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The Silent Connection: Unveiling the Cardiovascular Impact of Paget's Disease on Heart Failure and Cardiac Care

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To the Editor,

Paget's disease of bone, or osteitis deformans, represents a chronic disorder that profoundly alters normal bone remodeling processes, leading to the development of structurally weak and often enlarged bones. The disease, second in prevalence among metabolic bone disorders after osteoporosis, affects roughly 1% to 2% of adults over 55 years, with a higher incidence noted in Western populations. While the disease's skeletal manifestations, such as deformities, fractures, arthritis, and pain, often capture primary clinical attention, its cardiovascular complications—most notably heart failure—deserve focused scrutiny in our practice. The relationship between abnormal bone turnover and increased cardiovascular strain has significant implications, particularly in advanced cases, highlighting the need for greater awareness. The pathological changes of Paget's disease primarily stem from a mismatch in bone resorption and formation, driven by abnormal osteoclast and osteoblast activity. This abnormal bone turnover not only weakens the bone mechanically but also substantially increases its vascularization. As metabolic demands surge to supply blood to hyperactive bone regions, a notable cardiovascular burden ensues. The enhanced vascularity can lead to systemic effects, straining cardiac function and, over time, potentially leading to heart failure. This cardiovascular involvement is often underappreciated, with signs that may be mistaken for other conditions or simply attributed to aging, delaying optimal treatment and management. A prominent cardiovascular manifestation in Paget's disease is high-output heart failure. Unlike traditional low-output heart failure, which stems from reduced cardiac output, high-output heart failure occurs when the heart's enhanced performance cannot meet the elevated metabolic demands of hyper vascularized bone regions. Blood flow through these areas significantly increases, prompting the heart to boost its output and dilate blood vessels to maintain adequate circulation. When this state persists, however, the cardiac reserve may be overwhelmed. Symptoms can include fatigue, edema, dyspnea, and other signs commonly associated with heart failure, though the hyperdynamic state and underlying bone-related causes are often overlooked. The sustained increase in cardiac workload due to high metabolic demands of affected bones can lead to structural changes in the heart. Over time, left ventricular hypertrophy (LVH) may develop as the heart muscle thickens to cope with the increased strain. LVH is often a compensatory response but, if prolonged, can exacerbate heart failure by reducing the efficiency of cardiac pumping. The strain of pumping against elevated vascular resistance also contributes to heart failure symptoms, such as shortness of breath, fatigue, and fluid retention. Perfusionists, particularly during surgery or when managing hemodynamic fluctuations, should be aware of these potential challenges in patients with extensive Paget's disease involvement[1].

Aortic Valve Calcification risk associated with Paget's disease is aortic valve calcification. The elevated turnover of calcium and phosphate in affected bones predisposes patients to ectopic calcification, including within the cardiovascular system. Calcification of the aortic valve, resulting in aortic stenosis, stiffens and narrows the valve, increasing afterload and adding further strain on the heart. This narrowing can make it more difficult for the heart to pump blood, aggravating heart failure symptoms and potentially requiring surgical intervention, such as valve replacement. The presence of aortic valve calcification is clinically significant and highlights the interconnected nature of bone and cardiovascular health in Paget's diseases[2].

Paget's disease is also at a heightened risk for developing arrhythmias, particularly atrial fibrillation. The combination of structural heart changes, altered hemodynamics, and increased metabolic demands creates a milieu that is more prone to electrical disturbances. Atrial fibrillation can compromise cardiac output, reduce stroke volume, and worsen symptoms of heart failure. As perfusionists, understanding and managing arrhythmias during procedures is vital, as even transient episodes can severely impact patient outcomes, particularly in high-risk groups such as those with Paget's disease. The diagnosis of heart failure can be complex due to overlapping symptoms with other conditions. Many patients may present with signs of congestive heart failure—peripheral edema, fatigue, and dyspnea—that can be mistakenly attributed to aging or other comorbidities. High-output heart failure, in particular, may be overlooked due to its atypical presentation. To differentiate and accurately diagnose heart failure in these patients, a high index of suspicion is essential, especially for those with extensive bone involvement. Imaging studies, such as X-rays and bone scans, can identify areas of high bone turnover, while echocardiography can assess for left ventricular hypertrophy and valvular disease. Biomarkers, including serum alkaline phosphatase (elevated in Paget's due to increased bone formation) and cardiac markers like NT-proBNP or BNP, may further guide diagnosis and severity assessment. Effective management of heart failure requires a comprehensive, multidisciplinary approach that addresses both skeletal and cardiovascular aspects. The primary treatment for Paget's disease involves bisphosphonates—medications like zoledronic acid and alendronate, which suppress abnormal bone turnover and reduce bone vascularity. This reduction in bone activity can alleviate the high-output state by decreasing metabolic demands and lessening the burden on cardiac function. Conventional heart failure therapies, including beta-blockers, ACE inhibitors, and diuretics, remain critical for symptom management and stabilization of cardiac output[3].

In advanced cases, surgical intervention may be necessary. For patients with aortic valve calcification, corrective orthopedic procedures may be indicated for severe skeletal deformities that further compromise cardiovascular health. Coordination between cardiologists, rheumatologists, and perfusionists is key to optimizing care for these complex patients. Paget's disease, while often perceived as a bone-centric disorder, poses significant cardiovascular regarding heart failure. Hypervascularity, increased metabolic demands, and structural heart changes are central to this elevated risk. Perfusionists and cardiovascular specialists must remain vigilant to the unique interplay between bone and heart health in Paget's patients, ensuring timely diagnosis and tailored management. Multidisciplinary care that addresses both skeletal and cardiovascular challenges can lead to improved patient outcomes and reduced cardiovascular morbidity in this complex population[4].

REFERENCES:

1. Ralston SH. Bisphosphonates in the management of Paget's disease. Bone. 2020 Sep 1;138:115465.

2. Ralston SH. Paget's disease of bone. New England Journal of Medicine. 2013 Feb 14;368(7):644-50.

3. Hadjidakis DJ, Androulakis II. Bone remodeling. Annals of the New York Academy of Sciences. 2006 Dec;1092(1):385-96.

4. Carletti A, Gavaia PJ, Cancela ML, Laizé V. Metabolic bone disorders and the promise of marine osteoactive compounds. *Cellular and Molecular Life Sciences*. 2024 Dec;81(1):11.