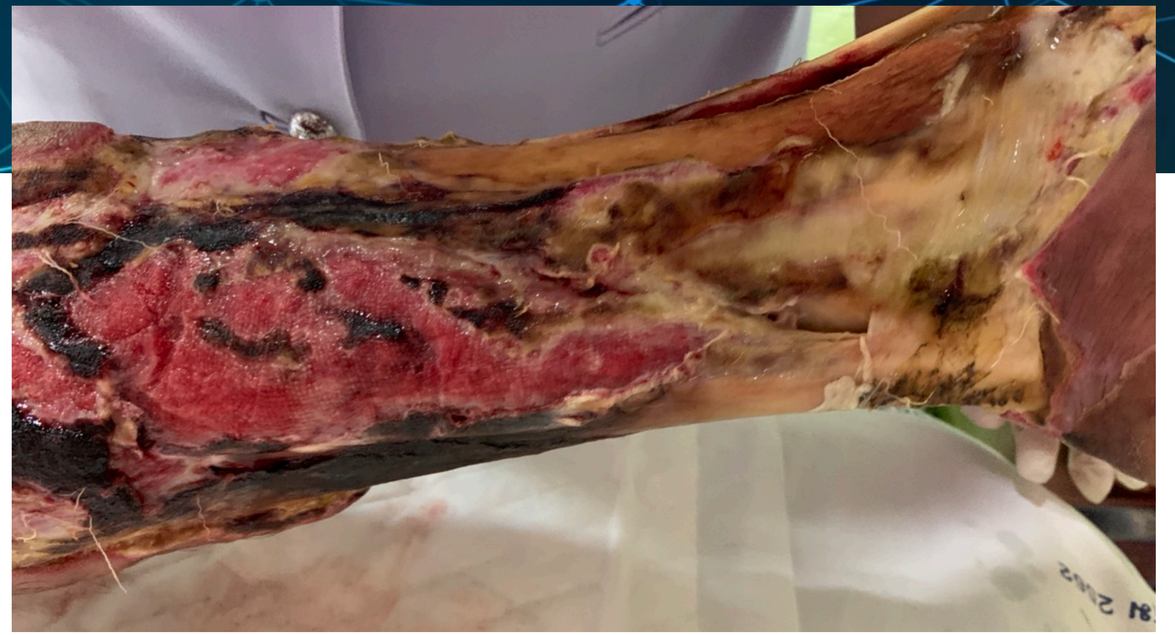
- POD 3

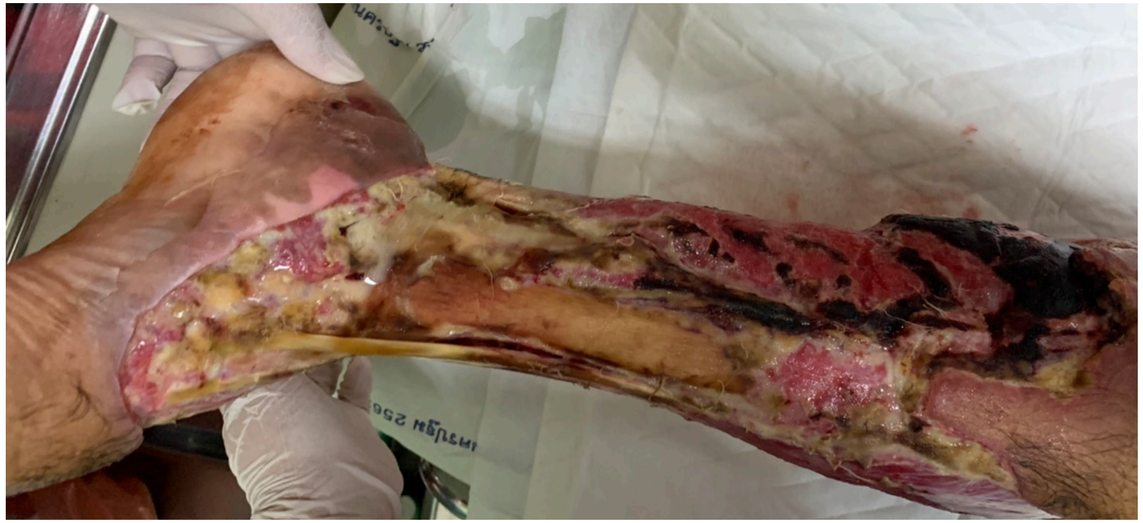




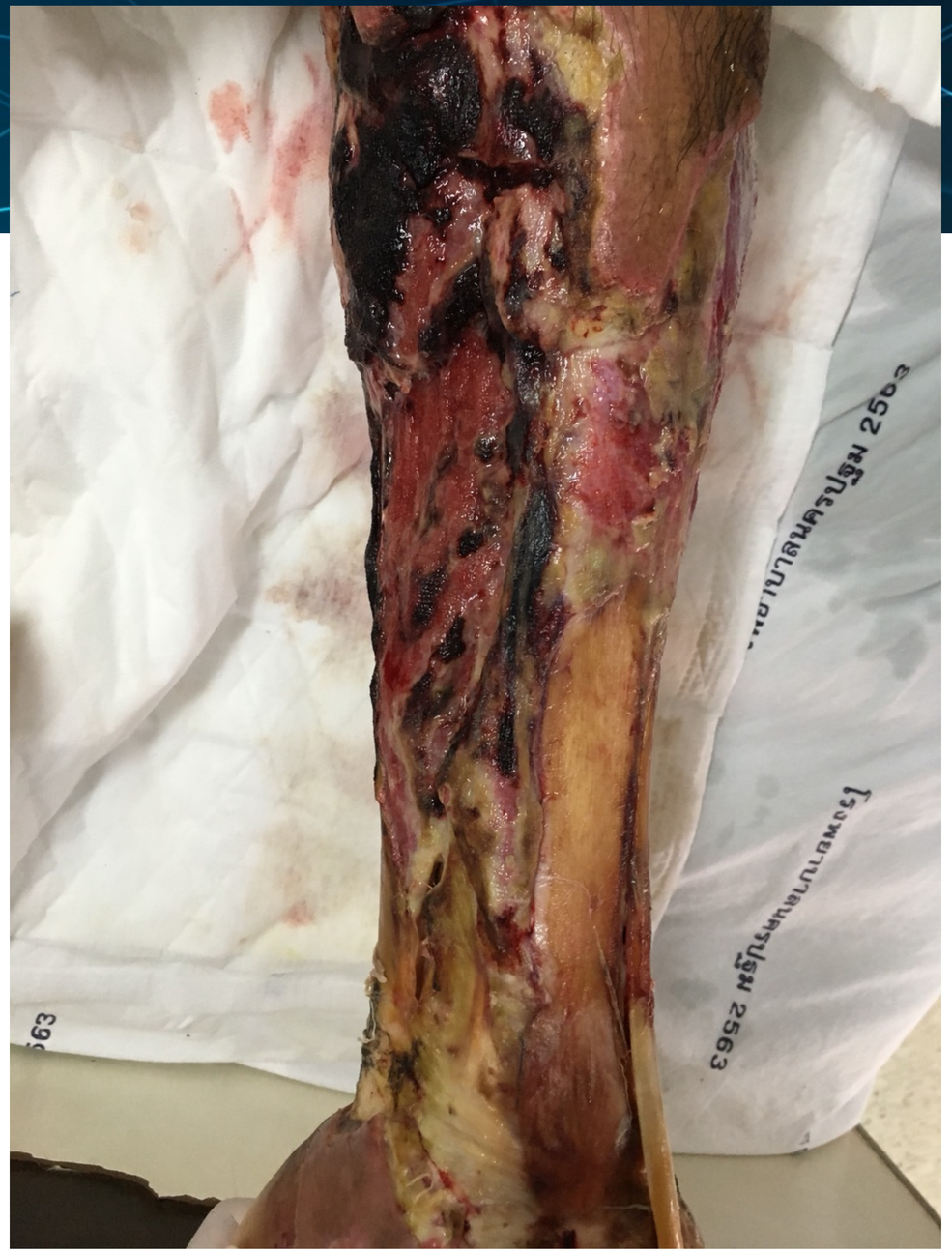
MANAGEMENT



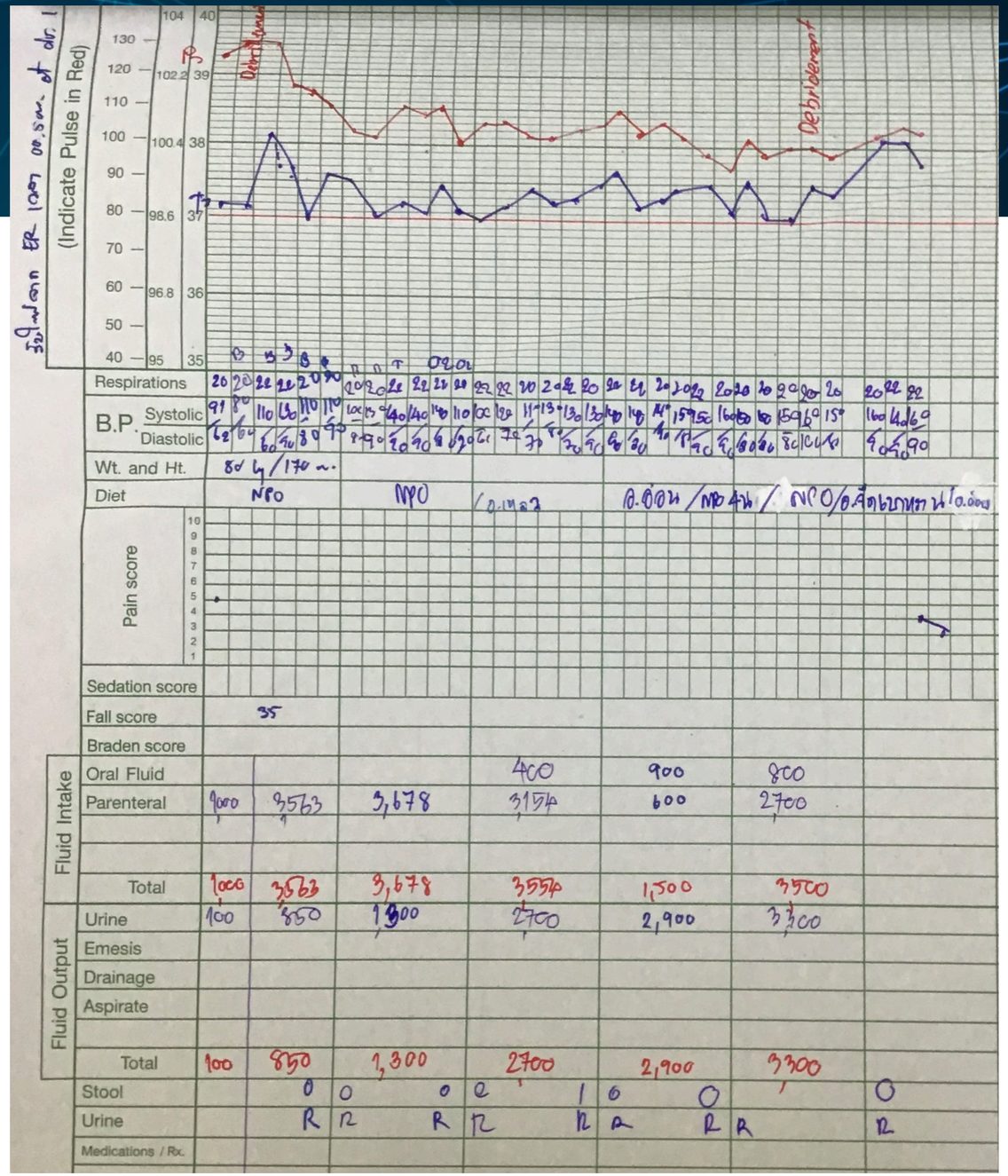




MANAGEMENT



MANAGEMENT


(Indicate Pulse in Red)
Respirations
B.P. Systolic / Diastolic
Wt. and Ht.
Diet
Pain score
Sedation score
Fall score
Braden score
Fluid Intake
Oral Fluid
Parenteral
Total
Fluid Output
Urine
Emesis
Drainage
Aspirate
Total
Stool
Urine
Medications / Rx.
Debridement
Debridement

NECROTIZING FASCIITIS

- Necrotizing fasciitis is an aggressive subcutaneous infection that tracks along the superficial fascia, which comprises all the tissue between the skin and underlying muscles .

- The term “fasciitis” sometimes leads to the mistaken impression that the muscular fascia or aponeurosis is involved, but in fact it is the superficial fascia that is most commonly involved.

- Necrotizing fasciitis is a surgical diagnosis characterized by
    - ➡️ friability of the superficial fascia
    - ➡️ dishwater-gray exudate
    - ➡️ absence of pus

- Multiple causes, risk factors, anatomical locations, and pathogenic mechanisms, but all such infections result in widespread tissue destruction, which may extend from the epidermis to the deep musculature

Table 1. Factors Conferring a Predisposition to Specific Necrotizing Soft-Tissue Infections.*

Predisposing Factor	Clinical Syndrome	Etiologic Agent
Major penetrating trauma: crush or deeply penetrating wound	Gas gangrene	*Clostridium perfringens*, *C. histolyticum*, or *C. novyi*
