**Discuss the involvement of T- and B-lymphocytes in the pathogenesis and progression of osteomyelitis and osteoarthritis(OA)**

**What is Osteomyelitis?**

**Osteomyelitis is inflammation of the bone that is caused by an infecting organism. Although bone is usually not susceptible to bacterial colonization, events such as trauma, surgery, the presence of foreign bodies, or the placement of prostheses may disrupt bony integrity and lead to the onset of bone infection (“Osteomyelitis Symptoms and causes,” 2018). Osteomyelitis tends to occlude local blood vessels, which causes bone necrosis and local spread of infection. Infection may expand through the bone cortex and spread under the periosteum, with formation of subcutaneous abscesses that may drain spontaneously through the skin.**

**Osteomyelitis is caused by**

* **Contiguous spread from infected tissue or an infected prosthetic joint**
* **Bloodborne organisms (hematogenous osteomyelitis)**
* **Open wounds (from contaminated open fractures or bone surgery)**

**Trauma, ischemia, and foreign bodies predispose to osteomyelitis.**

**Osteomyelitis may form under deep pressure ulcers.**

**Contiguous spread from adjacent infected tissue or open wounds causes about 80% of osteomyelitis; it is often polymicrobial.**

**What is osteoarthritis?**

**Osteoarthritis is a chronic disease and it is the most common type of arthritis. It results from damage to articular cartilage induced by a variety of factors which may be genetic, metabolic, biochemical, and biomechanical factors, followed by activation of inflammatory response involving the interaction of cartilage, subchondral bone, and synovium . Many factors, some of which are modifiable, contribute to an increased risk of osteoarthritis and they include obesity, genetics, aging and trauma to the joint. In most patients without a strong genetic predisposition, osteoarthritis is thought to start as a result of damage to the joint tissue by physical forces as a single event of trauma or by repeated micro trauma, due to altered mechanical loading of the joint. These factors cause the cartilage that cushions the ends of the bones to wear over time.**

**Functions of T and B – lymphocytes**

**Genetic, metabolic or mechanical factors cause an initial injury to the cartilage resulting in release of several cartilage specific autoantigens, which trigger the activation of immune response. Immune cells including T cells, B cells and macrophages infiltrate the joint tissues, cytokines and chemokines are released from different kind of cells present in the joint, complement system is activated, cartilage degrading factors such as matrix metalloproteins (MMPs) and prostaglanding E2(PGE2) are released, resulting in further damage to the articular cartilage.**

**The T and B lymphocytes (T and B Cells) are involved in the acquired or antigen-specific immune response given that;**

1. **they are the only cells in the organism able to recognize and respond specifically to each antigenic epitope.**
2. **The B Cells have the ability to transform into plasmocytes and are responsible for producing antibodies. Thus, humoral immunity depends on the B Cells while cell immunity depends on the T Cells (Cano, 2013).**
3. **Infiltrates of immune cells including T-cells, B-cells and macrophages have been detected in synovial tissue of OA patients. T cells derived from peripheral blood and synovial fluid of OA patients showed a strong response to autologous chondrocyte and fibroblast membrane preparations.**
4. **B cells both respond to and produce the chemokines and cytokines that promote leukocyte infiltration into the joints, formation of ectopic lymphoid structures, angiogenesis, and synovial hyperplasia.**

**References** (APA)

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