Callus formation during fracture healing

Bone fractures are a common injury and the healing process is complex. [1] Bone is one of the few tissues that is able to heal without forming a fibrous scar. There are two types of fracture healing – indirect (secondary) and direct healing (primary). [2] Fracture of 4th Metatarsal Bone Direct healing occurs when the bony fragments are attached along with compression. There's no calluses. The legged ends are joined together and cured by osteoclast and osteoblast activity. [1] Indirect healing is more common than direct healing and includes both endochondral and intramembranous bone healing. [2] Anatomical reduction and stable conditions are not necessary for indirect healing to occur. [2] Rather, there is a small amount of motion and weight that carry at the break, causing a soft calluses to form, leading to secondary leg formation. [1] It should nevertheless be noted that too much charge/movement can result in delayed healing or non-union,[2] occurring in 5-10% of all fractures. [3] Indirect healing usually occurs in: non-operative fracture treatment surgical treatments where some movement occurs at the fracture site, such as: Intramedullary nailing External fixation Internal fixation of comminuted fractures. [2] Stages of indirect healing acute inflammatory response The acute inflammatory response peaks within 24 hours and ends after 7 days and is essential for healing. [2] A hematoma forms immediately after trauma. This consists of cells from the peripheral and intramedullary blood and bone marrow cells. The inflammatory reaction causes the hematoma to solidify around the fracture ends and in the medulla, creating a model for callus formation. [2] Recruitment of Mesenchymal Stem Cells Bone is not able to regenerate unless specific mesenchymal stem cells are recruited, multiplied and differentiated in osteogenic cells. It is currently not understood exactly where these cells come from. [2] Generation of cartilage-like and Periosteal Bony Callus After the hematoma has formed, a fibrin-rich granulation tissue forms. Endochondral formation occurs between the fracture ends and beyond the periosteal sites in this tissue. These areas are less stable, so the cartilage tissue forms a soft callus, giving the fracture more stability. [2] In animal studies, soft calluses peak at 7 to 9 days when type II procol layers and proteoglycan core protein extracellular markers are at their highest level. [2] Simultaneously, an intramembranous ossification response occurs immediately subperiostally by the fracture ends. This creates a hard callus. The bridging of this central hard callus provides the fracture with a semi-rigid structure that allows weight bearing. [2] Revascularization and Neoangiogenesis Sufficient blood supply is necessary for bone repair. pathways, chondrocyte apoptosis and creaky degradation are essential for this process, as cells and extracellular matrices need to be removed to ensure that ships can go to the repair site. [2] Mineralization and Resorption of the Cartilage Calluses The primary soft cartilage calluses should be resorbed and replaced with a hard bony callus for bone regeneration to continue. [2] In some respects, this stage repeats the development of the embryological bone and involves cellular proliferation and differentiation, as well as an increase in cellular volume and matrix deposition. [2] Bot remodeling While the hard callus is stiff and provides stability, this does not mean that the fracture site has all the properties of normal bone. A second restorative phase is necessary. This phase results in the redesign of the hard callus into a slatted bone structure with a central medullary cavity. [2] Remodeling occurs when the hard callus is resorbed by osteoclasts and lamellar bone is deposited by osteoblasts. This starts at 3-4 weeks, but the whole process can take years. Remodeling can be faster in younger patients (and other animals). [2] Bother device is the result of the production of electric polarity. This happens when pressure is applied in a crystalline environment. [2] When axial charge of long bones occurs, an electropositive convex surface and an electrongegative concave surface are created this activates osteoclastic and osteoblastic activity. As a result, the external calluses are slowly replaced by a slat bone structure. In addition to this, the internal calluses remodel which re-creates a medullar cavity, similar to diaphyseal bone. [2] Bone remodeling will only be successful if there is sufficient blood supply and a gradual increase in mechanical stability. If not, complications such as non-union can occur. [2] Direct Fracture Healing Immediate Healing requires reduction of fracture ends, without any gaping, as well as stable fixation. So, it usually comes not naturally, but after open reduction and internal fixation surgery. [2] Direct bone healing can occur through direct remodeling of lamelbeen, the Haver's channels and blood vessels. The process usually takes from months to years. [2] Primary healing of fractures occurs by: contact healing or gap healing. Both processes consist of an attempt to recreate the bone structure of lamellar. Direct bone healing is possible only when the fracture ends are compressed together and rigid fixation is used to reduce the interfragmentary load. [2] [1] Contact Healing A fracture can be reconciled by contact healing when the gap between each bone end is less than 0.01 mm and the interfragmentary strain is less than 2%. [2] In such cases, cones cut shape at the ends of the osteons through the fracture site. The ends of the cutting cones consist of osteoclasts. These tips cross the fault line and generate longitudinal cavities. [2] The cavities are eventually filled by bone that produced by osteoblasts. This causes the benexian union to generate, while also restoring the Haversian systems, which are formed in an axial direction. The Haversian systems allow blood vessels