Lecture 9

The skeletal system III - Bone Disorders

Plan of the Lecture

1. Fractures and Their Repair
    a. Types of Fractures
    b. The Healing of Fractures
        i. Formation of hematoma and granulation tissue
        ii. Formation of a soft callus
        iii. Conversion to hard callus
        iv. Remodeling
    c. The Treatment of Fractures
2. Other Bone Disorders
    a. Osteitis deformans
    b. Osteomyelitis
    c. Osteogenesis imperfecta
    d. Osteosarcoma
    e. Achondroplastic dwarfism
    f. Ectopic ossification
    g. Osteomalacia
    h. Rickets
    i. Mastoiditis

LEARNING OUTCOMES

1. name and describe several bone diseases;
2. name and describe the types of fractures;
3. explain how a fracture is repaired; and
4. discuss some clinical treatments for fractures and other skeletal disorders.

There are multiple ways of classifying bone fractures. A stress fracture is a break caused by abnormal trauma to a bone, such as fractures incurred in falls, athletics, and military combat. A pathological fracture is a break in a bone weakened by some other disease, such as bone cancer or osteoporosis, usually caused by a stress that would not normally fracture a bone. Fractures are also classified according to the direction of the fracture line, whether the skin is broken, and whether a bone is merely cracked or broken into separate pieces. An uncomplicated fracture heals in about 8 to 12 weeks, but complex fractures take longer and all fractures heal more slowly in older people. The healing process occurs in the following stages: Formation of hematoma and granulation tissue. A bone fracture severs blood vessels of the bone and periosteum, causing bleeding and the formation of a blood clot

(fracture hematoma). Blood capillaries soon grow into the clot, while fibroblasts, macrophages, osteoclasts, and osteogenic cells invade the tissue from both the periosteal and medullary sides of the fracture. Osteogenic cells become very abundant within 48 hours of the injury. All of this capillary and cellular invasion converts the blood clot to a soft fibrous mass called granulation tissue. Formation of a soft callus. Fibroblasts deposit collagen in the granulation tissue, while some osteogenic cells become chondroblasts and produce patches of fibrocartilage called the soft callus.

Conversion to hard callus. Other osteogenic cells differentiate into osteoblasts, which produce a bony collar called the hard callus around the fracture. The hard callus is cemented to dead bone around the injury site and acts as a temporary splint to join the broken ends or bone fragments together. It takes about 4 to 6 weeks for a hard callus to form. During this period, it is important that a broken bone be immobilized by traction or a cast to prevent reinjury.

Remodeling. The hard callus persists for 3 to 4 months. Meanwhile, osteoclasts dissolve small fragments of broken bone, and osteoblasts deposit spongy bone to bridge the gap between the broken ends. This spongy bone gradually fills in to become compact bone, in a manner similar to intramembranous ossification. Usually the fracture leaves a slight thickening of the bone visible by X-ray; such thickenings may serve as forensic evidence of child abuse. In some cases, however, healing is so complete that no trace of the fracture can be found.

The Treatment of Fractures. Most fractures are set by closed reduction, a procedure in which the bone fragments are manipulated into their normal positions without surgery. Open reduction involves the surgical exposure of the bone and the use of plates, screws, or pins to realign the fragments.

The most common bone disease is osteoporosis (literally, “porous bones”), a severe loss of bone density. It affects especially spongy bone, since this is more metabolically active than dense bone and has more surface area exposed to bone-dissolving osteoclasts. As a result, the bones become brittle and highly subject to pathological fractures from stresses as slight as sitting down too quickly. Fractures occur especially in the hip, wrist, and vertebral column. As the vertebrae lose bone, they become compressed and the spine is often deformed into a condition called kyphosis (“widow’s hump”). Osteoporosis occurs in both sexes and at all ages from adolescence to old age. Postmenopausal white women, however, are at the greatest risk. In contrast to men and black women, they have less bone density; white women begin losing it earlier (as young as age 35); and they lose it more rapidly. By age 70, the average white woman loses 30% of her bone mass. Until menopause, estrogen maintains bone density by inhibiting osteoclasts. At menopause, the ovaries cease to secrete estrogen and osteoclast activity begins to outpace bone deposition by osteoblasts.

Black women also lose bone density after menopause, but having denser bones to begin with, they usually do not lose enough to cross the threshold into osteoporosis. Osteoporosis is now diagnosed with dual-energy X-ray absorptiometry (DEXA), which uses low-dose X-rays to measure bone density. DEXA allows for early diagnosis and more effective drug treatment. Treatments for osteoporosis are aimed at slowing the rate of bone resorption. Estrogen replacement therapy is out of favor because it increases the risk of breast cancer, stroke, and coronary artery disease.

Check yourself! The questions for self-control

1. Name and describe four types of bone fractures.
2. Why would osteomyelitis be more likely to occur in an open fracture than in a closed fracture?
3. What is a callus? How does it contribute to fracture repair?
4. Describe the difference between a stress fracture and a pathological fracture; stages in the healing of a fractured bone; and approaches to the clinical treatment of fractures
5. List causes of osteoporosis; describe its risk factors, pathological effects, diagnosis, treatment, and prevention

Recommended readings:

1. Kenneth S Saladin - Anatomy & Physiology. The Unity of Form and Function (2016, McGraw-Hill Education)
2. Barbara Gylys - Medical Terminology Systems (2012, F.A. Davis Company)