Minor penetrating trauma	NF type II	
Freshwater laceration		*Aeromonas hydrophila*
Saltwater laceration		*Vibrio vulnificus*
Minor nonpenetrating trauma: muscle strain, sprain, or contusion	NF type II or streptococcal myonecrosis	*Streptococcus pyogenes*
Mucosal breach: mucosal tear (rectal, vaginal, urethral); gastrointestinal, genitourinary or gynecologic surgery	NF type I	Mixed aerobic and anaerobic organisms
Skin breach		
Varicella lesions	NF type II or streptococcal myonecrosis	*S. pyogenes*
Insect bites	NF type II or streptococcal myonecrosis	*S. pyogenes*
Injection drugs	Gas gangrene	*C. perfringens*, *C. histolyticum*, *C. novyi*, or *C. sordellii*
Immunocompromised state		
Diabetes with peripheral vascular disease	NF type I	Mixed aerobic and anaerobic organisms
Cirrhosis and ingestion of raw oysters	NF type II	*V. vulnificus*
Neutropenia	Gas gangrene	*C. septicum*
In women: pregnancy, childbirth, abortion (spontaneous or medically induced), gynecologic procedures or surgery	NF type II, streptococcal myonecrosis, or clostridial myonecrosis	*S. pyogenes*, *C. perfringens*, or *C. sordellii*
Occult factors: colonic lesions, including carcinoma	Spontaneous gas gangrene	*C. septicum*

* Gas gangrene is also known as clostridial myonecrosis.

Epidemiologic Features and Causes

Epidemiologic Features and Causes

- The annual incidence of necrotizing fasciitis ranges from 15.5 cases per 100,000 population in Thailand to 0.3 to 5 cases per 100,000 in other regions.

Table 1 | Classification of responsible pathogens according to type of infection.

