ER-INF COMBINE CONFERENCE

報告者: R3許力云 指導者: VS蕭蔚全 101.08.18

Basic data

• ER visit on day1 08:55 AM

• 檢傷主訴:病患來診為左下肢疼痛

Gender: maleAge: 75 y/oCons: E4V5M6Vital signs:

SpO2: 100%, TPR: 36.5/65/20, BP: 97/41

mmHg
• Triage III

Present illness

- Left leg pain since morning
- 患者自述昨晚有感冒去LMD打IV
- 今天早上開始左腿會痛
- Fever:不確定, Chillness(+)

Past history

Allergy : NKACAD s/p CABG

Physical examination

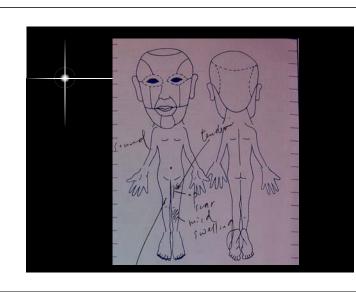
• Consciousness : E4V5M6

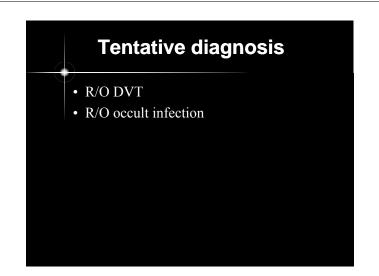
• HEENT : supple neck,

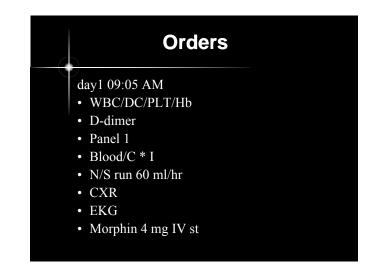
Chest: clear breathing soundAbdomen: soft, no tenderness point

