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Dystrophic Calcified Mass Found in the Semimembranosus Muscle in a Cadaver: A Case Report

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ABSTRACT

Most of the soft tissue calcified mass formations are sporadic incidences that occur in an idiopathic form. Ectopic calcification changes were seen in various soft tissues beyond the skeleton. They are often asymptomatic masses located in the superficial or deeper aspect of the body, but may not be related to the bone directly. Increased levels of serum calcium, phosphate, or Vitamin D hypervitaminosis may be associated with the calcification process, causing mass accretion in the tissues like bones of limbs, and cranium, or showing some dental abnormalities. During the routine dissection of the hamstring compartment of the left lower limb in a male cadaver aged around 70 years in the Department of Anatomy, we noticed nodular fragments and lengthy slender pieces, which were hard in consistency and bony in its appearance found within the semimembranosus muscle. The histopathology of these masses was revealed as "Dystrophic Calcified Masses". A holistic description of calcification changes in the soft tissues will be considered in the article.

Keywords: calcification, dystrophy, hypervitaminosis

INTRODUCTION

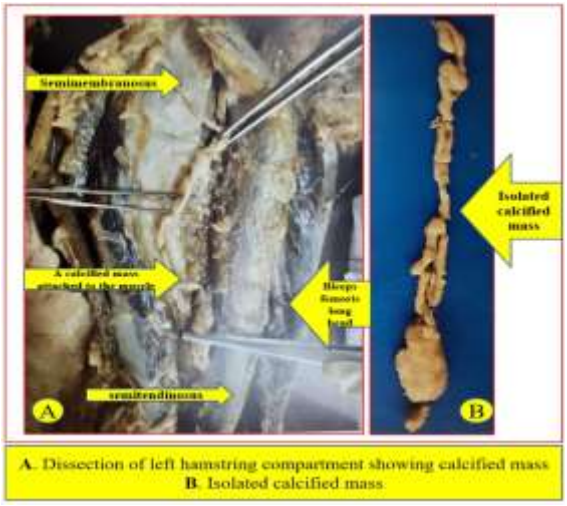
Calcium is one of the largest components of micronutrient contents in the body; calcium in cell signaling as a secondary messenger. It plays a vital role in several physiological events like muscle contraction, nerve conduction, haemostasis, bone formation[1], etc. Soft tissue calcification is a common phenomenon affecting a wide range of tissues in the body; it could be of different varieties like dystrophic, iatrogenic, metastatic, or idiopathic[2]. Tissue calcification may involve various types of calcium salt depositions like calcium carbonate, calcium phosphate, hydroxyapatite, calcium apatite, calcium oxalate, etc. Undue tissue calcification may escalate the risk of cardiovascular diseases. Where deteriorating cell metabolic waste scavenging activity in prone tissues may add to its risk[3].

Ectopic calcification occurs due to deranged physiological events, where there is an imbalanced calcium regulation causing undue soft tissue changes that often may lead to mechanical stress. Later may involve various tissues like bone, muscle, cartilage, blood vessel, etc. The ectopic calcification may lead to disorganization of anatomical entities leading changes which in turn affects its functionality[4]. Calcification stands as an important part of the physiological or pathological event which is tightly regulated by various genetic mechanisms and influenced by host and environmental factors. All undue soft tissue

ectopic calcifications may not lead to bone formation, but it stands as a mere deposition of various forms of calcium salts leading to the calcification in various tissues[5]. One such case of skeletal muscle dystrophic calcification will be considered for discussion

CASE REPORT

During the dissection of the left hamstring compartment of the thigh, we noticed an irregular shaped hard mass that was found intimately attached to the tendinous portion of the semimembranosus muscle (**Figure: A**). The mass was isolated from the soft tissue and subjected to histopathology examination and confirmed as a "Dystrophic Calcified Mass" (**Figure: B**).



A. Dissection of left hamstring compartment showing calcified mass
B. Isolated calcified mass

DISCUSSION

Dystrophic calcification refers to the undue deposition of calcium and phosphorous salts in soft tissues of the body. It may be associated with systemic diseases, or it may be a local phenomenon that may occur without indicating explicit underlying metabolic impairment[6] Le. Calcification is seldom appreciated as a mere advanced aging process or it could be associated with a grievous underlying condition like cancer metastasis which accounts for tissue metabolic calcification change[7]. Soft tissue insults due to injury, inflammation, infection, etc. may result in calcium deposition in that the local site may gradually transform into complete or partial calcification of tissue[8]. As a part of senile changes, the calcification of costal cartilages and structures like ligamentum nuchae is commonly seen and is often considered physiological[9]. The secondary form of soft tissue calcification is seen with underlying disease conditions like chronic renal failure, parathyroid dysfunction[10], etc. Based on the particular tissue involved, location, and underlying factors, the calcifications can be categorized into four main varieties idiopathic, iatrogenic, dystrophic, and metastatic. Calcinosis is seen in diversified anatomical structures and locations in the body that may involve muscle, tendon, ligament, bone, vessels, etc. The joint is not an exception, it is rarely presented with the deposition of calcium pyrophosphate crystals in the articular disc due to chondrocalcinosis causing arthropathy. Often such changes are seen in radiological findings with systemic metabolic derangements with or without chronic renal dysfunctions. Such explicit radiological findings may create a bias in diagnosis, where clinical and biochemical data analysis will complement to narrow the bias toward diagnosis[2].

