Osteomyelitis complicating secondarily infected atopic eczema: two case reports and a narrative literature review

Josiah T. Masuka¹,²*, Katherine Troisi³ and Zamambo Mkhize¹,²

Abstract

Background: Atopic eczema is a relapsing, itchy chronic cutaneous inflammatory disease that commonly affects children. The disease is often complicated by cutaneous infections such as eczema herpeticum, eczema vaccinatum and a varied number of bacterial infections – impetigo, cellulitis and erysipelas. However, rare case reports of infective endocarditis, otitis media and osteo-articular infections have been associated with atopic eczema. These associations possibly represent the extracutaneous infectious complications of atopic eczema.

Case presentation: Here we present two cases of osteomyelitis in HIV negative children with habitual scratching of poorly managed and/or uncontrolled atopic eczema respectively. Both cases presented to the orthopaedic surgeons and were admitted as acute phalangeal osteomyelitis and acute – on – chronic tibial osteomyelitis respectively. The first case was an 8 year old girl who had moderate-severe poorly-controlled atopic eczema and contiguously spread phalangeal osteomyelitis. The second case was an 11 year old pre-pubertal boy who had untreated atopic eczema and tibial osteomyelitis possibly from haematogenously spread Staphylococcus aureus infection. Both were successfully discharged from hospital and currently have well controlled eczema. The 11 year old patient is also being reviewed monthly by the orthopaedic surgeons and is chronic suppressive antibiotics. He may require sequestrectomy, should it be needed.

Conclusions: Invasive staphylococcal and streptococcal osteo-articular (OA) infection can arise as an extracutaneous infectious complication of poorly controlled atopic eczema. It is more common in the 3 to 15 year age group and especially in boys with a septic arthritis to osteomyelitis ratio of around 29:5. Clinicians should maintain a high index of suspicion in patients with moderate-severe atopic eczema and they ought to promptly manage these OA infections with intravenous antibiotics to avoid further complications.

Keywords: Osteomyelitis, Osteo-arthritis, Atopic eczema, Secondary infection, Staphylococcus aureus

Background

Osteomyelitis has rarely been associated with secondarily infected atopic eczema [1–4]. In addition, phalangeal and/or hand osteomyelitis has rarely been described in literature as contiguous contamination secondary infection in atopic eczema or on the hands [5, 6]. In one reported case series, three cases of acute phalangeal osteomyelitis due to habitual scratching in children with severely infected atopic eczema has been documented [5]. In all three children, no apparent indicators of haematogenous bacterial dissemination such as elevated erythrocyte sedimentation rate (ESR) or pyrexia were observed [7]. On the other hand, invasive staphylococcal bacteremia and haematogenous osteo-articular infections have also been reported sporadically in association with atopic eczema [3, 8, 9]. These cases represent some of the infrequently reported invasive complications of secondarily infected atopic eczema which require prompt and definitive management to prevent further complications [4].

Here we present two unusual cases of osteomyelitis possibly resultant from secondarily infected atopic...
eczema in African children. These children were referred on the same day to our dermatology clinic from orthopaedic surgeons for the management of the underlying atopic eczema after initial admission for phalangeal and tibial osteomyelitis respectively. In addition, a narrative literature review of similar case presentations has also been carried out to describe the demographic and clinical features of osteo-articular infections associated with