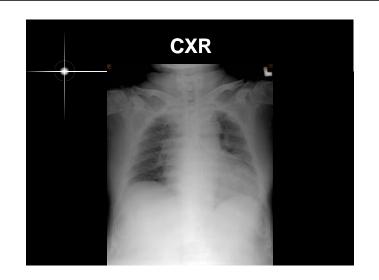
rodomen . son, no tenderness pom

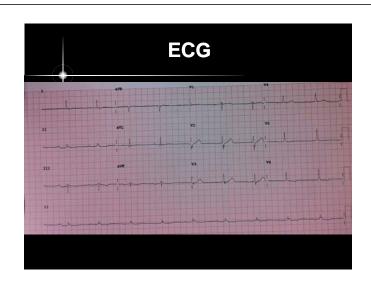
• Extremities : left thigh tenderness

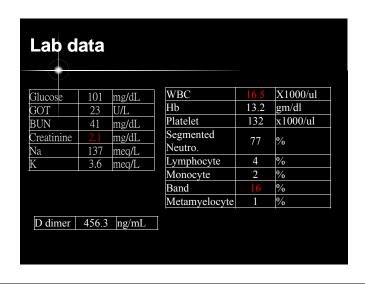


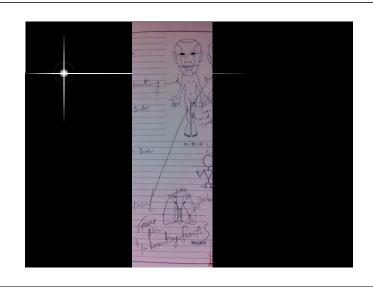


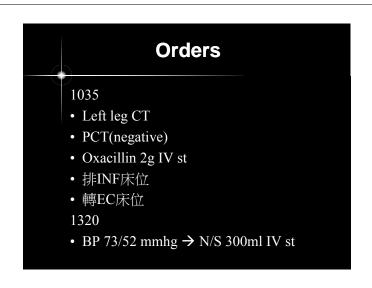


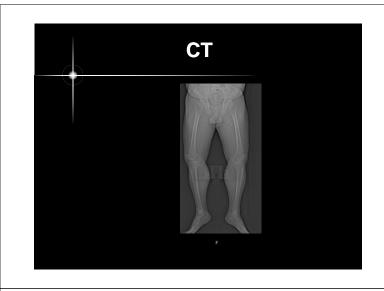


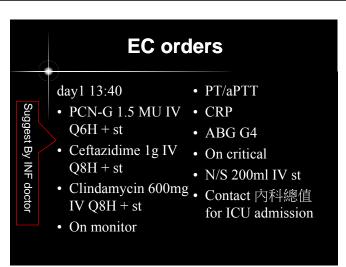


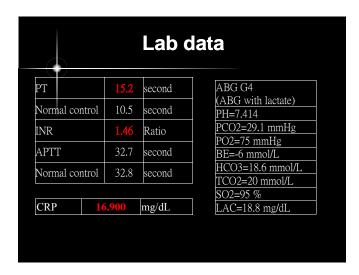


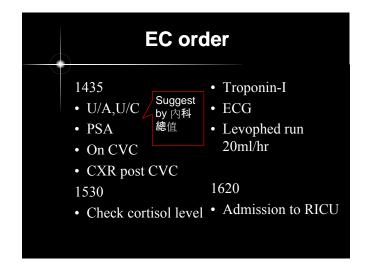


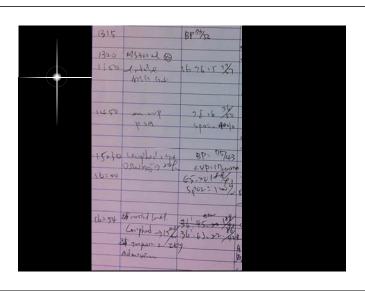


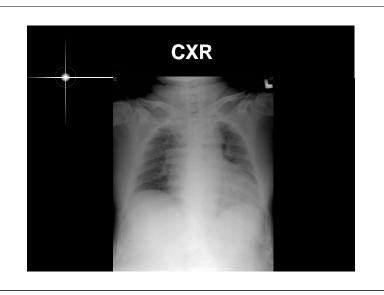


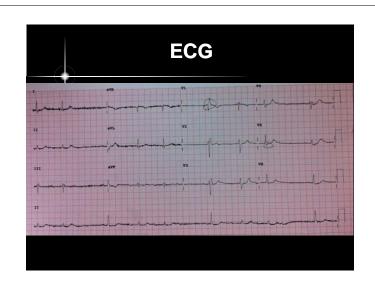


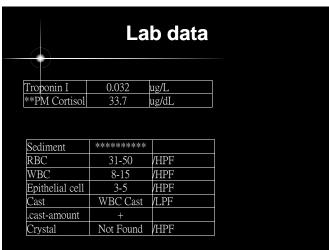










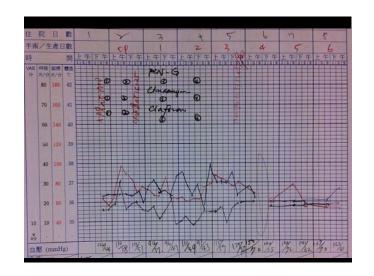


Pre-OP picture

Admission course

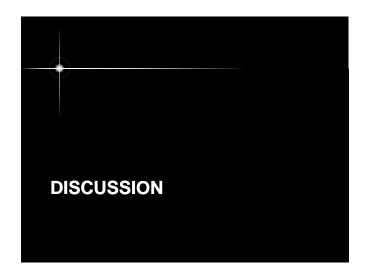
- After admission to RCU INF and PS were consulted for further management
- Fasciotomy was performed at 08/05 evening due to rapid progression of left calf swelling and hemorrhagic bulla formation
- 08/07 blood culture \rightarrow GAS \rightarrow change abx to clindamycin + PCN-G
- Patient was transferred to 5A on 08/08 under PS/INF combine care and s/p several times of debridement,