Microbiological type	Pathogens	Site of infection	Co-morbidities
Type I (polymicrobial)	Obligate and facultative anaerobes	Trunk and perineum	Diabetes mellitus
Type II (monomicrobial)	Beta-hemolytic streptococcus A	Limbs	
Type III	*Clostridium* species	Limbs, trunk, and perineum	Trauma
	Gram-negative bacteria		Seafood consumption (for *Aeromonas*)
	Vibrios spp.		
	Aeromonas hydrophila		
Type IV	*Candida* spp.	Limbs, trunk, perineum	Immunosuppression
	Zygomycetes		

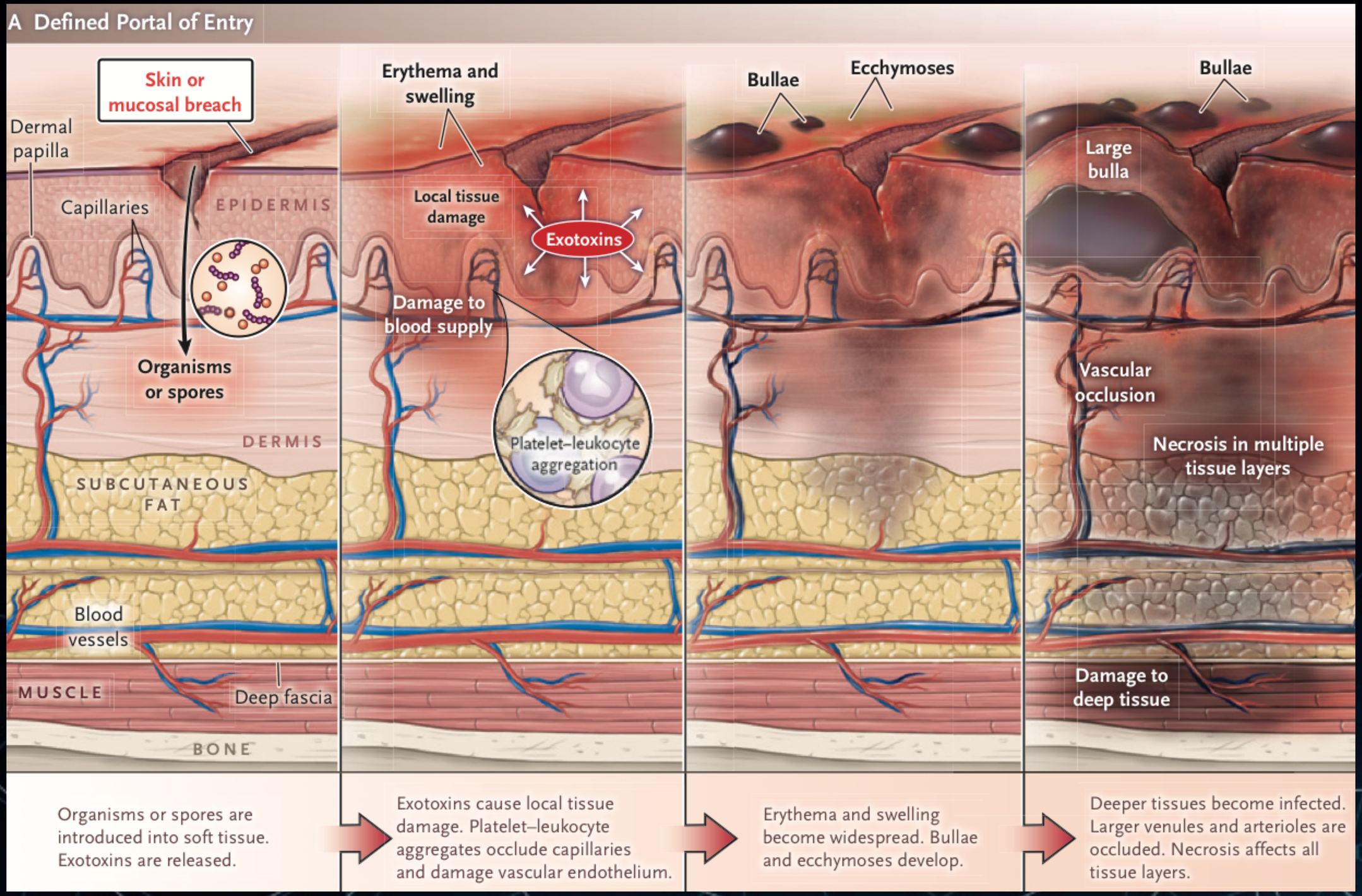
Necrotizing fasciitis type I

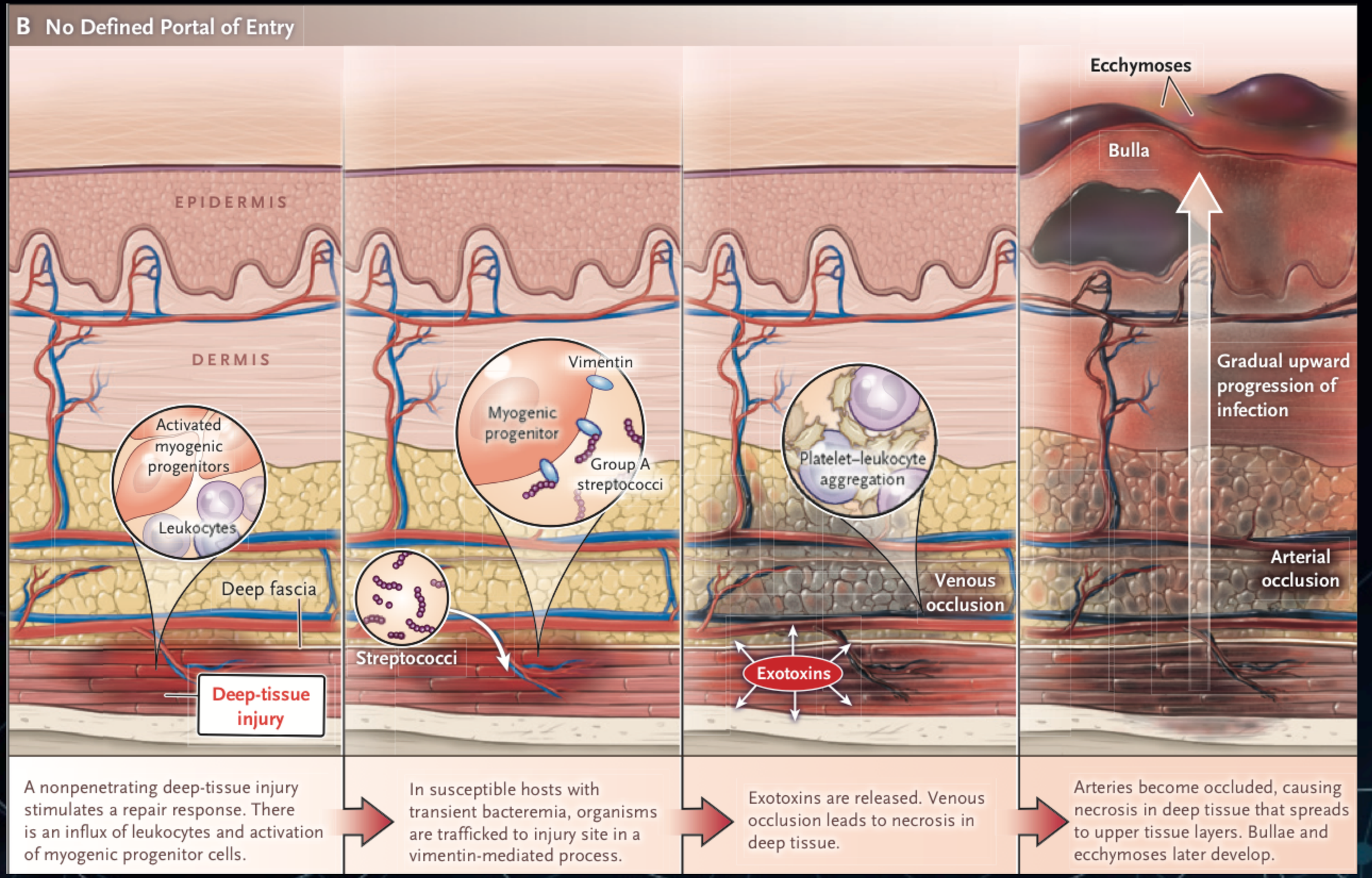
- ➡️ polymicrobial infection involving aerobic and anaerobic organisms.
- ➡️ elderly or in those with underlying illnesses.
- ➡️ most commonly associated with 4 clinical settings:

(1) perianal abscesses, penetrating abdominal trauma, or surgical procedures involving the bowel
(2) decubitus ulcers
(3) injection sites in illicit drug users
(4) spread from a genital site such as Bartholin abscess, episiotomy wound, or a minor vulvovaginal infection.