| Case  | Author - Year | Diagnosis                                      | OA type | Age (Yr) | Gender | Reported atopic eczema severity | Micro-organism |
|-------|---------------|------------------------------------------------|---------|----------|--------|---------------------------------|---------------|
| 1     | Patel D; 2015 [4] | Septic arthritis and osteomyelitis - left shoulder | OAOM    | 8        | F      | Severe                          | MRSA          |
| 2     | Sayaka I; 2013 [21] | Costochondral abscess                          | OA      | 20       | M      | NS                             | NS            |
| 3     | Tsutsumi R; 2010 [25] | Cervical spondylitis spondylitis              | OM      | 3        | M      | Severe                          | SA            |
| 4     | Kitamura S; 2000 [8] | Septic arthritis - hip                         | OM      | 15       | F      | Recent flare                    | SA            |
| 5     | Boiko S; 1988 [5] | Osteomyelitis - distal phalanx                 | OM      | 4        | F      | Severe                          | MSSA, Strep   |
| 6     | Boiko S; 1988 [5] | Osteomyelitis - distal phalanx                 | OM      | 2        | F      | Severe                          | MSSA, Strep   |
| 7     | Boiko S; 1988 [5] | Osteomyelitis - nail plate                     | OM      | 4        | M      | Severe                          | MSSA          |
| 8     | Sharma A; 1997 [2] | Osteomyelitis - fibula                         | OM      | 13       | M      | Moderate-severe                 | MSSA          |
| 9     | Nassif A; 1994 [1] | Septic bursitis - mid-tibial, olecranon        | OA      | 60       | M      | Severe                          | MRSA          |
| 10    | Kusunoki T; 2015 [19] | Right hip osteoarthritis                      | OA      | 0.25     | M      | Severe                          | MSSA          |
| 11    | Kusunoki T; 2015 [19] | Left hip osteoarthritis                       | OA      | 11       | M      | Recent flare                    | NS            |
| 12    | Numazaki H; 2017 [22] | Tibial osteomyelitis (complication of ACL reconstruction) | OM      | 24       | M      | Severe                          | MSSA          |
| 13    | Ohno et al.; 2000 [19] | Right sacroiliac                              | OA      | 13       | M      | Moderate-severe                 | MSSA          |
| 14    | Ueda et al.; 2001 [19] | Left hip osteoarthritis                       | OA      | 3        | M      | Unknown                         | SA            |
| 15    | Ono et al.; 2003 [19] | Knee SA                                       | OA      | Infant   | Unknown | Moderate-severe                 | SA            |
| 16    | Hidaka et al.; 2004 [19] | Right knee                                    | OA      | 5        | M      | Moderate-severe                 | MRSA          |
| 17    | Yamagata et al.; 2004 [19] | Right hip and knee SA                         | OA      | 12       | M      | Moderate-severe                 | MRSA          |
| 18    | Kimura et al.; 2005 [19] | Left hip                                      | OA      | 0.25     | F      | Moderate-severe                 | MSSA          |
| 19    | Nakamura & Fujioka; 2006 [26] | Left hip                                     | OA      | 5        | M      | Moderate-severe                 | MSSA          |
| 20    | Moriwaki et al.; 2006 [19] | Left sacroiliac                              | OA      | 21       | F      | Unknown                         | MSSA          |
| 21    | Nakamura & Fujioka; 2006 [26] | Right knee and femur                          | OA      | 0.83     | M      | Moderate-severe                 | MRSA          |
| 22    | Nakamura & Fujioka; 2006 [26] | Right hip                                    | OA      | 0.5      | F      | Moderate-severe                 | MSSA          |
| 23    | Nakamura & Fujioka; 2006 [26] | Right hip                                    | OA      | 0.58     | M      | Moderate-severe                 | MSSA          |
| 24    | Hiyan et al.; 2007 [19] | Left hip                                      | OA      | 11       | M      | Unknown                         | NS            |
| 25    | Matsushita et al.; 2008 [19] | Left knee                                    | OA      | 1        | F      | Mild                            | MSSA          |
| 26    | Nagai et al.; 2008 [19] | Left tibia                                    | OA      | 3        | M      | Mild                            | NS            |
| 27    | Nagai et al.; 2008 [19] | Left femur                                    | OA      | 3        | M      | Moderate-severe                 | MSSA          |
| 28    | Nagai et al.; 2008 [19] | Left hip                                      | OA      | 0.92     | F      | Moderate-severe                 | MSSA          |
| 29    | Suzuki et al.; 2009 [19] | Right knee                                    | OA      | 7        | F      | Moderate-severe                 | SA            |
| 30    | Kinugasa et al.; 2009 [19] | Right hip                                    | OA      | 23       | F      | Unknown                         | Strep         |
| 31    | Hashi et al.; 2012 [27] | Right hip                                    | OA      | 5        | F      | Moderate-severe                 | MRSA          |
| 32    | Yamagata et al.; 2012 [19] | Right hip                                    | OA      | 12       | M      | Moderate-severe                 | MRSA          |
| 33    | Yasuda & Nisimatsu; 2012 [28] | Right sacroiliac                             | OA      | 15       | M      | Moderate-severe                 | Strep         |
| 34    | Kyo; 2014 [23] | Knee                                         | OA      | 13       | M      | NS                             | SA            |
| 35    | Kyo; 2014 [23] | Knee                                         | OA      | 27       | M      | NS                             | SA            |
| 36    | Current case 1 | Phalangeal osteomyelitis                      | OM      | 8        | F      | Mild                            | NS            |
| 37    | Current case 2 | Tibial osteomyelitis                          | OM      | 11       | M      | Severe                          | NS            |

