

Multiple Nodular Swelling in Both Upper and Lower Limbs

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Fig.-1: Showing Soft tissue calcification involving the forearm and hand



Fig.-2: Showing nodular swelling with chocky surface in hand and forearm



Fig.-3: Showing Soft tissue calcification involving the foot

A 17 year- old- boy presented with multiple nodular swelling in right upper limb and feet for 2 years ,and proximal muscle weakness for 2 months. Two years ago patient notice a small nodular swelling over wrist, which was farm later become hard ,with the passage of time he develop multiple nodular swelling without any limitation of daily activities. For last 2 month he develop difficulty in standing from sitting position. Such types of illness didn't run in his family, patient have not histry of taking myopathic drugs . General examination reveals multiple nodular swelling involving ulnar surface of right forearm, 2nd & 5thmetacarpophalangeal joint ,wrist ,right foot , which are nontender , most of them are hard with few firm in consistency, fixed with underlying structure but free from overlying skin without discharging sinus, largest one is (2,2 cm). On CNS examination only muscle power of proximal group of lower limb 4/5 ,feature of proximal myopathy. Investigation shows Hb 11.2g/dl, ESR 15 mm (1stHr), TC 15,000., CPK : 2881 U/L, S. Creatinine, RBS , S.uric acid , S.calcium , S.albumin,Thyroid function test, S. electrolytes and Urine R/M/E are normal. CRP is negative, SGPT : 110 U/L , SGOT : 223 U/L. ANA and Anti-Centromere Ab are negative. Muscle biopsy features consistent with dermatomyositis. Prednisolone 40mg was administered daily with symptomatic improvement. In a recent follow up patient muscle weakness was improved but no exacerbation or resolution of calcinosis was observed.

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Discussion:

When calcification is processed any tissue other than bone and teeth is termed calcinosis and can occur in many condition including connective tissue disease, hyper-parathyroidism, renal failure and vitamin D intoxication¹. Calcinosis may be divided into four categories according to the pathogenesis as follows; dystrophic, metastatic, idiopathic and iatrogenic. In connective tissue disease, calcinosis is mostly of dystrophic type and it seems to be localized process rather than an imbalance of calcium homeostasis. Calcinosis in connective tissue disease about 9% patient with scleroderma^{2,3} and 5% to 20% of adult⁴ and 40% to 74% of children with dermatomyositis. The existence of calcinosis is indicative of a good prognostic sign of survival but may also be incapacitating.

Dermatomyositis is an idiopathic inflammatory myopathy with characteristic cutaneous manifestation, including heliotrope rash, Gottron papules, periungual telangiectasias, photosensitive erythema, poikiloderma and alopecia. Although heliotrope rash and Gottron papules are specific features, calcinosis may occur up to 40% of children or adolescent.

The laboratory hallmarks are elevated creatine kinase, aldolase and transaminase, and a characteristic pattern of EMG-spotty muscle necrosis, regeneration, and inflammation are the pathological hallmarks. Calcinosis can be a disability complication that may affect the skin, subcutaneous tissue. It occurs most during the course of juvenile dermatomyositis³. Calcinosis usually occurred two or three years after onset of dermatomyositis, after that the deposition remains stable and spontaneous resolution has been occasionally reported⁵. The cause and mechanism of calcification are unknown. Calcium deposition is often in those muscles that were most severely affected during the acute phase of disease. Serum calcium, phosphate and urinary calcium values are within the normal range². The calcinosis can be demonstrable both clinically and radiologically. A whole body scan with ^{99m}Tc pyrophosphate and CT scan can also identify

calcinosis⁶. Aggressive treatment with high doses of prednisolone and physical therapy can decrease the incidence of calcinosis⁵. The use of bisphosphonate in the treatment of soft tissue calcification has varying results^{7,8}. Two studies show suppression of GIa synthesis by warfarin sodium may prevent deposition and allow for removal of existing calcinosis. Large and localized masses may be removed surgically^{9,10}.

Conclusion:

Calcinosis often signals a improved prognosis. Spontaneous regression of calcification was reported up to 50% of the cases.

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