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A Comprehensive Review of Avascular Necrosis (AVN) Treatment Modalities in Orthopedics

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Abstract

Avascular necrosis (AVN), also known as osteonecrosis, aseptic necrosis, or ischemic bone necrosis, describes a pathological condition characterized by bone tissue death due to disrupted blood supply, leading to bone death and potential joint collapse.[1, 3] This review aims to provide an overview of the current treatment strategies for AVN, encompassing both conservative and surgical interventions. Emphasis is placed on recent advancements, including biological therapies and novel surgical techniques, to guide orthopedic practitioners in evidence-based decision-making.

Keywords

AVN, avascular necrosis, Orthopaedics, orthopaedic surgery, osteonecrosis, aseptic necrosis, ischemic bone necrosis

Introduction

Avascular necrosis (AVN) is characterized by the death of bone tissue due to compromised blood flow. AVN can affect any bone, but it most commonly develops in the ends of long bones, notably the femoral head. The engagement of bones connected to the joint, along with years of wear and tear, can lead to their damage and the development of arthritis. Less frequently, osteonecrosis affects bones in the elbows, ankles, feet, wrists, and hands.[2-4] Osteonecrosis occurs when blood supply to a bone is disrupted, leading to tissue damage. In minor cases, the body may repair the affected area, but often the bone deteriorates, fractures, and may eventually collapse. Traumatic osteonecrosis results from vessel damage due to injury, while nontraumatic cases may involve blood clots or increased pressure within the bone.[2] Avascular necrosis has been linked to various medical conditions, including pancreatitis, Gaucher's disease, HIV/AIDS, systemic lupus erythematosus, sickle cell anemia, decompression sickness (commonly known as divers disease or the bends), and certain cancers like leukemia. The primary nontraumatic cause of osteonecrosis is steroid use.[5] Untreated avascular necrosis progresses, potentially leading to bone collapse. It can also cause the bone to lose its smooth shape, increasing the risk of severe arthritis, necessitating total joint arthroplasty.[1] There are no specific laboratory tests that indicate or confirm the diagnosis of avascular necrosis. For diagnosing AVN, plain radiography serves as the preferred initial imaging modality, though it often appears normal in the early stages. MRI remains the most sensitive and specific diagnostic tool. Early detection and timely intervention can postpone the need for joint replacement; however, most patients are diagnosed at an advanced stage. Without treatment, the disease typically progresses, resulting in joint destruction within five years. In the early stages of AVN, conservative management includes pain relief with analgesics and reduced weight-bearing using crutches. Additional therapeutic strategies that have been explored include alendronate, iloprost, statins, extracorporeal shockwave therapy, and hyperbaric oxygen therapy[6]

Medical management of avascular necrosis is primarily determined by the disease's location, severity, and the patient’s age and overall health. Treatment success is closely linked to the disease stage, and no medical therapy has been proven to prevent or halt its progression.

Conservative management

Conservative approaches include pain management with analgesics and restricted weight-bearing using crutches. These measures may be beneficial as an initial strategy, particularly when the affected bone segment is less than 15% and located outside the weight-bearing zone. Immobilization can also be useful in specific cases, such as AVN of the distal femur or tibia. However, in advanced stages, the disease progression is independent of activity and will ultimately necessitate surgical intervention.[6]

Bisphosphonates, such as alendronate, may provide therapeutic benefits. A systematic review indicated that alendronate may offer short-term pain relief, improve joint function, slow bone collapse, and delay the need for total hip replacement in adults with AVN.[7]

Iloprost, a vasoactive prostaglandin analog approved for pulmonary hypertension treatment, has demonstrated clinical and radiographic improvements in early-stage AVN when administered intravenously. Its vasodilatory effects enhance microcirculation, reduce bone marrow edema, alleviate pain, inhibit platelet aggregation, and reduce oxidative stress.[8]

