

Morphological aspects of osteomyelitis: a mini-review

Sato, SK.^{1*} and Pimenta-Rodrigues, MV.²

¹Faculdade de Ciências Biológicas e da Saúde, Centro Universitário da Grande Dourados – UNIGRAN, Rua Balbina de Mattos, 2121, Jardim Universitário, CEP 79824-900, Dourados, MS, Brasil

²Departamento de Microbiologia e Imunologia, Faculdade de Medicina, Universidade do Oeste Paulista – UNOESTE, Rua José Bongiovani, 700, Cidade Universitária, CEP 19050-900, Presidente Prudente, SP, Brasil
E-mail: sidneysato@hotmail.com

Abstract

A revision and up-to-date in bone infection is made. We point out the relevance of bacterial infections in the etiology of this pathology. The pathogenesis and forms of the infection were revised. Concerning the clinical presentation, a classification of these infections as to their evolution was addressed. In the diagnosis of osteomyelitis the importance of radiographic aspects are stressed.

Keywords: osteomyelitis, morphological aspects.

1 Introduction

1.1 General considerations

Osteomyelitis is the term used to describe infection of the bone. Morbidity and mortality of such infections have declined since the correct administration of appropriate antimicrobial and surgical treatments despite the increase in antibiotic-resistant microorganisms. The difficulty in the treatment of osteomyelitis can be attributed to the antibiotic half-life, blood flow deficit in the infected area and systemic toxic effect of the antibiotic (COSTERTON, 2005). By their pathogenesis they may be classified as hematogenous and secondary osteomyelitis, and these include those derived from a contiguous focus of infection, such as postoperative osteomyelitis in surgical and prosthetic replacement and the post-traumatic fractures of long bones (CRÉ MIEUX and CARBON, 1997; MADLER, CRIPPS and CALHOUN, 1999; SMELTZER and GILLASPY, 2000; BERENDT and BYREN, 2004).

The following classification describes four stages of development of osteomyelitis: stage 1: the infection is confined to the bone marrow cavity, stage 2: the infection is superficial, involving only cortical bone whose origin is via a direct inoculation or contiguous focal infection, stage 3: localized infection involving the cortical and the medullary bone; stage 4: diffuse infection involving the entire thickness of the bone. (CIERNY, MADER and PENNINCK, 2003).

According to their evolution, symptoms, clinical signs and pathological changes they can be classified as acute and chronic. Due to the bone tissue characteristics such as ischemia and slow recovery observed in the infected tissues and low blood flow in bones, the acute form of osteomyelitis that primarily affects children, is treated with parenteral antibiotics for two to four weeks, while the chronic form observed in adults, is treated for six weeks or more (LAZZARINI, MADLER and CALHOUN, 2004). Most cases of acute hematogenous osteomyelitis occur by *Staphylococcus aureus* (COLE, DALZIEL and

LEITL, 1982). In cases of chronic osteomyelitis there have been isolated besides *S. aureus*, *Staphylococcus epidermidis*, *Pseudomonas aeruginosa*, *Serratia marcescens*, *Escherichia coli*, *Enterobacteriaceae* and *Streptococcus*. (MACKOWLACK, JONES and SMITH, 1978). Without proper treatment, osteomyelitis can lead to chronic pain, septic arthritis, crippling joint destruction, abnormal bone remodeling, vascular compromise among other different conditions (BERENDT and BYREN, 2004; YIN, CALHOUN, THOMAS et al., 2008).

2 Material and methods

This work consisted of a literature review where it was conducted the search for scientific articles in the Pubmed, Scielo and ISI Web of Knowledge databases. In search strategies, It has been used the following keywords: Osteomyelitis morphological aspects. The period of research articles were from 1980-2011.

3 Results

For the maintenance of homeostasis of bone tissue it is necessary to balance the activity of osteoblasts and osteoclasts. This balance can be changed due to infections caused by pathogens such as *S. aureus* that are capable of inducing an inflammatory response by releasing cytokines that lead to bone and joint destruction by inducing apoptosis in particular of the tumoral necrosis factor alpha (TNF α), interleukin 1 (IL-1) and interleukin 6 (IL-6) (WRIGHT and NAIR, 2010).

In the infected bone, foci of acute osteomyelitis have a local purulent exudate, lesions in the bone matrix, compression and destruction of the vasculature as the infection spreads to the surrounding tissues, exacerbating osteonecrosis (LAZZARINI, MADER and CALHOUN, 2004; LEW and WALDVOGEL, 2004).

Factors that may contribute to osteonecrosis are trauma, surgeries as they can produce devitalized bone fragments, ischemia creating a suitable environment for bacterial culture with formation of abscess and sequestrum within a week (EMSLIE and NADE, 1983).

Morphological changes related to the presence of osteomyelitis are observed in radiographic studies where osteolysis is present, periosteal reaction and sequestrum which is the presence of segments of necrotic bone separated from living tissue and forming isolated foci of infection and that due to lack of vascularization make is inaccessible to antimicrobial agents or autoimmune response that may lead to chronic persistent infection (CAREK, DICKERSON and SACK, 2001; LAZZARINI, MADER and CALHOUN, 2004).