Necrotizing fasciitis type II

- Among gram-positive organisms
- group A streptococcus remains the most common pathogen, followed by Staphylococcus aureus (MRSA)
- may occur in any age group and in persons without any underlying ill


A Defined Portal of Entry
Skin or mucosal breach
Dermal papilla
Capillaries
EPIDERMIS
Organisms or spores
DERMIS
SUBCUTANEOUS FAT
Blood vessels
MUSCLE
Deep fascia
BONE
Erythema and swelling
Local tissue damage
Exotoxins
Damage to blood supply
Platelet–leukocyte aggregation
Bullae
Ecchymoses
Bullae
Large bulla
Vascular occlusion
Necrosis in multiple tissue layers
Damage to deep tissue
Organisms or spores are introduced into soft tissue. Exotoxins are released.
Exotoxins cause local tissue damage. Platelet–leukocyte aggregates occlude capillaries and damage vascular endothelium.
Erythema and swelling become widespread. Bullae and ecchymoses develop.
Deeper tissues become infected. Larger venules and arterioles are occluded. Necrosis affects all tissue layers.


B No Defined Portal of Entry
EPIDERMIS
DERMIS
Activated myogenic progenitors
Leukocytes
Deep fascia
Deep-tissue injury
Vimentin
Myogenic progenitor
Group A streptococci
Streptococci
Platelet–leukocyte aggregation
Venous occlusion
Exotoxins
Ecchymoses
Bulla
Gradual upward progression of infection
Arterial occlusion
A nonpenetrating deep-tissue injury stimulates a repair response. There is an influx of leukocytes and activation of myogenic progenitor cells.
In susceptible hosts with transient bacteremia, organisms are trafficked to injury site in a vimentin-mediated process.
Exotoxins are released. Venous occlusion leads to necrosis in deep tissue.
Arteries become occluded, causing necrosis in deep tissue that spreads to upper tissue layers. Bullae and ecchymoses later develop.


Diagnosis

Pitfalls Diagnostic

Table 2. Pitfalls in the Diagnosis of Necrotizing Soft-Tissue Infection.*

Pitfall	Explanation
Absence of fever	Fever is often absent in patients with necrotizing soft-tissue infections because of NSAIDs that are self-administered or prescribed in the emergency department or in postsurgical settings. Fever is also absent in patients with necrotizing infection due to *C. sordellii*.
Absence of cutaneous manifestations	Patients with spontaneous or cryptogenic necrotizing infections (i.e., infections without an obvious bacterial portal of entry) that begin in the deep soft tissues often do not have cutaneous signs of infection until late in the course of the disease.
Attributing severe pain to injury or procedure	Severe pain is a key finding in patients with necrotizing infections. However, when such infections develop after surgery or parturition, pain may be erroneously attributed to the procedure itself. Similarly, perineal pain may be attributed to hemorrhoids, epididymitis, or vaginal or rectal trauma. Severe pain associated with spontaneous or cryptogenic infections is often wrongly attributed to muscle strain or venous thrombosis. If pain is out of proportion to the suspected cause or requires opioids or ketorolac for management, a developing necrotizing infection should be considered. Pain may be absent because of the use of narcotics or NSAIDs or because of neuropathy in patients with diabetes.
Nonspecific imaging tests	In patients with necrotizing infections, radiographs may show only edema, with no evidence of gas in the deep tissue. Since this finding is consistent with noninfectious causes (e.g., soft-tissue injury and postsurgical and postpartum conditions), it may confound the diagnosis.
Attributing systemic manifestations to other causes	Nausea, vomiting, and diarrhea may be early manifestations of toxemia from group A streptococcal infection, though they are often wrongly attributed to food poisoning or viral illness.

* NSAIDs denotes nonsteroidal antiinflammatory drugs.

Clinical Findings

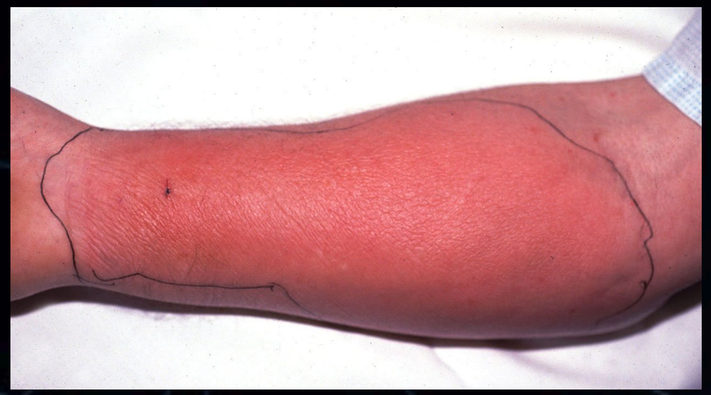
- features that suggest involvement of deeper tissues include

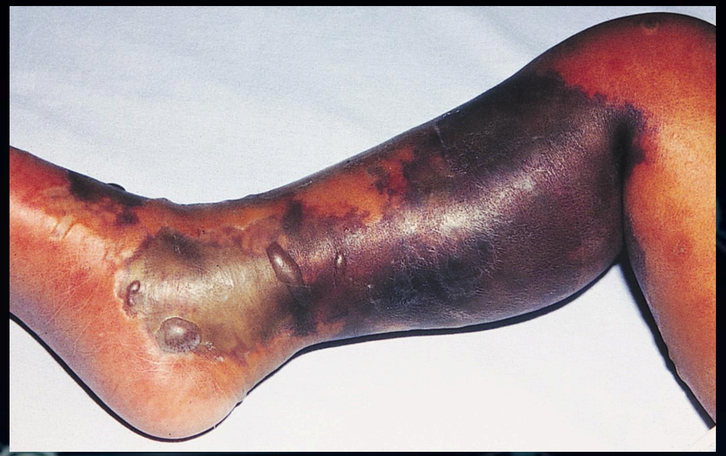
(1) severe pain that seems disproportional to the clinical findings
(2) failure to respond to initial antibiotic therapy
(3) the hard, wooden feel of the subcutaneous tissue, extending beyond the area of apparent skin involvement
(4) systemic toxicity, often with altered mental status
(5) edema or tenderness extending beyond the cutaneous erythema
(6) crepitus, indicating gas in the tissues
(7) bullous lesions
(8) skin necrosis or ecchymoses.

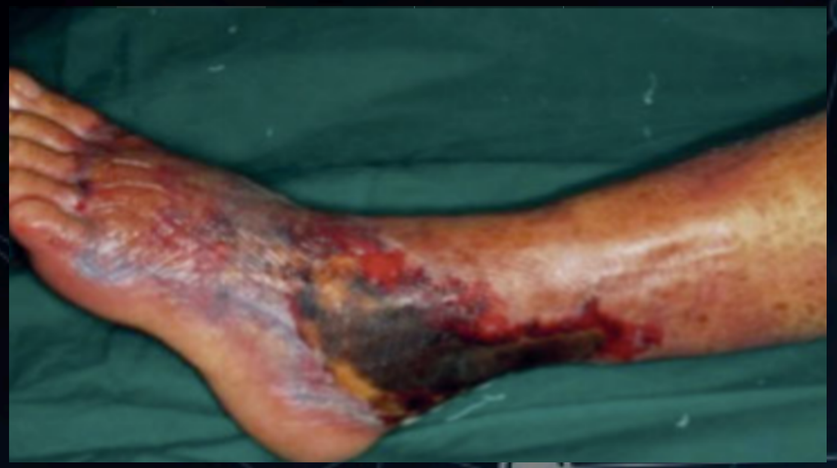
factors that differentiated necrotizing fasciitis from cellulitis

- recent surgery
- pain out of proportion
- clinical signs
- hypotension
- skin necrosis
- hemorrhagic bullae

Clinical stages of necrotizing fasciitis	Stage 1 (early)	Stage 2 (intermediate)	Stage 3 (late)
Clinical features	Tenderness to palpation (extending beyond the apparent area of skin involvement) Erythema Swelling Warm skin	Blister or bullae formation (serous fluid)	Crepitus Skin anesthesia Erythema Skin necrosis with dusky discoloration







