

Clinical photo and X-ray quiz: an infected toe

臨床相片及 X 光照片猜謎：一只感染了的腳趾

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Case scenario

A 36-year-old manual worker complained of persistent discharge from a wound on his right fifth toe for a week. The patient had a history of right fifth toe contusion, which resulted in bruising and haematoma formation two weeks earlier. At that time he consulted a bonesetter who punctured the haematoma and applied herbal medicine locally. He attended our emergency department (ED) because of discharge from and redness of the puncture wound. The wound discharge was sent for culture and sensitivity tests. X-ray examination of the toe was performed and was normal. He was discharged with a course of ampicillin and cloxacillin. He was called back

to the ED a week later because the wound swab grew methicillin-resistant *Staphylococcus aureus* (MRSA). A clinical photo was taken (Figure 1) and a second X-ray examination (Figure 2) was performed.

Questions

1. What are the findings in the clinical photo and X-ray?
2. What is the diagnosis?
3. What is the treatment?
4. If the sensitivity test reveals the MRSA isolate is sensitive to erythromycin, what is the clinical implication? What laboratory investigation could be done to confirm the suspicion?



Figure 1. Clinical photo of the right 5th toe.



Figure 2. X-ray of the right 5th toe.

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Discussion

The clinical photo reveals small chronically infected ulcers with granulation formation over the dorsal aspect of the right fifth toe. The surrounding skin showed pinkish discoloration. The X-ray reveals an osteolytic lesion and soft tissue swelling over the distal phalanx of the fifth toe. Based on the above history and physical findings, the patient is suffering from MRSA osteomyelitis of the distal phalanx of the right fifth toe. Conventional radiography is usually recommended as the initial investigation,¹ however, plain X-ray findings may be normal in the first 10–21 days of an infection. Early X-ray findings include soft tissue swelling and localized osteopenia. Subsequent findings may include cortical lucencies, periosteitis, involucrum formation, and pathological fracture. In our case, the initial X-ray was normal, cortical lucencies were present only one week after the initial presentation. Emergency physicians should be suspicious of the diagnosis, basing on the history and physical examination. CT scan and MRI may be indicated for early diagnosis¹ but their usefulness depends on the cost, local availability and urgency of the diagnosis.

The patient was admitted into the orthopaedic department. Infection was confirmed down to the bone during the debridement of the wound. Wound culture was repeated and again grew MRSA. The isolate was sensitive to amikacin, fusidic acid, rifampicin, co-trimoxazole and vancomycin. The standard treatment for MRSA osteomyelitis is surgical debridement plus a long course of parenteral vancomycin. However, this treatment carries the risks of complications associated with long hospital stay and prolonged use of intravenous catheter for the vancomycin administration, not to mention the enormous associated personal and economic costs.² Some authors suggest a potential role for oral antibiotic regimens at home in suitable and compliant patients.^{2,3} Early consultation with a microbiologist is therefore advisable. In our case, the clinical microbiologist was consulted and he suggested two weeks of intravenous vancomycin. The patient was then discharged with four weeks of fusidic acid and rifampicin. However, he developed

neutropenia three weeks after the above oral antibiotics and was readmitted to hospital for another week of intravenous vancomycin. Finally he recovered with follow-up X-ray study showing no further bone erosion.

If the MRSA isolate is sensitive to erythromycin, it may be a community-acquired MRSA infection. The presence of staphylococcal chromosomal cassette type IV gene (SCCmec) in the isolate and genetic typing of the isolate should be determined. The *mecA* gene codes for *Staphylococcus aureus* methicillin resistance. In community-acquired MRSA, *mecA* is carried within a genetic element called staphylococcal chromosomal cassette *mec* (SCCmec) type IV, which is distinct from the SCCmec (types I, II, III) typically found in hospital-associated MRSA.⁴ One study has identified MRSA in 51% of skin and soft tissue infections in a US urban ED and most appeared to be community-associated.⁵ Community-acquired MRSA tends to be more susceptible to a broader array of antibiotics.⁴ Ho et al reported the first case of community-acquired MRSA in Hong Kong in 2004.⁶ The isolate reported was susceptible to the following antibiotics: gentamicin, erythromycin, clindamycin, fusidic acid, ciprofloxacin, co-trimoxazole, tetracycline, chloramphenicol, rifampicin and vancomycin.⁶ Many community-acquired MRSA were found to carry the genes for Panton-valentine leukocidin, a virulence factor that is associated with furunculosis and necrotizing pneumonia.⁴

Skin and soft tissue infection such as cellulitis and wound infection are empirically treated with cloxacillin and penicillin (or ampicillin) to cover *Staphylococcus aureus* and streptococci respectively.⁷ In our case, the initial ED antibiotic regimen was likely to fail because of the presence of MRSA. However, the prevalence of community-acquired MRSA in Hong Kong is not known. Prospective epidemiologic studies of ED patients with clinically important skin and soft tissue infections should be conducted to define the local extent of community-acquired MRSA and the pattern of in vitro antimicrobial susceptibility. The result of these studies may change our future choice of empiric therapy for skin and soft tissue infection in the ED.

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