In the course of infection, bacteria leads to osteolysis which is the local bone destruction contributing to the spread and persistence of infection. An acute inflammation will cause an intense inflammatory response, thrombosis of endosteal and periosteal vessels, bone infarcts with subsequent abscess formation and sequestrum. A slow Inflammation, sluggish will produce a response of mild to moderate and little or no ischemic necrosis, a balance is struck between bone resorption and new bone formation, and sequestrum is less likely to develop (CIAMPOLINI and HARDING, 2000).

The presence of a bone abscess during the subacute or chronic stage of hematogenous osteomyelitis is known as Brodie abscess (BOUTIN, BROSSMANN, SARTORIS et al., 1998).

4 Conclusion

Osteomyelitis is an important bone infection that may result in serious consequences if not treated properly. Despite its various etiologies some morphological characteristics are the same and are observed through radiographic images where one could notice osteolysis, periosteal reaction, injury to the bone matrix, production of devitalized bone fragments and bone necrosis, abscess formation and bone sequestrum.

References

BERENDT, T. and BYREN, I. Bone and joint infection. *Clinica Médica*, 2004, vol. 4, p. 510-518. PMID:15656476.

BOUTIN, RD., BROSSMANN, J., SARTORIS, DJ., REILLY, D. and RESNICK, D. Update on imaging of orthopedic infections. *Orthopedic Clinics of North America*, 1998, vol. 29, p. 41-66. PMID:9405777.

CAREK, PJ., DICKERSON, LM. and SACK, JL. Diagnosis and Management of Osteomyelitis. *American Family Physician*, 2001, vol. 63, p. 2413-2420.

CIAMPOLINI, J. and HARDING, K.G. Pathophysiology of chronic bacterial osteomyelitis. Why do antibiotics fail so often? *Postgraduate Medical Journal*, 2000, vol. 76, p. 479-483. PMID:10908375. PMCID:1741709. <http://dx.doi.org/10.1136/pmj.76.898.479>

CIERNY, G., MADER, JT. and PENNINGCK, JJ. A clinical staging system for adult osteomyelitis. *Clinical Orthopaedics and Related Research*, 2003, vol. 414, p. 7-24. PMID:12966271. <http://dx.doi.org/10.1097/01.blo.0000088564.81746.62>

COLE, WG., DALZIEL, RE. and LEITL, S. Treatment of acute osteomyelitis in childhood. *Journal of Bone & Joint Surgery*, 1982, vol. 64, p. 218-23.

COSTERTON, JW. Biofilm theory can guide the treatment of device-related orthopaedic infections. *Clinical Orthopaedics and Related Research*, 2005, vol. 437, p. 7-11. PMID:16056019.

CRÉ MIEUX, AC. and CARBON, C. Experimental models of bone and prosthetic joint infections. *Clinical Infectious Diseases*, 1997, vol. 25, p. 1295-1302. PMID:9431367. <http://dx.doi.org/10.1086/516135>

EMSLIE, KR. and NADE, S. Acute haematogenous staphylococcal osteomyelitis: a description of the natural history in an avian model. *American Journal of Physiology*, 1983, vol. 110, p. 333-45.

LAZZARINI, L., MADER, JT. and CALHOUN, JH. Osteomyelitis in Long Bones. *Journal of Bone & Joint Surgery*, 2004, vol. 86-A, p. 2305-2318. PMID:15466746.

LEW, DP. and WALDVOGEL, FA. Osteomyelitis. *Lancet*, 2004, vol. 364, p. 369-379.

MACKOWLACK, PA., JONES, SR. and SMITH, JW. Diagnostic value of sinus-tract cultures in chronic osteomyelitis. *JAMA*, 1978, vol. 239, p. 2772-5. <http://dx.doi.org/10.1001/jama.1978.03280530036018>

MADLER, JT., CRIPPS, MW. and CALHOUN, JH. Adult posttraumatic osteomyelitis of the tibia. *Clinical Orthopaedics and Related Research*, 1999, vol. 360, p. 14-21. PMID:10101306. <http://dx.doi.org/10.1097/00003086-199903000-00004>

SMELTZER, MS. and GILLASPY, AF. Molecular Pathogenesis of Staphylococcal Osteomyelitis. *Poultry Science*, 2000, vol. 79, p. 1042-1049. PMID:10901208.

WRIGHT, JA. and NAIR, SP. Interaction of staphylococci with bone. *International Journal of Medical Microbiology*, 2010, vol. 300, n. 2-3, p. 193-204. PMID:19889575. PMCID:2814006. <http://dx.doi.org/10.1016/j.ijmm.2009.10.003>

YIN, LY., CALHOUN, JH., THOMAS, JK., SHAPIRO, S. and HOFFMANN, AS. Efficacies of Ceftriaxone Medocaril and Comparators in a Rabbit Model of Osteomyelitis Due to Methicillin-Resistant *Staphylococcus aureus*. *Antimicrobial Agents and Chemotherapy*, 2008, p. 1618-1622.

Received November 2, 2011

Accepted March 21, 2012