Disorder	Characteristic
Cellulitis/adiposities (nonnecrotizing)	Erythematous, edematous, indurated tissue with normal appearing subcutaneous fat and fascia
Myonecrosis	Noninfectious inflammation/necrosis of muscle only
Lymphedema	Indurated, edematous extremity without systemic signs of infection
Noninfectious fasciitis (eosinophilic fasciitis)	Chronic disorder, diagnosed by biopsy, treated with steroids
Phlegmasia cerulea dolens	Edema of the entire affected extremity
Myxedema	Systemic manifestations of severe hypothyroidism

Imaging Tests

Plain film - subcutaneous gas

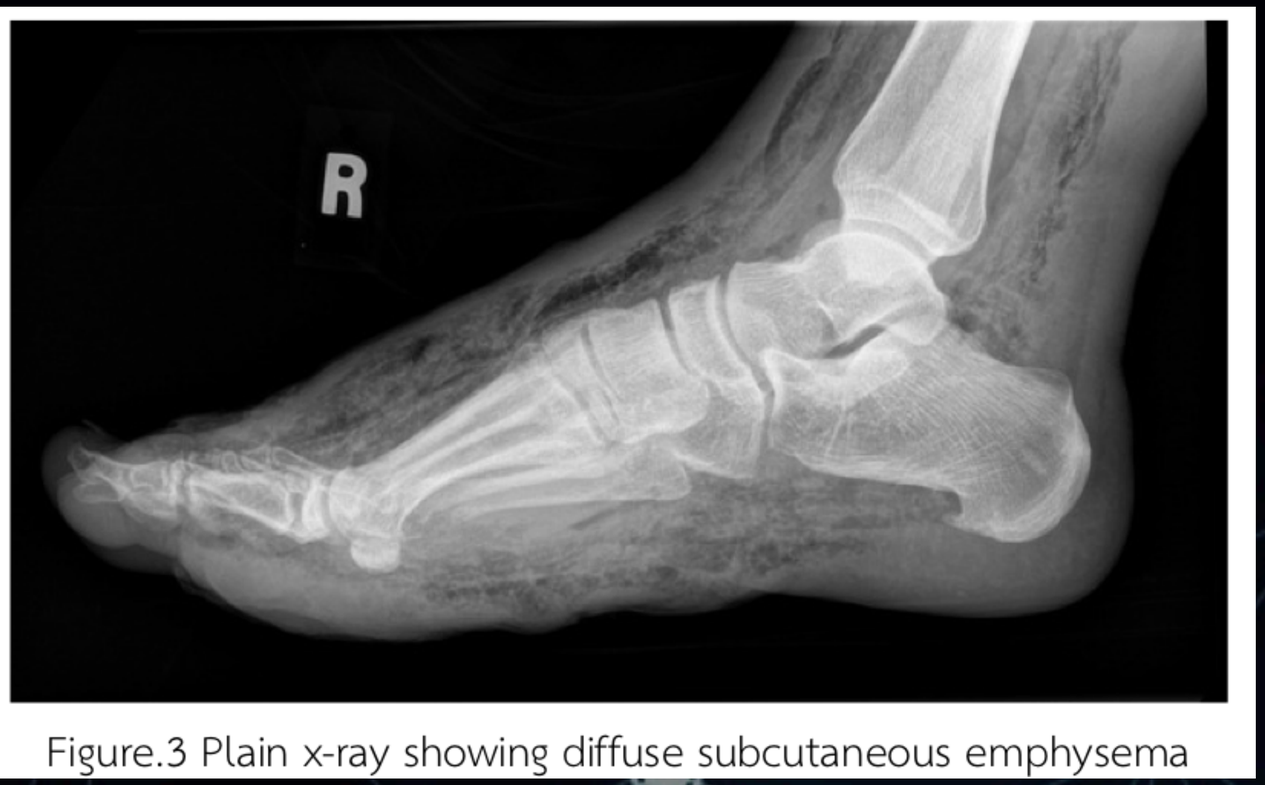
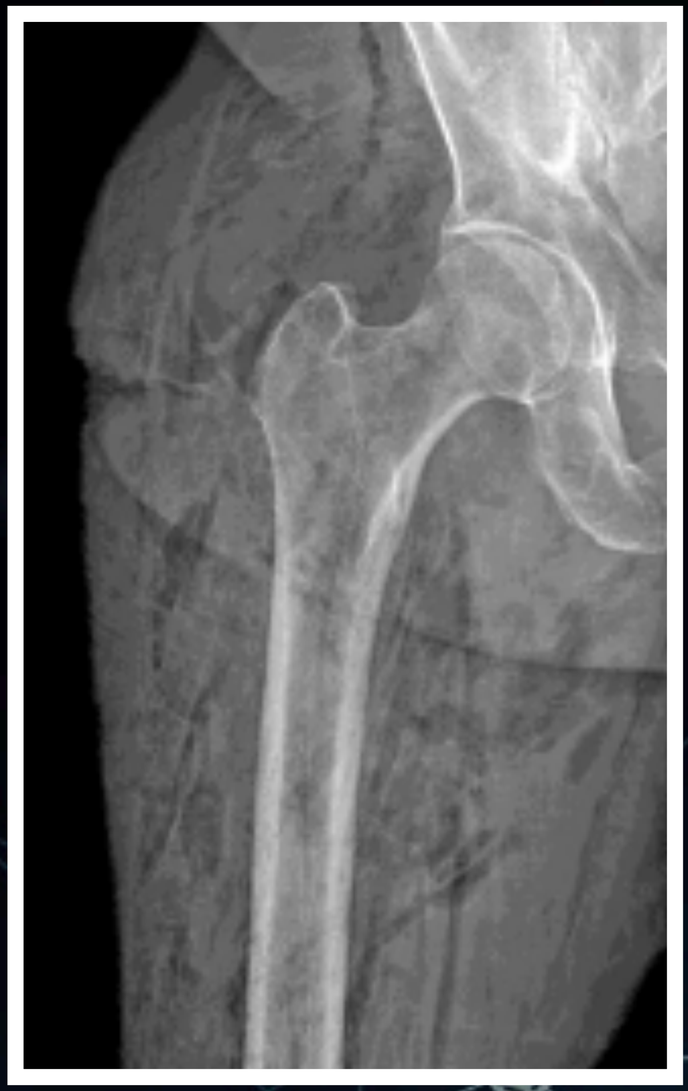


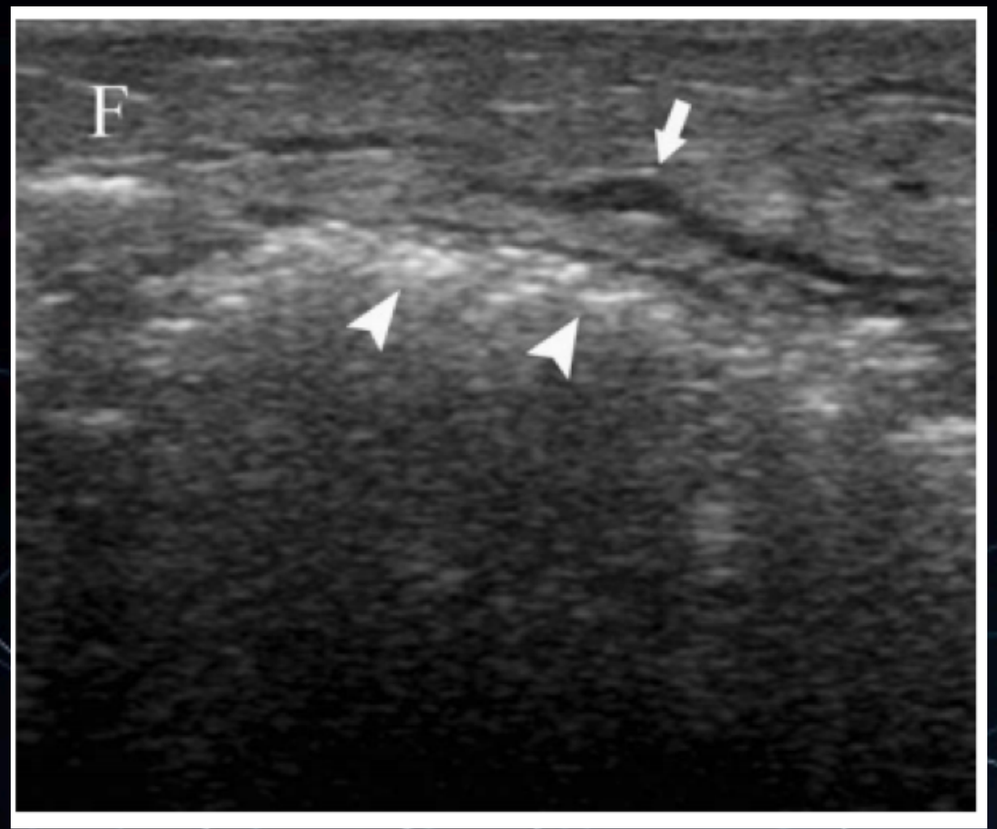
Figure.3 Plain x-ray showing diffuse subcutaneous emphysema