Statins may provide a protective effect against the development of osteonecrosis in patients requiring steroid therapy. Steroid treatment can cause hyperlipidemia, which often leads to osteoporosis but rarely causes osteonecrosis. However, about one-third of osteonecrosis cases are linked to steroids. It is impossible to predict which patients will develop osteonecrosis, so it seems reasonable to use statins as a prophylaxis of potential adverse effects of steroid drugs in patients requiring continuous steroid therapy.[9]

Extracorporeal shockwave therapy (ESWT) has shown potential benefits in early-stage AVN of the femoral head, helping to alleviate pain, enhance hip function, and promote AVN regression.[10, 11]

Hyperbaric oxygen therapy (HBOT) has demonstrated positive effects in early AVN of the femoral head, reducing self-reported pain, localized edema, and lesion size on imaging studies. Its efficacy may be attributed to the modulation of inflammatory markers and reactive oxygen species.[12]

Surgical Management

Various surgical procedures have been employed to treat AVN, with differing success rates. However, there is no consensus on a single surgical approach as the standard of care. In the early, precollapse stages of AVN, core decompression, with or without bone grafting, is regarded by many clinicians as the preferred treatment. In advanced stages, marked by femoral head collapse, deformity, and secondary osteoarthritis, total hip arthroplasty remains the most suitable intervention.[6]

Core Decompression combined with Bone Grafting

This procedure entails removing a core of bone from the necrotic area to reduce intraosseous pressure and promote revascularization and is most effective in early-stage AVN before structural collapse occurs. Core decompression, frequently performed alongside bone grafting, is a widely recognized joint-preserving surgical technique. Research indicates that drilling holes to remove necrotic bone and alleviate intraosseous pressure facilitates revascularization. The addition of structural bone grafts aids in preventing subchondral collapse and supports bone remodeling through multiple mechanisms.[13-16]

Bone grafting options include structural cortical or medullary grafts, as well as vascularized grafts, which can be performed using a muscle-pedicle bone graft or a free vascularized fibular graft.[17] Bone grafting is typically combined with: core decompression, which may help disrupt the ischemic cycle, sequestrum excision, which can facilitate revascularization of the femoral head, and a period of restricted weight-bearing. Free vascularized bone grafts offer several advantages over total hip arthroplasty, including the potential for a healed femoral head to allow greater physical activity, the absence of foreign body–related complications, and the possibility of lifelong femoral head preservation if performed in the early stages of AVN. Additionally, this procedure preserves the option for total hip arthroplasty in the future if needed. However, free vascularized grafts also have notable disadvantages, such as a prolonged recovery period, less complete pain relief, variable success rates, and limited effectiveness in advanced-stage disease.

Osteotomy

This procedure is typically reserved for specific cases where joint preservation is feasible. Osteotomies are performed with the goal of altering the spatial orientation of the necrotic region of the femoral head, redistributing weight-bearing forces to healthier bone. By repositioning the affected area, osteotomies aim to reduce mechanical stress on the necrotic segment, potentially slowing disease progression, alleviating pain, and preserving joint function for a longer period.[18]

Joint Replacement

Arthroplasty has proven to be an excellent treatment option for patients with avascular necrosis who experience debilitating pain and significant functional impairment. In cases where joint-preserving procedures such as core decompression, bone grafting, or osteotomy fail to halt disease progression, or when AVN is diagnosed in its advanced stages with femoral head collapse and secondary osteoarthritis, with joint involvement, including subchondral fractures, femoral head flattening, joint space narrowing, and acetabular alterations, total hip arthroplasty (THA) becomes the most appropriate intervention.[19] THA provides effective pain relief, restores joint function, and significantly improves the patient's quality of life. Modern advancements in implant technology, surgical techniques, and perioperative management have enhanced the durability and outcomes of hip replacements in AVN patients, even in younger individuals. However, challenges remain, including higher revision rates in younger and more active patients due to implant wear and loosening over time. Other arthroplasty options, such as hemiarthroplasty or resurfacing arthroplasty, may be considered in select cases, particularly in younger patients where femoral head preservation is still a priority. However, their long-term efficacy compared to THA remains a subject of ongoing research.