**Abbreviations:** OA Osteoarthritis, OM Osteomyelitis, MRSA Methicillin Resistant Staph. Aureus; SA Staph. Aureus, Strep Streptococcus, NS Not specified
atopic eczema. The occurrence of the two presented cases in light of similar previous case reports may not be coincidental and calls upon clinicians to be aware of the potential complications of atopic eczema [4].

Case presentation

Case 1
An 8 year old, black female child was referred to the dermatology team from orthopaedic surgeons with a 3 month history of a swollen right index finger. On further enquiry, the patient was noted to be atopic with comorbid chronic asthma and atopic eczema. The child was being managed on aqueous cream baths, topical beta-methasone cream, a non-sedating antihistamine – loratadine and liquid paraffin as an emulsifying ointment and an asthma medication pump. On the current dermatology consultation, the child’s caregiver mentioned that the child had been scratching the itchy right index finger. In the period prior to the presentation, the child’s finger got swollen and was painful prompting the hospital visit and subsequent admission. No history of phalangeal trauma or diabetes mellitus was elicited from the patient’s caregiver, which was also confirmed in subsequent tests. The child was HIV negative.

Examination revealed a swollen, mildly fluctuant index and middle phalanges with draining sinuses. There was an eczematous plaque with scaling and no lichenification. The fingers were tender and warm to palpation. No dysmorphic features were observed and the child’s body temperature was unremarkable at 37.2 °C. The X-ray findings were consistent with osteomyelitis and the child was admitted by the orthopaedic surgeons for intravenous antibiotics for intravenous antibiotics (amoxicillin/clavulanic acid, due to the unavailability of cloxicillin in our institution, which would be the drug of choice) to control the acute infection as surgical drainage was not warranted. The patient was discharged a week later to complete a 1 month oral antibiotic therapy course at home and to continue with her eczema medication. On review, the cellulitis had healed and the eczema lesions had been unmasked. The phalanges displayed the eczematous plaque with diffusely demarcated borders. No weeping or crusting was observed. Her initial calculated (Eczema Area and Severity Index) EASI score and severity levels on presentation were 9.60 and moderate severity respectively [10]. The patient was HIV negative.

However no pus swab was recorded in the patient’s charts. His initial calculated EASI score and severity levels on presentation were 9.60 and moderate severity respectively [10].

The patient was initially treated by orthopaedic surgeons as an acute-on-chronic osteomyelitis and intravenous amoxicillin/clavulanic acid was administered to manage the acute sepsis together with paracetamol as analgesia. On review by the dermatology team, the diagnosis was modified to chronic osteomyelitis secondary to infection with underlying chronic eczema. Potassium permanganate baths, topical betamethasone, silver-sulphur dialazine, an emulsifying ointment and a non-sedating antihistamine – loratadine were added to his medical management. The patient was subsequently discharged to complete a course of rifampicin and trimethoprim/sulfamethoxazole, (orthopaedics current chronic osteomyelitis protocol for older children/adult patients) and was to be reviewed in both the dermatology and orthopaedic out-patient clinics. Further orthopaedic management has consisted of monthly clinic reviews, chronic suppressive antibiotics and sequestrectomy might be considered, should it be needed.
Discussion and conclusions
In this case series, we have presented two unusual cases of osteomyelitis associated with severely infected atopic eczema in HIV uninfected children. The cases represent two of the three generally accepted mechanisms of osteomyelitis infection – contiguous spread and haematogenous spread [11]. The first case developed possibly after direct infection of the distal phalangeal bone due to an overlying septic eczema focus. The second case possibly had haematogenous spread to the tibial metaphysis. This was secondary to a bacteraeemia resultant from the septic eczema, which then settled in the metaphysis as commonly happens in immature long bones [12, 13]. Both children had habitual, excessive scratching of their dry, fissured skin in poorly or untreated eczema respectively. In addition, there were no other apparent sources of infection in both cases.