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Blood culture data
                     SALCHLK2
                      SHLBWCG0
Two bottles of bottle set were positive cultured and final report
pending.
Microscopic finding: Gram (+) coccus in chain
FINAL BLOOD CULTURE REPORT:
..... DECOU COLIUKE KEPURT:
Organism:
1.eta -Streptococcus group A (pyogenes )
/////
Antibiotic/Culture:ST39 eta -Streptococcus group A (pyogenes ) AM CC CMZ CTX CZ E FEP LUX P UA S S S S S S S S S
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Final diagnosis

• Necrotizing fasciitis of left leg, GAS infection with STSS.



Clinical feature of necrotizing fasciitis

- Pain is often out of proportion to PE findings! (Cellulitis may not have this presentation)
- Crepitus and brawny edema on the painful
- Skin bronze or brownish discoloration with
- In Vibro vulnificus infection may have CV collapse and rapid menta

From Tintinali's text book

Clinical Clues to Diagnosis of Necrotizing Soft Tissue Infection

- Skin: ➤ Erythema
- ➤ Tense edema
- Grayish or other discolored wound drainage
- ➤ Vesicles to bullae
- > Necrosis
- > Ulcers Crepitus
- Pain that extends past margin of apparent infection
- Severe pain that appears disproportionate to physical findings
- > Decreased pain or anesthesia at apparent site of infection
- **■** General features:
- Fever
- Tactile temperature
- Diaphoresis
- Tachycardia
- Toxic delirium

	Characteristic
Cellulitis/adipo (nonnecrotiz	
Myonecrosis	Pain that extends past margin
Lymphedema	of apparent infection is
(eosinophilic fasciitis) Phlegmasia ceri	
dolens	Systemic manifestations of severe

Symptoms and sing of necrotizing fasciitis

Table 3. Symptoms/Signs Associated with Necrotizing Soft

Finding	Percent of patients ⁶ (n = 89)	Percent of patients ³¹ (n = 192)	Percent of patients ³² (n = 22)
Erythema	100	66	95
Pain or tenderness beyond margins of erythema	98	73	95
Swelling	92	75	86
Crepitus or skin necrosis	13	31	0
Induration	12	45	
Bullae	45	23	41
Fluctuance	11		
Fever	53	32	
Hypotension	18	11	

Pain is ofetn out of proportion to PE findings! (Cellulitis may not have this presentation)

J Am Coll Surg. 2009 Feb;208(2):279-88

Image tool

- Imaging studies, such as soft tissue x-rays, CT scan and MRI are most helpful if there is gas in the tissue.
- An emergent non-contrast CT examination to assess for the presence of air along the fascial planes may be the most expedient radiographic approach given the associated morbidity with a delay in diagnosis.
- MRI may not be adequate to delineate findings of air along the fascial planes.

Table 5. Laboratory Risk Indicator for Necrotizing Fasciitis Variable C-reactive protein <150 ≥150 15-25 Hemoglobin (g/dL) 0 11-13.5 Sodium (mmol/L) ≥135 <135 Creatinine (mcg/L) ≤141 >141 Glucose (mmol/L) ≤10 >10 A sum ≥6 has a high correlation with necrotizing soft-tissue infection

LAB data to predic NF

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Commom bacteria in necrotizing fasciiting

Table 1. Causative Bacteria of Type 1 and Type 2 Necrotizing Fasciitis

type

polymicrobial infections including anaerobes.

type 2

Streptococcus pyogenes (Group A Streptococcus)
Staphylococcus aureus, including methicillin-sensitive and resistant

Staphylococcus aureus, including methicilini-sensitive and resista

Other microbiological etiologies

Vibrio vulnificus

Aeromonas hydrophila

Enterobacteriaceae (Escherichia coli , Pseudomonas spp., and Klebsiella spp)