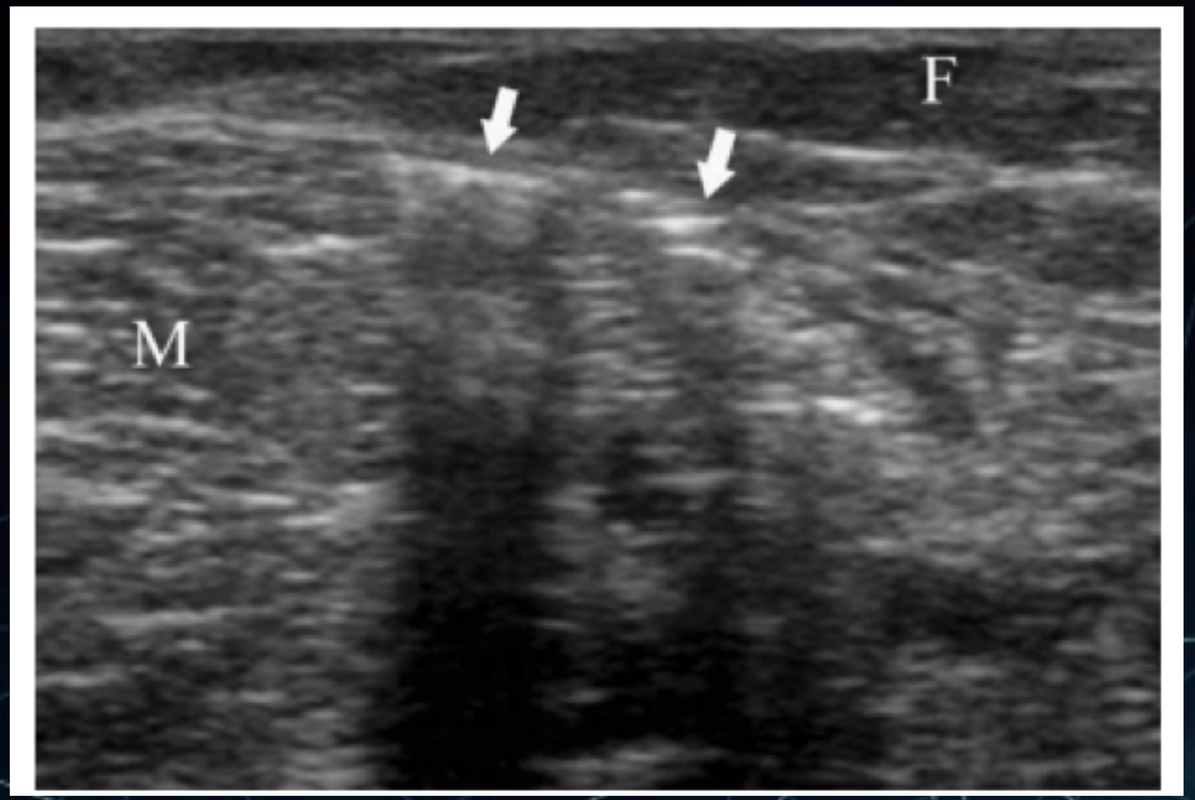


Imaging Tests

Ultrasound - hyperechoic soft-tissue emphysema with acoustic shadow

- The overlying subcutaneous fat shows increased echogenicity with interlacing anechoic spaces representing perifascial fluid spreading along the fascial planes (cobblestone appearance)



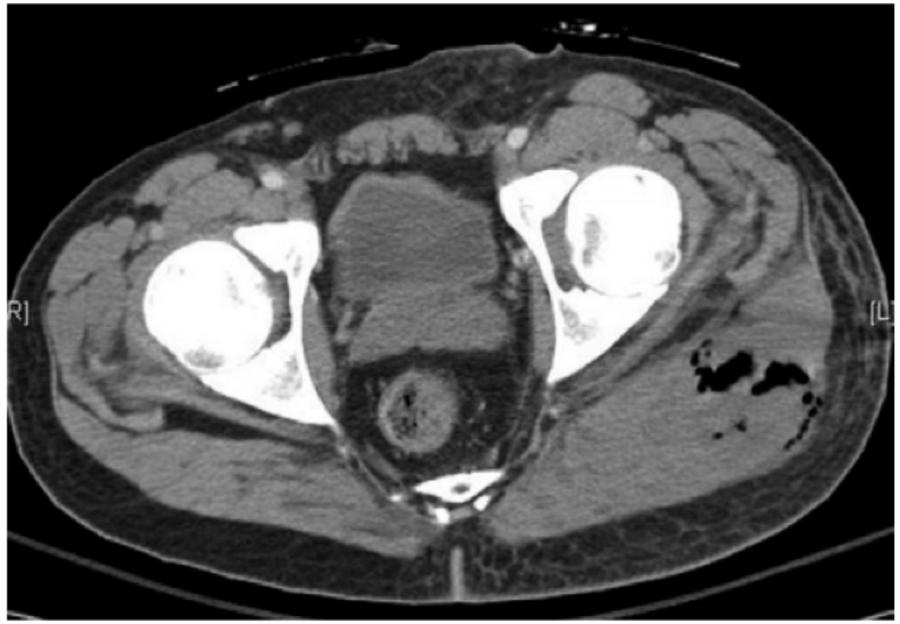


Imaging Tests

CT , MRI

- soft-tissue swelling in patients with group A streptococcal infection
- gas in the tissues of patients with gas gangrene or necrotizing fasciitis type I

Soft-tissue gas associated with fluid collections within the deep fascia
Fascial thickening
Fascial edema
Absent of fascial enhancement ซึ่งเป็นลักษณะที่มีความจำเพาะสูงมากแสดงถึงการมี fascial necrosis



Imaging Tests

CT , MRI

- soft-tissue swelling in patients with group A streptococcal infection
- gas in the tissues of patients with gas gangrene or necrotizing fasciitis type I

Soft tissue or fascial thickening > 3 mm (hyperintensity signal) บนภาพ T2-weight
Peripheral contrast enhancing บนภาพ T1-weight

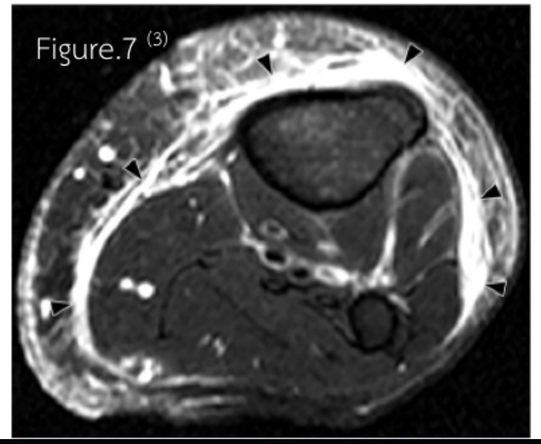


Figure.7 (3)

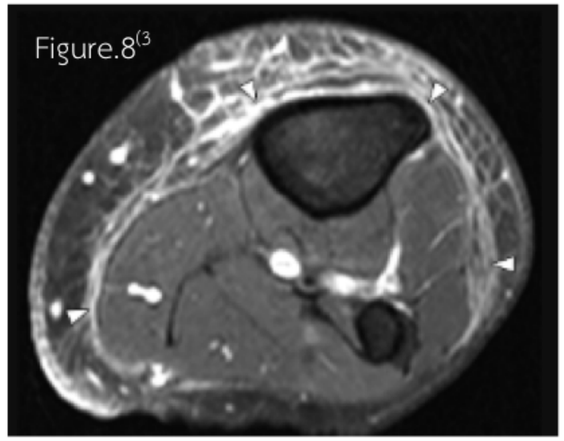


Figure.8(3

Imaging evidence of gas in the tissues, or the presence of crepitus, should prompt immediate surgical consultation.