The phalangeal osteomyelitis case is similar to three cases reported by Boiko et al. [5]. There was insidious onset of radiologically confirmed osteomyelitis without any associated fever or ESR elevation. This signified a localized infection as opposed to the second case which presented with tibial osteomyelitis associated with a raised ESR. The raised ESR in the second case indicates haematogenous spread, possibly of invasive staphylococcal infection presenting as osteomyelitis [5]. Even though no bacterial cultures were done prior to initiation of antibiotic therapy in both cases, we strongly suspect that Staphylococcus aureus was the causative organism. The lack of bacterial culture results may be a major limitation to our study. However, staphylococcal bacterial colonization has been shown to be more common in atopic eczematous skin compared to normal skin [14].

Furthermore, it has been postulated that recurrent bacterial and viral infections often complicate atopic eczema possibly due to an interplay between staphylococcal enterotoxins (super-antigens), cutaneous barrier defects and the dysfunctional cutaneous innate immune system [15]. The latter is characterized by increased skin pH and decreased antimicrobial peptides – human β defensins and cathelicidins coupled to increased CD4+ Th2 cytokines - IL-4 and IL-13 [15, 16]. Decreases in antimicrobial proteins are caused by the skewing of the lymphocytic response towards the CD4+ Th2 direction with increased cytokines IL-4 and IL-13 resultant from increased keratinocyte derived thymic stromal lymphoprotein expression in atopic eczema [15]. Additionally, the relapsing, itchy chronic inflammation of atopic eczema is also perpetuated by the reduced staphylococcal bacterial inhibition and low “natural moisturizing factor” (NMF) resultant from the lack-of-function filaggrin gene defects [15, 17]. The reduced NMF exacerabtes atopic eczema due to the dry skin aggravating and perpetuating the “itch – scratch” cycle [15, 18]. Chronic scratching of the itchy skin potentially worsens the already weak cutaneous barrier, thereby facilitating the entry of allergens and pathogens into the skin [4, 19]. Consequently, cutaneous and extra-cutaneous infections occur with higher incidence in atopic eczema patients compared to non-atopic eczema patients [20]. Eczema herpeticum, erysipelas, impetigo, cellulitis, otitis media and streptococcal throat infection have been widely documented in patients with atopic eczema [15, 17, 20].

However, clinicians need to keep a high index of suspicion for invasive staphylococcal infection especially in toxaemic eczematous patients [8, 16]. Systemic infections such as osteo-articular infections, infective endocarditis and pneumonia have rarely been diagnosed in association with eczema [1, 2, 5, 9, 16]. Overall, a total of at least 35 case reports of invasive osteo-articular infection have been published in association with atopic eczema including 6 case reports first collated in 2005 by Benenson et al. [9]. Most of these retrieved case reports, 30 (85.71%) have been reported in Japanese patients with the remainder described in other settings. Twenty nine (29) out of the 35 case reports were on septic arthritis whilst 5 were on osteomyelitis and 1 case report had both osteomyelitis and septic arthritis [4, 9, 19, 21–23]. Septic-arthritis mostly involved the hip joint 14 (48.28%) followed by the knee joint 8 (27.59%) Table 1.

The majority of the osteo-articular cases, 28 (80.0%) were observed in paediatric patients below 18 years of age whilst the remainder was mostly in young adults. The median and average ages of the patients described in the case reports were 6 (IQR; 2.75:15.0) years and 10.72 ± 12.20 years respectively with a 22:12 M; F ratio. The mode of the presumed severity level of the atopic eczema on presentation with osteo-articular disease was moderate – severe disease. Staphylococcus aureus was cultured in 28 (80.0%) whilst streptococcus (β haemolytic group B streptococcus or Streptococcus viridans) was cultured in 6 (17.14%) of the retrieved case reports. Methicillin resistant and methicillin sensitive Staphylococcus aureus were documented in 8 (22.86%) and 13 (37.14%) case reports of osteo-articular infection respectively. Both Staphylococcus aureus (all MRSA) and streptococcus were both cultured in 3 (8.57%) whilst the culture result was unknown in 4 (11.43%) of the case reports.