Common microbial in type I necrotizing soft tissue infection

Table 2. Common Microbial Causes of Type I Necrotizing Soft-Tissue Infection

Organism	Gram stain	Percent of isolates ⁹ (n = 162)	Percent of isolates ² (n = 272)
Staphylococcus aureus	Gram-positive cocci	16	22
Streptococcus species	Gram-positive cocci	19	17
Klebsiella species	Gram-negative rod	10	
Escherichia coli	Gram-negative rod	7	
Gram-negative bacteria			18
Anaerobic bacteria		7	18
ot 111 1 1		,-	

*Clostridia species (gram-positive rods) are a rare cause of necrotizing softtissue infection.

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\$taging of necrotizing fascii

Table 1. Clinical features of necrotizing fasciitis as the disease progress through clinical stages

Stage 1 (Early)	Stage 2 (Intermediate)	Stage 3 (Late)
Tenderness to palpation (extending beyond the apparent area of skin involvement) Erythema Swelling Warm to palpation	Blister or bullae formation (serous fluid) Skin fluctuance Skin induration	Hemorrhagic bullae Skin anesthesia Crepitus Skin necrosis with dusky discoloration progressing to frank gangrene

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Management of NF

- Treatment of necrotizing fasciitis consists of early and aggressive surgical exploration and debridement of necrotic tissue, antibiotic therapy, and hemodynamic support as needed.
- The best indication for surgical intervention is severe pain, toxicity, fever and elevated CPK with or without radiographic findings.

Treatment of Necrotizing Fasciitis, First-line Antimicrobial Agent, by Infection Type Streptococcus infection Ampicillin-sulbactam plus pipellacillin-tazobactam clindamycin S. aureus infection clindamycin Cefazolin plus Vancomycin(for resistant strains) ciprofloxacin Clindamycin Imipenem/cilastatin Clostridium infection Meropenem Clindamycin Cefotaxime Penicillin plus Vibro vulnificus metronidazole 3rd -generation cephalosporin Plus

Toxic Shock Syndrome (TSS)

- Acute, toxin-mediated illness; characterized by fever, rash, hypotension, multiple-organ involvement and desquamation
- **Staphylococcal** toxic shock syndrome: female during menstruation, with recent surgery or any localized staphylococcal abscess
- **Streptococcal** toxic shock syndrome: associated with severe pain and tenderness signifying infection at a site of local trauma

Infect Dis Clin North Am 1996; 10:727

STSS

tetracycline

Inter Med 49: 1051-1057, 2010

- Isolation of group A Streptococcus from a normally sterile site (blood, CSF, joint, pleural or pericardial fluid)
- Hypotension

Clindamycin

- 2 or more of the following
- (1) Renal impairment (Cr>2, 2X than baseline) (2) Coagulopathy(Platelets ≤100,000, DIC) (3) Liver impairment(AST/ALT > 2倍正常值, 2X than baseline)
- (4) ARDS
- (5) A generalized erythematous macular rash that may
- desquamate
 (6) Soft tissue necrosis

Streptococcal Toxic Shock Syndrome

- Incidence in US: 3.5 cases per 100'000 population
- **Clinical features**:
- Early symptoms: myalgia, malaise, chills, fever, nausea, vomiting and diarrhea; pain at the site of minor trauma (necrotizing fasciitis)
- ➤ Phase 2: tachycardia, fever, tachypnea; increasing pain at site of infection
- ➤ Phase 3: persistent fever, excruciating pain at the infection site; shock and organ failure
- > Evidence of renal impairment precedes hypotension (50%) at the time of admission

N Engl J Med: 1996, 334: p.240

Streptococcal Toxic Shock Syndrome: **Treatment**

- Antibiotic therapy:
- Clindamycin, combined with penicillin
- Surgical exploration and debridement
- Fluid resuscitation
- **IVIG**

N Engl J Med: 1996, 334: p.240