Diagnostic

Tissue Biopsy, Histologic Tests, and Gram's Staining

- Gram's staining of surgically obtained material is crucial for determining the cause of infection and guiding empirical treatment.
- Percutaneous biopsy and examination of a frozen section has been proposed to aid in the diagnosis of necrotizing infection
- Open surgical inspection and biopsy >> gold standard
  dishwater, foul-smelling discharge on fascia,
  fascial necrosis (non-bleeding fascia) ,loss of tissue resistance

Diagnostic

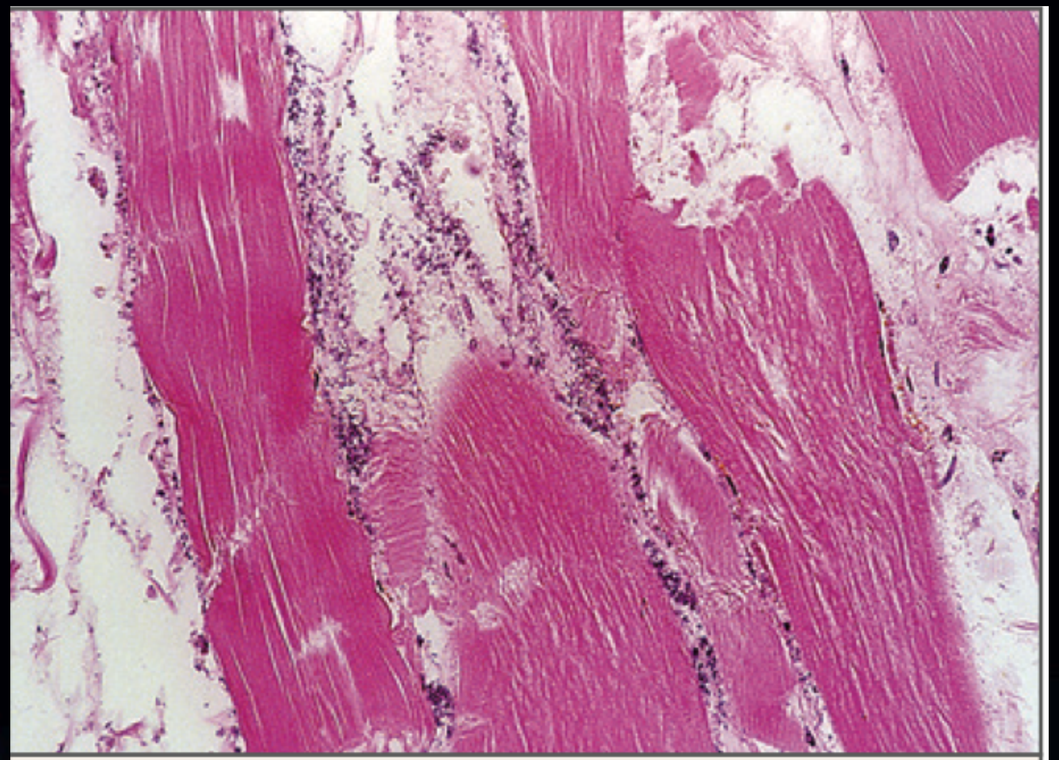


Figure 3. Histopathological Features of Group A Streptococcal Necrotizing Fasciitis and Myonecrosis.

Routine hematoxylin and eosin staining of a muscle specimen from a patient who died from cryptogenic group A streptococcal infection shows the classic features of this infection: widespread tissue destruction, lack of a tissue inflammatory response, and large numbers of bacteria in the tissues.

Necrosis of superficial fascia
PMNs infiltration in dermis and fascia
Fibrinous thrombi of arteries and veins
Angiitis and fibrinous necrosis of vessel wall
Presence of microorganism in necrosis fascia and absent of muscle involvement

histologic criteria

Surrogate Markers for Early Diagnosis

C-reactive protein level of more than 200 mg per liter

The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) scoring system uses

Value	LRINEC score, points
C-reactive protein, mg/L	
<150	0
>150	4
WBC count, cells/mm^3	
<15	0
15–25	1
>25	2
Hemoglobin level, g/dL	
>13.5	0
11–13.5	1
<11	2
Sodium level, mmol/L	
⩾135	0
<135	2
Creatinine level, mg/dL	
⩽1.6	0
>1.6	2
Glucose level, mg/dL	
⩽180	0
>180	1

Table.7 Laboratory Risk Indicator For Necrotizing Fasciitis (LRINEC) [5]

Risk category	LRINEC score, points	Probability of NSTI, %
Low	⩽5	<50
Intermediate	6–7	50–75
High	⩾8	>75

Table.6 Laboratory Risk Indicator For Necrotizing Fasciitis (LRINEC) [5]