An unusual solid mass formation within the soft tissues needs to elucidate between calcification and ossification. The histopathology analysis can guide us to differentiate between these two phenomena. The microscopic findings showing trabecular or cortical pattern is suggestive of bone formation. In contrast, the calcification may appear as an amorphous hard mass with the deposition of hydroxyapatite, calcium pyrophosphate, calcium pyrophosphate dihydrate[11], etc. In the present case report, histopathology findings show dystrophic calcification, which is suggestive of calcific tendinopathy because of its close structural association with the tendinous part of the semimembranosus muscle. Calcific tendinopathy is more frequently presented findings in the population, such calcification can be recognized apparently under radiological aid. In vivo experiment has shown that the

dystrophic calcification changes are initiated by soft tissue injury. If dystrophic calcification is extensive, it may lead to heterotrophic ossification may result in bone formation. A greater dystrophic calcification process can potentiate the induction of the heterotrophic ossification process by suppressing TGF-β1 signaling due to reduced immune response leading to the formation of bone tissue[12].

Myositis ossificans is a condition that implies inflammatory change followed by ossification of soft tissue which resembles calcification. It is a benign condition showing bone formation suggestive of ossification changes within the muscle tissue, most of the time this condition is underlined with previous subtle injuries to the muscle fibers which is common in athletes. Soft tissue calcifications can occur in diversified locations in the body with nonspecific local reactions. Myositis ossificans lesion is one such condition seen with multiplying fibroblast and osteoblast cells[13]. Dystrophic calcification in the soft tissue may mimic myositis ossificans which needs clear evaluation[14]. Though the pathophysiology of myositis ossificans remains uncertain, it is considered that acquired tissue calcification can happen with the previous tissue insult due to injury. Later initiates endothelial-mesenchymal transition where the vessel wall endothelium transforms into mesenchymal stem cells through a cascade of molecular events that later transform either into osteoblast or chondroblast cells, leading to the formation of bone or cartilage respectively[15]. Such unusual changes can be recognized apparently under radiological aid.

Excess dietary intake of calcium with excess circulating serum calcium could result in undue bone mass accretion[16]. The process of calcification may not spare any particular tissues in the body, the calcium deposition can involve the heart papillary muscle which stands as a substantial structural change but is not uncommon. Such changes may simultaneously be associated with changes in the coronary arteries or other structural entities of the heart like the atrioventricular valve, semilunar valvular, or other systemic diseases involving chronic renal malfunction[17]. Calcification changes were noticed even during the oral and maxillofacial radiological evaluation of the head and neck region. A study has shown incidental calcification changes in soft tissues like salivary ducts, where the sialolith was the most frequent clinical finding among other salivary duct calcifications[18]. Several factors like genetic, nutrient, host, and environmental interactions, etc., will influence the calcification process in the body. Later is enhanced by an underlying disease like diabetes mellitus, and other biological factors like leptin, calcitriol, TNF α (tumor necrosis factor), etc. Its inhibitors are including magnesium, BMP (bone morphometric protein), pyrophosphates, parathyroid hormones, etc. Undue extraosseous calcification phenomenon may likely influence the various soft tissues that may emerge as a way long and progressive subclinical biological phenomenon unless it is explicitly manifest as a disease pertaining to specific tissues in the body. Where the involvement of the coronary artery may result in life-threatening consequences[19]. Hence calcification changes may need much attention from a clinical standpoint.

CONCLUSION

Calcification can happen as a hereditary phenomenon or it could be a spontaneous mutational change that transforms soft tissues of the body into calcified material. Based on the location and size of the calcified mass it may draw immediate clinical attention or may remain asymptomatic hence often they are incidental radiological findings. The present case report has shown the formation of dystrophic calcification change only in the left semimembranosus hamstring muscle, indicating incidental asymmetry; hence the probability of traumatic calcification in the hamstring muscles cannot be ruled out. Though our case report is a mere isolated

calcified mass in the soft tissues met during dissection involving preclinical medical students, where the absence of first-hand clinical information opens large assumptions about the diversified possibilities in understanding the disease holistically.

Declaration by Authors

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