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Implications of Underlying Pathophysiology of Osteomyelitis in Diabetics for Nursing Care

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Introduction

Osteomyelitis is an infection of the bone caused by either bacteria, fungi, parasites, viruses or mycobacteria (McCance, et. al., 2014). The infection can enter bone either through an outside source or through the blood from a blood borne infection (McCance, et. al., 2014). The most common bacteria involved with osteomyelitis is Staphylococcus aureus (Hatzenbuehler & Pulling, 2011, p. 1027). Recently, at Dublin Methodist Hospital there has been an increase in the number of osteomyelitis cases in chronic uncontrolled diabetic patients requiring below the knee amputations. Two of the cases specifically were linked to chronic diabetic foot ulcers. Each of these cases were only planned to be partial foot amputations but resulted in below the knee amputations causing much stress for the patients and families. Diabetics are highly susceptible to diabetic foot ulcers and subsequent infections for two reasons. The first reason is that they have peripheral neuropathy and thus may not be able to feel a trauma to their foot or limb; and, secondly, because they have peripheral vascular disease limiting blood flow and thus healing to a wound site (Hatzenbuehler & Pulling, 2011, p. 1028). The pathophysiology of osteomyelitis is linked to bone degeneration and often the infection itself can be prevented with adequate and proactive nursing care. New ways to diagnosis and treat osteomyelitis are being explored through new research as will be examined in this research poster. Implications for better patient care and prevention will be obtained through gathering of the research

Pathophysiological Processes

Signs and Symptoms

- Because osteomyelitis is an infection of the bone, a precursor infection must occur in which the pathogenic bacteria transfer from a soft tissue injury to the bone in closest proximity.
- The preceding symptoms of infection would be the same as any infection including warm, redness and tenderness at the site. At times, fever accompanies infection.
- Hatzenbuehler and colleagues from the American Academy of Family Physicians state that symptoms of osteomyelitis itself may not present for approximately two weeks after the invading pathogens get to the bone (Hatzenbuehler & Pulling, 2011, p. 1027).
- It is stated that necrotic bone from osteomyelitis may not occur for six weeks after onset of infection (Hatzenbuehler & Pulling, 2011, p. 1027).
- "Clinical symptoms include chronic pain, persistent sinus tract or wound drainage, poor wound healing, and sometimes fever" (Hatzenbuehler & Pulling, 2011, p. 1028).
- Osteomyelitis is also often associated with diabetes and peripheral vascular disease. These two conditions make a person more susceptible to wounds of the extremities because of neuropathy, unawareness of a wound and decreased blood flow resulting in poor wound healing and ultimately the opportunity for further infection.

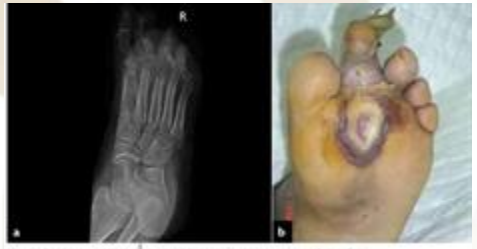
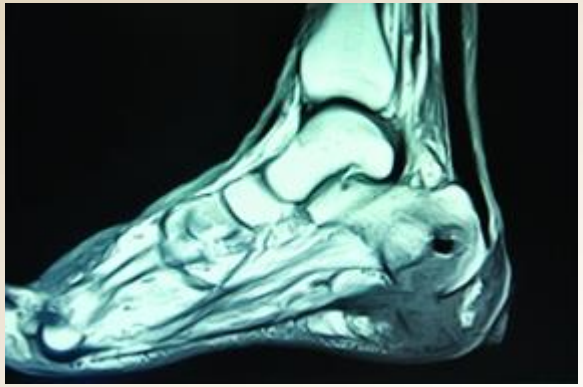


Fig 1. Oblique radiograph showing chronic osteomyelitis of the metatarsalphalangeal joints (a) and plantar metatarsal ulcer with involvement of the digit (dactylitis; b)

Journal of Wound Care, Jun2013, Vol. 22 Issue 6, p318-323, 5p
Color Photograph; found on p320

Underlying Pathophysiology

The pathophysiology of osteomyelitis starts with some sort of insult to the bone whether that be hematologic in origin or from a proximal wound of a diabetic ulcer. The original insult will spreads to the deeper structures and finally to the bone (Malone, Gannass, et. al. 2013, p. 318). *Staphylococcus aureus* is the most common invading pathogen that causes osteomyelitis. As with any other infections, the pathological process with osteomyelitis includes "synthesis of proinflammatory cytokines, activation and mobilization of phagocytic cells, thrombosis, [and] necrosis" and in osteomyelitis specifically: "bone sequestration, and formation of new bone" occur (Grbic, et. al., 2014, p. 1). *S. aureus* is capable of interacting with osteoblasts, bone-forming agents in bones, to alter bone reabsorption (Cassat, et. al., 2013). One mechanism that occurs is that *S. aureus* causes osteoclastogenesis, or the production of osteoclasts which are bone-reabsorbing cells through the expression of proinflammatory cytokines and the osteoclast-activator molecule receptor system (RANK-L) (Cassat, et. al., 2013, p. 759). And secondly, the invasion of *S. aureus*-infected osteoclasts causes cell death (Cassat, et. al., 2013, p. 760). Cassat and colleagues who explored the pathophysiology of osteomyelitis in depth state that "pathogen-induced bone destruction…destroys the vascular architecture of the infected bone and limits antimicrobial penetration to the infectious focus (Cassat, et. al., 2013, p. 766). Furthermore, Cassat and colleagues summarize their findings by stating that "*S. aureus* induces profound changes in bone remodeling, in part through the production of osteolytic exoproteins that modulate osteoblast proliferation…suggesting that bacterial modulation of bone remodeling involves complex changes in bone physiology" (Cassat, et. al., 2013, p. 766). Finally, damage is done to the bone in osteomyelitis by the oxidative stress that is produced by highly reactive oxidants of the leukocytes that move to the site of infection. Oxidative stress has been found to increase osteoclast activity and suppress the maturation of osteoblasts further increasing harmful bone remodeling (Grbic, et. al., 2014, p. 1).



