Ocular Manifestations of a Child with ALL after Chemotherapy

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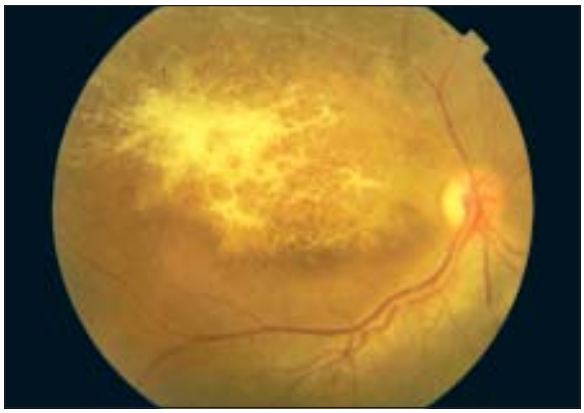

Fig.-1 A: *Fundus photograph of Right eye at presentation shows scared retina in the posterior pole involving macula.*

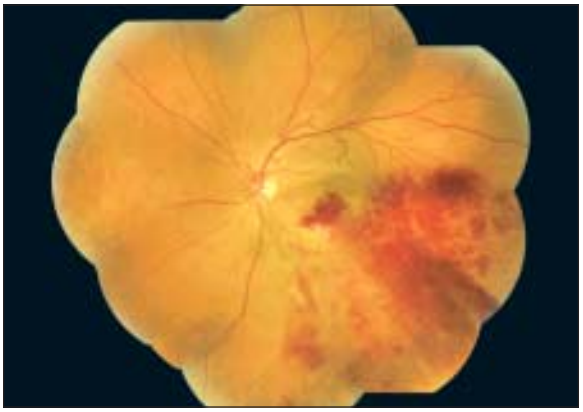

Fig.-1 B: *Fundus photograph of Left eye shows retinal hemorrhage against a background of edematous retina in the posterior pole, also there is vascular sheathing.*

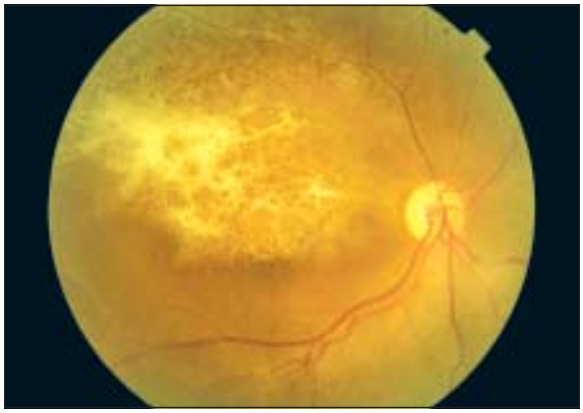

Fig.-2A: *Fundus photograph of Right eye after treatment shows scared thined retina in the posterior pole involving macula.*

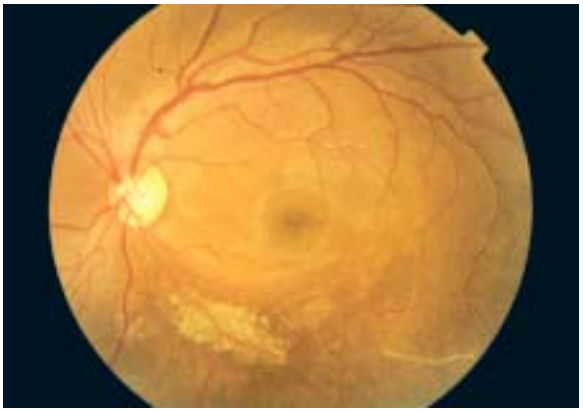

Fig.-2B: *Fundus photograph of Left eye shows healed lesion leaving behind small scar outside the macula.*

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A nine years old boy presented with sudden loss of vision both eyes. On examination his best corrected visual acuity was HM in right eyes , 6/60 in left eye, anterior chamber was quiet in both eyes, crystalline lens were clear in both eyes, vitreous was clear in both eyes. The fundus examination with indirect ophtalmoscope right eye shows granular area with thinned scared retina in the posterior pole involving macula(Fig:1A), left eye

shows a larger areas of retinal hemorrhage against a background of edematous retina in the posterior pole extending from disc to the vascular arcades, along the distribution of nerve fibers and associated blood vessels (Fig:1B). This type of retinal hemorrhage, retinal edema and vascular sheathing may be due to vasculitis retinae due to any cause of vasculitis (e.g- Eales disease, tubercular, sarcoid, syphilis, SLE, Behcet's disease etc),CMV retinitis. Retinal thinning and scar may be due to any type healed retinal necrosis (Acute retinal necrosis, Cytomegalovirus retinitis, Progressive outer retinal necrosis, Behcet's disease, SLE etc). Clinically we diagnosed as healing CMV retinitis in right eye and active CMV retinitis in his left eye. He is a diagnosed case of acute lymphoblastic leukaemia under chemotherapy since 31 months. He was medicated with vincristine, asparaginasae, methotrexate, daunorubicin, 6-mercaptopurine,cyclophosphamide, Cytosine Arabinoside, oral prednisolone. He was negative for HBs Ag/HIV/HCV. Laboratory tests done by oncologist after diagnosis of CMV retinitis showed blood count within the normal range, erythrocyte hemosedimentation rate of 60 mm, normal C3 and C4, normal urine routine,microcopical examination and absence of proteinuria in the 24 hour collection, positive IgM and IgG CMV serology, positive PCR for CMV from blood. After 4th cycle of chemotherapy he developed ocular symptoms. The induction was initiated with I/V ganciclovir 12 hourly for four weeks followed by I/V ganciclovir once daily for 40 days, up to negative CMV DNA from blood under supervision of oncologist. Subsequently the lesion started to regress. Serial CBC, liver function test and DNA for CMV from blood were done 2 weekly. After 4 weeks of treatment anterior uveitis and vitritis developed which was treated with topical and oral predisolone. Two months after initiation of ganciclovir therapy CMV DNA was negative from blood. The fundus showed large area scarred thinned retina involving macula in right eye(Fig:2A) and small area of scar in left eye sparing macula (2B). The final visual acuity was 6/60 in right eye, 6/9 in left eye.