osteoblasts to enter the area. Bridging osteons eventually mature to form lamellar bone, resulting in fracture healing without a periosteal callus. [2] Gap Healing Gap healing is unique in that benety union and Haversian remodeling doesn't happen at the same time. [2] In order for the gap healing to occur, the gap should be less than 800 μm to 1 mm. [2] During this process, the fracture site is largely filled by lamellarbot that runs perpendicular to the long axis and needs secondary osteonal reconstruction. The primary bone structure is eventually replaced by along-in-the-way revascularized osteons, which carry osteoprogenitor cells that differentiate themselves in osteoblasts. These osteoblasts then produce lamellar bone on every surface of the gorge. [2] The lamellarbot is placed perpendicular to the long axis, which means that it is not strong. This process takes between 3 and 8 weeks. This is followed by a secondary remodeling phase, which is similar to the cascade of cutting cones in contact healing. [2] References Fracture healing occurs naturally after traumatic legged disturbance. This process begins with bleeding and progresses through three phases: inflammatory remodeling This process can be supported by several treatment options with immobilization a mainstay; inappropriate treatment can lead to several complications. Depending on the fracture site, a normal healing in adults can take 3-12 weeks. For a normal healing of fractures, a number of requirements must be met: the viability of fragments (i.e. intact blood supply) mechanical rest: this can be achieved by non-movement and external immobilization; e.g. cast or internal fixation absence of infection The healing process is different depending on the configuration of the fracture fragments and can be divided into three main categories: spontaneous (indirect/secondary) healing contact (angiogenic/primary) healing gap healing This is the most common 'natural' healing process, where the fracture ends are placed close together (but not applied), with intermediate hematoma and variable displacement and/or angulation. Although the healing process of a fracture can be divided into several phases, it should rather be conceived as a biological continuum. The periosteum, endosteum and Haversian channels are the sources of pluripotent mesenchymal stem cells that initiate the formation of the healing tissues. The bridging narrower seen on X-rays mainly stems from the periosteum. The fracture hematoma initiates the healing reaction. Within 48 hours, chemotactic signaling mechanisms attract the inflammatory cells needed to promote the healing process. Within 7-14 days, granulation tissue is formed between the fragments, leading to vascularization of the hematoma. X-rays can be this stage increased lucency of the fracture occur due to bone resorption. Precursor cells in the granulation tissue multiply and begin to differentiate into fibroblasts and chondroblasts. They an extracellular organic matrix of fibrous tissue and cartilage, in which woven bone is deposited by osteoblasts. This phase usually lasts 4-16 weeks. The newly formed calluses are still damaged by shear forces, while axial traction and pressure promote matrix formation. The mesh of woven bone is then replaced by lamellarbot, which is organized parallel to the shaft of the bone. Eventually, the remodeling of the bone takes place, restoring the normal cortical structure, depending on the distribution of the load. This is an ongoing process that can take several years. In children, remodeling happens faster than in adults and can compensate for malunion to some extent. Contact healing occurs between directly apposed fragments when there is less than 0.1 mm distance and neutralization of interfragmentary strain. This is the purpose of rigid internal fixation. The process is initiated by osteoclasts that form cutting cones that cross the fault line at 50-100 μm/day. Capillaries can then occupy the newly formed cavities and are accompanied by endothelial cells and osteoblast precursor cells that form lamellar bone of osteons primarily oriented in the axial direction (Haversian remodeling). In stable osteosynthesis, there should be no periosteal calluses. Otherwise, this should be considered as a sign of instability (irritation callus). If the internal fixation leaves an opening of even less than 1 mm between the fragments, haversian remodeling is preceded by the determination of woven bone scaffolding, after which lamellarbot is deposited perpendicular to the long axis. Normal fracture healing can be disrupted in many ways: delayed healing of the commitment takes about twice as long as expected for a specific location non-union (pseudoarthrosis) fracture healing does not occur within 6-9 months common sites: scaphoid bone, femoral neck, tibial shaft malunion healing in a non-anatomical position can be partially compensated by remodeling the bone (excluding rotation malunion) refracturing as a line of thumb, fixation material should be removed within about 18 months after consolidation of the fracture leaving it in place can lead to cortical atrophy and a higher risk of breaking permanent damage to the healing tissue (blastema), usually in the chondrale phase. Risk factors are: local: insufficient immobilization, distraction, soft parts interposition, bone loss, reduced blood flow, soft tissue damage, incorrect fixation method, infection general/systemic: old age, poor nutritional status, drugs, and medications, metabolic diseases hypertrophic pseudoarthrosis rich in calluses (elephant foot) bad in calluses (horse hoof) oligotrophic pseudoarthrosisno callus on radiography, rounded fractures atroffic pseudoarthrosis resorption or reduced blood flow hypotrophic (non-vital) pseudoarthrosisnecrotic fragments (increasing fragment density without defecting from the formation of kalnos) kalnos) adequate immobilization electrical stimulation bone transplantation review of internal fixation fixation