American Family Physician, 11/1/2011, Vol. 84 Issue 9, p1027-1033, 7p, 1 Color Photograph, 2 Black and White Photographs, 3 Charts
Color Photograph; found on p1030

Significance of Pathophysiology

The significance of the pathophysiology of osteomyelitis lies in the implications that the infectious process has for treatment, diagnosis and nursing and medical care. Treatment of osteomyelitis is complicated by the pathogen-induced bone destruction and remodeling and therapies should target limiting the bone destruction that does occur (Cassat, et. al., 2013, p. 766). Cassat and colleagues at the Vanderbilt University Health System developed a bone model to support the theory that therapy needs to be targeted at bone physiology rather that only at antibiotic treatment with immune therapies that limit pathologic bone remodeling during osteomyelitis (Cassat, et. al., 2013, p. 768). This produces significant implications for treatment preferences and options. The preferred method of treatment for osteomyelitis is long term intravenous antibiotics. The appropriate antibiotic to use is determined by results of wound or bone cultures and biopsies (Hatzenbuehler & Pulling, 2011, p. 1031). Intravenous therapy should continue for two to six weeks with a transition to oral antibiotics (Hatzenbuehler & Pulling, 2011, p. 1032). It has been found that intravenous vancomycin is the first drug of choice for treatment of osteomyelitis (Hatzenbuehler & Pulling, 2011, p. 1032). Furthermore, since the pathophysiology of osteomyelitis does involve altered bone remodeling the gold standard in diagnosis has been bone biopsies (Malone, Bowling, et. al, 2013, p. 547). Bone biopsies, though, are expensive and painful for patients so other alternatives for diagnosis need to be found based on the pathophysiology discovered (Malone, Bowling, et. al, 2013, p. 547). Malone and colleagues found that "deep wound cultures correlate well with osseous cultures and provide a sensitive method in assessing and targeting likely pathogens that cause osseous infections. This will help aid the clinician in guiding antibiotic therapy" (Malone, Bowling, et. al, 2013, p. 546). The wound cultures that they used were specifically from deep wounds or taken from in wound sinus tracts (Malone, Bowling, et. al, 2013, p. 549). Another diagnostic tool that relates to the pathophysiology of osteomyelitis and inflammation is the increase in inflammatory markers that can be found on diagnostic lab work. A study that examined the levels of inflammatory markers C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), white blood cells (WBC), and procalcitonin (PCT) in osteomyelitis compared to soft tissue injuries found that all inflammatory markers were significantly higher in osteomyelitis (Michail, et. al., 2013, p. 96). Furthermore, the study found that ESR was actually the best marker to monitor the response to therapy (Michail, et. al., 2013, p. 96). Surgical intervention may be needed in some cases due to failure to respond to antibiotics, infected surgical hardware and chronic osteomyelitis with necrotic bone (Hatzenbuehler & Pulling, 2011, p. 1032).

Implications for Nursing Care

- Diabetes and osteomyelitis clearly coincide so special attention needs to be pain to diabetics with chronic ulcers and peripheral vascular disease
- Not only do diabetic patients have peripheral neuropathy leading to an increased risk of not recognizing a wound advancing into an infection, but they also have "microvascular abnormalities and phagocytosis defects" which "are likely to facilitate the spread of bacterial infection throughout the medulla and cortical bones" (Senneville, et. al., 2012, p. 59).
- Poor glycemic control leads to the complications of diabetes that are linked to osteomyelitis so better glycemic control can limit these complications and risk factors. It can be implied that nurses can take an active role in diabetic education that will truly make a difference in patient outcomes in preventing osteomyelitis and limiting complications from osteomyelitis.
- Furthermore, nurses can educate about other aspects of diabetes management such as good foot care, need for routine follow-up and getting healthcare in a timely manner (Malone, Gannass, et. al. 2013, p. 323).

Conclusions

Osteomyelitis can be difficult to diagnose and treat so clinicians must be aware of the special care that these patients require (Lipsky, et. al., 2012, p. 1679). Treatment and diagnosis need to be based on current knowledge of the pathophysiology of osteomyelitis and targeted specifically towards that. Special nursing care and education can be given to diabetic patients at risk for developing osteomyelitis to maintain adequate glycemic control. These patients also require specific follow up after suffering from osteomyelitis to be sure that it does not reoccur including good wound care, a completed antibiotic therapy course and follow ups with specialist. A multidisciplinary foot team can help improve patient and hospital outcomes (Lipsky, et. al., 2012, p. 1679).

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