Japanese individuals predominated in the retrieved case reports [19] possibly because of the increased use of sensitive Magnetic Resonance Imaging (MRI) and bone scans [23, 24] for patient work-up in Japan compared to Africa for instance. Nevertheless, a significant number were also observed in other populations and were associated with osteomyelitis. This underscores the need for aggressive management of the underlying atopic eczema and a high index of suspicion for osteo-articular infections as they can cause growth disturbances, deformities...
eczematous lesions sometimes with subsequent systemic infection. Patients present with a persistent fever, an elevated ESR and/or CRP. Various studies have shown that atopic dermatitis is associated with an increased risk of infection. This is partly due to an impaired local and systemic immune response in atopic dermatitis patients. The infection may be local or systemic, and may result in joint infection or septic arthritis. In severe cases, acute haematogenous osteomyelitis can develop, which may lead to serious complications and even death [24]. This is especially so in patients who present with a persistent fever, an elevated ESR and/or CRP.

**Abbreviations**

CD: Cluster of Differentiation; CRP: C Reactive Protein; ESR: Erythrocyte Sedimentation Rate; HIV: Human Immunodeficiency Virus; NMF: Natural moisturizing factor

**Acknowledgements**

The authors are grateful for the assistance provided by Sr Z Ndlovu and Sr L Ndaba and other staff at Edendale Hospital in preparing this manuscript.

**Authors’ contributions**

JM, KT and ZM collected the clinical information about the two cases. JM and KT reviewed the manuscript and approved the final version.

**Competing interests**

The authors declare that they have no competing interests.

**Availability of data and materials**

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

**Funding**

No external sources of funding were used for this study.

**Consent for publication**

Written informed consent for publication of the patients’ clinical details and/or clinical images was obtained from the patients/guardians/relative of the patients.

**Ethics approval and consent to participate**

This case report was approved by the Edendale Hospital Ethics Committee.

**Consent for publication**

All patient data was de-identified.

**Ethics approval**

This case report was approved by the Edendale Hospital Ethics Committee.

**Availability of data and materials**

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

**Acknowledgements**

The authors are grateful for the assistance provided by Sr Z Ndlovu and Sr L Ndaba and other staff at Edendale Hospital in preparing this manuscript.

**Authors’ contributions**

JM, KT and ZM collected the clinical information about the two cases. JM and KT reviewed the manuscript and approved the final version.

**Funding**

No external sources of funding were used for this study.

**Consent for publication**

Written informed consent for publication of the patients’ clinical details and/or clinical images was obtained from the patients/guardians/relative of the patients. Copies of the consent forms are available for review by the Editor of this journal.

**Competing interests**

The authors declare that they have no competing interests.

**Author details**

1. Department of Dermatology, Nelson R Mandela School of Medicine, Private Bag X7, Congella, Durban 4013, South Africa.
2. Department of Dermatology, Edendale Hospital, 89 Selby Msimang Rd, Pleissislaer, Pietermaritzburg 3201, South Africa.
3. Department of Orthopaedics, Edendale Hospital, 89 Selby Msimang Rd, Pleissislaer, Pietermaritzburg 3201, South Africa.

