Implications of Underlying Pathophysiology of Osteomyelitis in Diabetics for Nursing Care

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Pathophysiological Processes

**Signs and Symptoms**

**Underlying Pathophysiology**

Because osteomyelitis is an infection of the bone, a precursor infection must occur in which the pathogens get to the bone (Hatzenbuehler & Pulling, 2011, p. 1027).

The providing symptoms of infection would be the same as any infection including redness, warmth, and tenderness at the site. At times, fever accompanies infection.

Hatzenbuehler and colleagues from the American Academy of Family Physicians report that symptoms of osteomyelitis itself may not present until 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 sinus drainage, poor wound healing, and sometimes fever (Hatzenbuehler & Pulling, 2011, p. 1027).

Vascular complications are also associated with diabetes and peripheral vascular disease (Hatzenbuehler & Pulling, 2011, p. 1028).

However, some conditions make more vulnerable to wounds of the foot. Diabetes and osteomyelitis clearly coincide so special attention needs to be paid to diabetes with chronic ulcers and peripheral vascular disease (McCance & Michael/Nurses, 2014, p. 10).

Not only do diabetic patients have peripheral vascular disease, and thus an increased risk of not recognizing a wound advancing into an infection, but they also have diabetes-induced modifications of bone remodeling and increased risk of osteomyelitis (Cassat, et al., 2013, p. 766). Poor glycemic control leads to the complications that are linked to osteomyelitis so better glycemic control can limit these complications. It must 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 that are important for patients who need routine follow-up and getting to hospital due to poor glycemic control (Malaro, Gaensle, et al., 2013, p. 53).

**Significance of Pathophysiology**

The significance of the pathophysiology of osteomyelitis lies in the implications that the infectious process has for treatment, diagnosis and nursing 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 than just antibiotic treatment with immune therapies that limit pathogenic bone remodeling during osteomyelitis (Cassat, et al., 2013, p. 766). 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 to 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 is characterized by bone destruction, destroys the vascular architecture of bone, limits intramedullary perfusion to the infectious focus (Cassat, et al., 2013, p. 766). Furthermore, Cassat and colleagues have explored the pathophysiology of osteomyelitis in this regard by stating that “S. aureus induces profound changes in bone remodeling, in part through the induction of pro-inflammatory and pro-apoptotic esrroid to explain osteolysis and bone destruction.” Suppressive modulation of bone remodeling involves complex changes in bone remodeling (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 phagocytic cells that the invading pathogens 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), plasma procalcitonin (PCT) in osteomyelitis compared to soft tissue infections 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 interventions 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 of Pathophysiology of Osteomyelitis in Diabetics for Nursing Care

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**Implications for Nursing Care**

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