Diagnostic

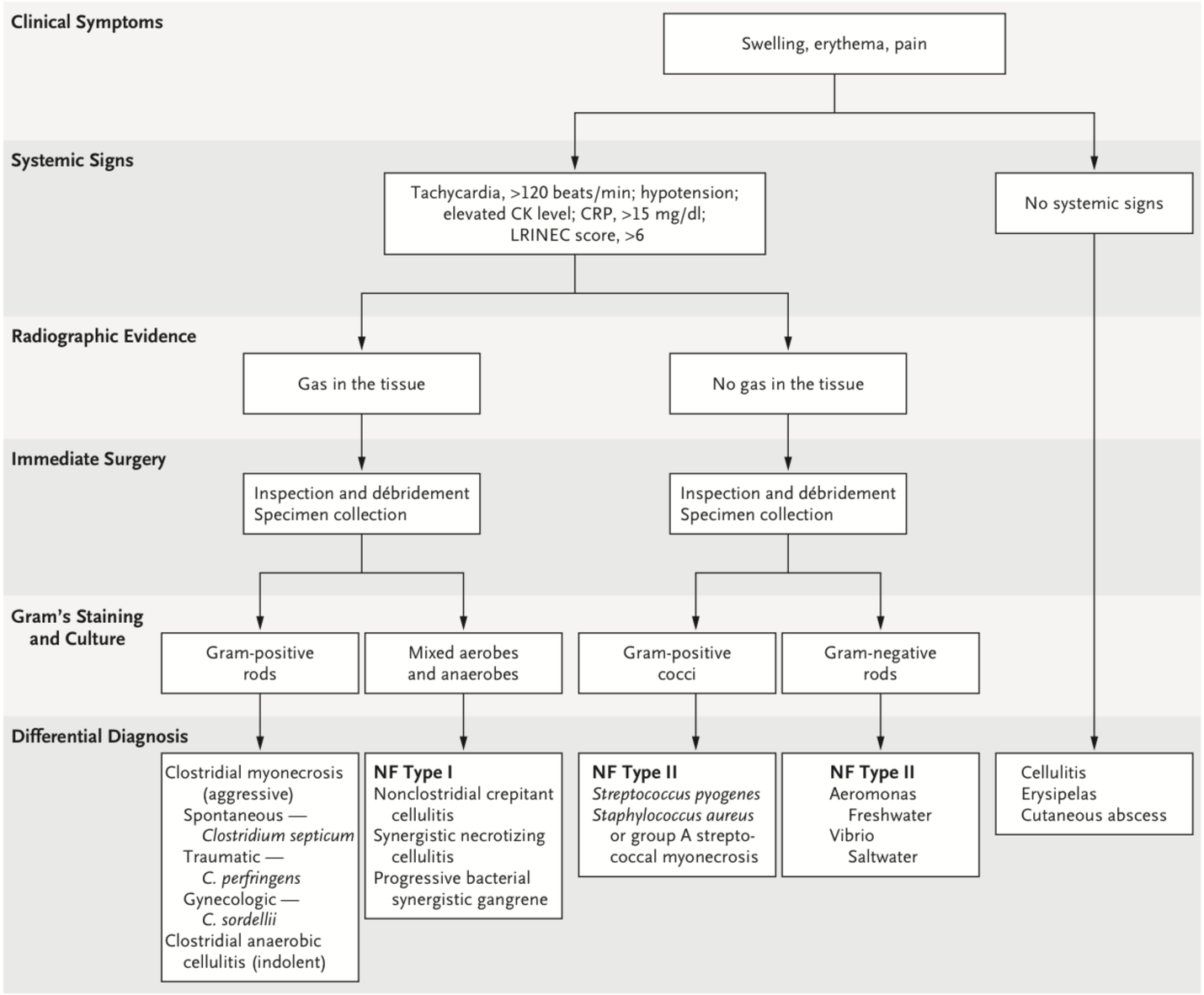


Figure 2. Algorithm for the Diagnosis of Necrotizing Infections.

In the algorithm, early clinical signs and symptoms and available results of laboratory tests and imaging studies are used to establish the diagnosis and cause of a diverse array of skin and soft-tissue infections. CK denotes creatine kinase, CRP C-reactive protein, LRINEC Laboratory Risk Indicator for Necrotizing Fasciitis, and NF necrotizing fasciitis.

Treatment

Treatment

Surgical Intervention

Surgical intervention is the primary therapeutic modality in cases of necrotizing fasciitis and is indicated when this infection is confirmed or suspected.

Features suggestive of necrotizing fasciitis

(1) the clinical findings

(2) failure of apparently uncomplicated cellulitis to respond to antibiotic

(3) profound toxicity; fever, hypotension, or advancement of the SSTI during antibiotic therapy

(4) skin necrosis with easy dissection along the fascia by a blunt instrument

(5) presence of gas in the soft tissues.

- ☑ to determine the extent of infection
- ☑ to assess the need for debridement or amputation
- ☑ to obtain specimens for Gram's staining and culture

Treatment

Surgical Intervention

- Most patients with necrotizing fasciitis should return to the operating room 24–36 hours after the first debridement and daily thereafter until the surgical team finds no further need for debridement.

- Although discrete pus is usually absent, these wounds can discharge copious amounts of tissue fluid, and aggressive fluid administration is a necessary adjunct.

Treatment

Pharmacologic Treatment

In the absence of definitive clinical trials, antimicrobial therapy should be administered until - further debridement is no longer necessary,
- the patient has improved clinically
- fever has been absent for 48–72 hours.

Empiric treatment of polymicrobial necrotizing fasciitis should include agents effective against both aerobes, including MRSA, and anaerobes

vancomycin, linezolid, or daptomycin combined with one of the following:

(1) piperacillin- tazobactam
(2) carbapenem (imipenem-cilastatin, meropenem, and ertapenem)
(3) ceftriaxone plus metronidazole
(4) fluoroquinolone plus metronidazole

Once the microbial etiology has been determined, the antibiotic coverage should be appropriately modified.

Treatment

Table 4. Treatment of Necrotizing Infections of the Skin, Fascia, and Muscle

Type of Infection	First-line Antimicrobial Agent	Adult Dosage	Pediatric Dosage Beyond the Neonatal Period
Mixed infections	Piperacillin-tazobactam plus vancomycin	3.37 g every 6–8 h IV 30 mg/kg/d in 2 divided doses	60–75 mg/kg/dose of the piperacillin component every 6 h IV 10–13 mg/kg/dose every 8 h IV
	Imipenem-cilastatin	1 g every 6–8 h IV	N/A
	Meropenem	1 g every 8 h IV	20 mg/kg/dose every 8 h IV
	Ertapenem	1 g daily IV	15 mg/kg/dose every 12 h IV for children 3 mo-12 y
	Cefotaxime plus metronidazole or clindamycin	2 g every 6 h IV 500 mg every 6 h IV 600–900 mg every 8 h IV	50 mg/kg/dose every 6 h IV 7.5 mg/kg/dose every 6 h IV 10–13 mg/kg/dose every 8 h IV
Streptococcus	Penicillin plus clindamycin	2–4 million units every 4–6 h IV (adult) 600–900 mg every 8 h IV	60 000–100 000 units/kg/dose every 6 h IV 10–13 mg/kg/dose every 8 h IV

Treatment

Staphylococcus aureus	Nafcillin	1–2 g every 4 h IV	50 mg/kg/dose every 6 h IV
	Oxacillin	1–2 g every 4 h IV	50 mg/kg/dose every 6 h IV
	Cefazolin	1 g every 8 h IV	33 mg/kg/dose every 8 h IV
	Vancomycin (for resistant strains)	30 mg/kg/d in 2 divided doses IV	15 mg/kg/dose every 6 h IV
	Clindamycin	600–900 mg every 8 h IV	10–13 mg/kg/dose every 8 h IV
Clostridium species	Clindamycin plus penicillin	600–900 mg every 8 h IV 2–4 million units every 4–6 h IV (adult)	10–13 mg/kg/dose every 8 h IV 60 000–100 00 units/kg/dose every 6 h IV
Aeromonas hydrophila	Doxycycline plus ciprofloxacin or ceftriaxone	100 mg every 12 h IV 500 mg every 12 h IV 1 to 2 g every 24 h IV	Not recommended for children but may need to use in life-threatening situations
Vibrio vulnificus	Doxycycline plus ceftriaxone or cefotaxime	100 mg every 12 h IV 1 g qid IV 2 g tid IV	Not recommended for children but may need to use in life-threatening situations

Treatment

IVIG

- The efficacy of intravenous immunoglobulin (IVIG) in treating streptococcal toxic shock syndrome has not been definitively established.
- As extracellular streptococcal toxins have a role in organ failure, shock, and tissue destruction, neutralization of these toxins theoretically could be beneficial.

Treatment

Care of Critically Ill Patients

Capillary Leak Syndrome

- Circulating bacterial toxins and host mediators cause diffuse endothelial damage. Intravenous fluid requirements may be extremely high (10 to 12 liters of normal saline per day).
- profound hypoalbuminemia (0.5 to 1 g per deciliter) is also common, and replacement with colloid (albumin) may therefore be necessary to maintain oncostatic pressure.

Treatment

Care of Critically Ill Patients

Intravascular Hemolysis

Bacterial hemolysins cause striking and rapid reductions in the hematocrit in the absence of disseminated intravascular coagulopathy. Thus, the hematocrit may be a better indicator of the need for transfusion than the hemoglobin level.

Care of Critically Ill Patients

Cardiomyopathy

- Global hypokinesia, as indicated by echocardiography and cardiac output, is seen in some patients with streptococcal toxic shock syndrome.[8]
- Among survivors, this cardiomyopathy is reversible, fully resolving in 3 to 24 months after infection.

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

Dan L. Longo, M.D., *Editor*

Necrotizing Soft-Tissue Infections

Dennis L. Stevens, Ph.D., M.D., and Amy E. Bryant, Ph.D.

IDSA GUIDELINE

Practice Guidelines for the Diagnosis and Management of Skin and Soft Tissue Infections: 2014 Update by the Infectious Diseases Society of America

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frontiers in SURGERY

REVIEW ARTICLE
published: 29 September 2014
doi: 10.3389/fsurg.2014.00036

Current concepts in the management of necrotizing fasciitis

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THANK YOU