**Received:** 11 September 2019 **Accepted:** 22 December 2019

**Published online:** 03 February 2020

**References**

1. Nassif A, Smith D, Hanifin J. Olecranon and pretibial bursitis: coincidence or association. J Am Acad. 1994;30:37–42.
2. Sharma A. Atopic dermatitis and staphylococcus aureus - induced osteomyelitis - a peculiar association in a case. Pediatr Dermatol. 1997;14(6): 453–5.
3. Carr T, Avila P. Recurrent deep-seated staphylococcus aureus infections of unclear aetiology. J Clin Immunol. 2011;31:529.
4. Patel D, Jahnke M. Serious complications from staphylococcal aureus in atopic dermatitis. Pediatr Dermatol. 2015;32(6):792–6.
5. Boiko S, Kaufman R, Anne W, Lucky A. Osteomyelitis of the distal phalanges in three children with severe atopic dermatitis. Arch Dermatol. 1988;124: 418–23.
6. Pinder R, Barlow G. Osteomyelitis of the hand. J Hand Surg Eur Vol. 2016; 41(4):341–40. https://doi.org/10.1177/1753193415612373.
7. Nade S. Acute haematogenous osteomyelitis in infancy and childhood. J Bone Joint Surg Br. 1983;65:109–19.
8. Kitamura S, Nakayam Y, Shirai Y, Hashiguchi S, Kim R. Septic arthritis of the hip associated with atopic dermatitis - a case report. J Nippon Med Sch. 2000;67(6):646–7.
9. Benenson S, Zimhony O, Dahan D, Solomon M, Raweh D, Scheinger Y, Yinnon A. Atopic dermatitis - a risk factor for invasive staphylococcal aureus infections: two cases and review. Am J Medicine. 2005;118(9):1048.
10. Leshem Y, Hajar T, Hanifin J, Simpson E. What the EASI score tells us about the severity of atopic dermatitis - an interpretability study. Br J Dermatol. 2015;172(5):1353–7.
11. Birt M, Anderson D, Toby E, Wang J. Osteomyelitis: recent advances in pathophysiology and therapeutic strategies. J Orth. 2017;14:45–52.
12. Paakkonen M, Killo M, Peitola H, Killo P. Antibiotic treatment and surgery for acute haematogenous calcanea osteomyelitis of childhood. J Foot Ankle Surg. 2015;54:480–3.
13. Francis J, Robson J, Wong D, Walsh M. Chronic recurrent multifocal Q fever osteomyelitis in children: an emerging clinical challenge. Pediatr Infect Dis J. 2005;24(10):921–4.
14. Higaki S, Morohashi M, Yamagishi T, Hasegawa Y. Comparative study of staphylococci from the skin of atopic dermatitis patients and from healthy subjects. Int J Dermatol. 1999;38(4):265–9.
15. Ong P, Leung D. Bacterial and viral infections in atopic dermatitis: a comprehensive review. Clin Rev Allerg Immunol. 2016;51(3):329–37.
16. Tsuibo Y, Yumoto T, Toyokawa T, et al. Staphylococcus aureus bacteremia complicated by psoas abscess and infective endocarditis in a patient with atopic dermatitis. Case Rep Infect Dis. 2017;2017:1–4. https://doi.org/10.1155/2017/4920182.
17. James W, Elston D, Berge T, Neuhau S. Andrews' diseases of the skin: clinical dermatology. 12th ed. Philadelphia: Elsevier; 2016.
18. Murota I, Katayama I. Escharotomizing factors of itch in atopic dermatitis. Allergol Int. 2016;66(1):8–13.
19. Kusunoki T, Shimozono F, Maruki M, Futarni T, Fuji T. Septic arthritis and atopic dermatitis: 2 cases and a review of the recent literature. J Investig Allerg Clin Immunol. 2015;25(3):214–36.
20. Nair S, Silverberg J. Association between childhood atopic dermatitis and cutaneous, extracutaneous and systemic infections. Br J Dermatol. 2018;178:1466–9.
21. Sayaka I, Kenich K, Hideaki T, Mowa M, Tomoya F, Katsuova E. A case of severe atopic dermatitis complicated by costal cartilage junction abscess. J Jap Prim Care Assoc. 2013;36(4):315–7.
22. Numazaki H, Kobbayashi H, Yoshida K, Hakozaki M, Konno S. Prolonged infection at the tibial bone tunnel after anterior cruciate ligament reconstruction. Fukushima J Med Sci. 2017;63(2):121–5.
23. Kyo H, Hayashi M, Yamawaki Y, Watanabe Y, Nakamura M, Takeuchi H, Ota S, Onishi E, Ikavi K, et al. Atopic dermatitis as a potential portal of septic arthritis. J Arthritis. 2014;2:32.
24. Yeo ARM. Acute haematogenous osteomyelitis in children. BMJ. 2014;348: g66.
25. Tsuchimi REA. A case of cervical spondylitis spondylitis with Staphylococcus aureus bacteremia that is considered to be the infection route of severe atopic dermatitis skin lesions. Neurology. 2010;5:516–8.
26. Nakamura K, Fujikawa S. Septic arthritis in children. Jap J Pediatr. 2006;59:115–20.
27. Hashi R, Miyake F, Takehara A, Sukamoto A, Yamauchi J, Wada K, Nakauchi S, Ohshima T. Five cases of septic arthritis in childhood. Jap J Pediatr. 2012; 65:217–21.
28. Yasuda T, Nisimatsu H. Acute sacroiliac joint infection in a rugby player with atopic dermatitis. Clin J Sport Med. 2012;22:508–10.

**Publisher's Note**

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.