Tantalum Implants

Tantalum implants have emerged as a potential alternative for patients with early-stage ONFH. These porous implants provide structural support and encourage bone ingrowth, delaying disease progression. However, their long-term efficacy remains under investigation.[19]

Arthrodesis and Resection Arthroplasty

Although rarely performed today, hip arthrodesis (joint fusion) and resection arthroplasty (removal of the femoral head) were historically used to treat ONFH in younger patients to preserve some degree of function. However, these procedures have been largely replaced by THA due to its superior functional outcomes.[19]

Mesenchymal stromal cell therapy

Mesenchymal stromal cell (MSC) therapy has been increasingly explored as a potential treatment for osteonecrosis due to its regenerative properties, particularly in bone repair and vascularization.[20]

MSCs are multipotent progenitor cells capable of differentiating into osteoblasts, chondrocytes, and adipocytes, making them an attractive option for bone regeneration. Their therapeutic potential in osteonecrosis is attributed not only to their direct osteogenic capacity but also to their ability to secrete paracrine factors that stimulate angiogenesis and modulate the inflammatory environment. In hypoxic conditions typical of osteonecrotic bone, MSCs have demonstrated increased proliferation and expression of osteogenic markers such as alkaline phosphatase, type I collagen, and osteocalcin.[21, 22]

Several clinical studies have assessed the safety and efficacy of MSC therapy in osteonecrosis. A pilot study by Müller et al. demonstrated the feasibility and safety of autologous MSC application in combination with core decompression, reporting improved clinical outcomes and mineralized bone formation in the necrotic area.[23] A randomized prospective trial by Zhao et al. found that only 2 of 53 hips progressed to later stages in patients treated with autologous bone marrow-derived MSCs (BM-MSCs) combined with core decompression, compared to 10 of 44 hips in the group treated with core decompression alone.[24] In another study, intra-arterial infusion of human umbilical cord-derived MSCs resulted in reduced necrotic lesion size and improved oxygen delivery index, suggesting a role for MSCs in revascularization.[25]

Despite promising preliminary results, MSC therapy for osteonecrosis remains in the experimental stage, with variability in treatment protocols, cell sources, and dosages contributing to inconsistent clinical outcomes. Further high-quality, randomized controlled

trials are needed to establish standardized protocols, optimize cell delivery methods, and confirm the long-term efficacy of MSCs in osteonecrosis treatment. Additionally, comparisons between autologous and allogeneic MSCs, as well as genetically modified MSCs with enhanced osteogenic and angiogenic properties, may help refine future therapeutic strategies.

Conclusion

The management of avascular necrosis requires a multidisciplinary approach tailored to the disease stage and patient-specific factors. While conservative treatments may provide symptomatic relief in early stages, surgical interventions, particularly when combined with biological therapies, offer promising outcomes in preserving joint function. Ongoing research into novel therapies holds the potential to further improve the prognosis for AVN patients.

Disclosure

Author's contribution

Conceptualization – Jan Mateńko; methodology – Julia Kiełbratowska, Maria Potrykus; software - Maria Potrykus, Przemysław Klasicki; check - Wiktor Możarowski, Agata Krupa and Maciej Mozer; formal analysis - Anna Krawczyk and Wiktoria Pietruszka; investigation - Jan Mateńko, resources - Michał Pałuchowski and Maciej Mozer; data curation – Jan Mateńko; writing - rough preparation - Anna Krawczyk and Michał Pałuchowski; writing - review and editing - Jan Mateńko and Agata Krupa; visualization -Wiktor Możarowski, Maciej Mozer, Julia Kiełbratowska; supervision - Jan Mateńko, project administration – Jan Mateńko.

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All authors declare that they have no conflicts of interest.

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