Discussion:

Cytomegalovirus (CMV) retinitis is a disease which mainly affects patients with acquired immunodeficiency syndrome (AIDS) as well as other immunosuppressed patients, such as the organ transplant recipients under immunosuppressive therapy, those on chemotherapy for malignant diseases, patients with autoimmune disorders like systemic lupus erythematosus (SLE) under immunosuppressive treatment.[1-4] There are a few published series of patient with CMV retinitis without HIV infection.[5,6]

Conclusion:

We believe it is important to inform the existence of this serious and rare clinical complication, especially in our community, where the chronic use of corticosteroids is a routine practice, warning of the irreversible consequences if early diagnosis and treatment are not established.

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Multiple Nodular Swelling in Both Upper and Lower Limbs

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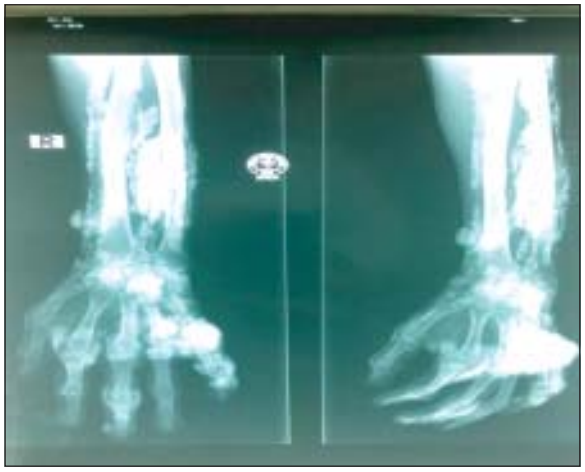

Fig.-1: *Showing Soft tissue calcification involving the forearm and hand*

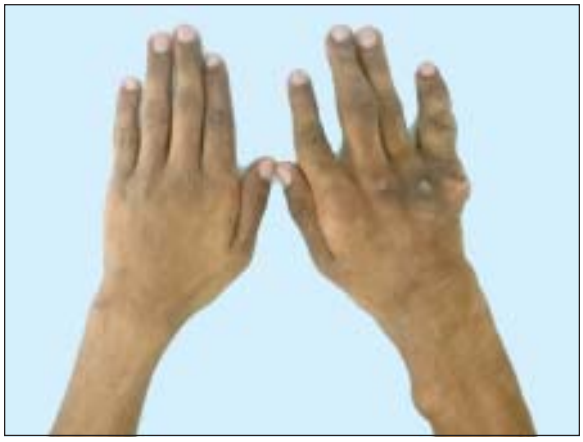

Fig.-2: *Showing nodular swelling with chocky surface in hand and forearm*

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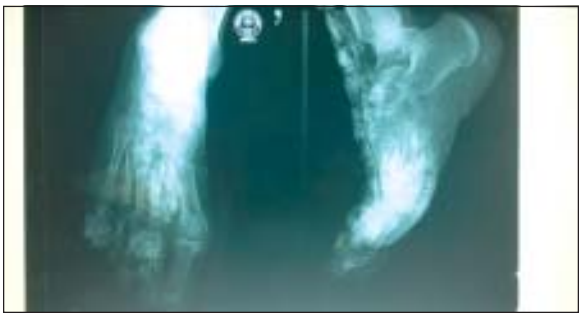

Fig.-3: *Showing Soft tissue calcification involving the foot*

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 passge 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.

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 intoxication[1]. Calcinosis may devided into four categories according to the pathogenesis as follows; dystrophic, metastatic, diopathic 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 seleroderma[2,3] and 5% to 20% of adult [4] and 40% to74% 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 helitroperash, gottronpapules, periungual telangiectasias, photo distribution erythema, poikiloderma and alopecia. Although helitrope rash and gottron papules are specific feature, calcinosis may occur upto 40% of children or adolescent.

The laboratory hallmark are elevated creatine kinase, aldolase and transaminase, and a characteristic pattern of EMG-spotty muscle necrosis , regeneration, and inflammation are the pathological hallmark. Calcinosis can be a disability complication that may affect the skin,subcutaneous tissue. It occur most during the course of juvenile dermatomyositis[3]. Calcinosis usually occurred two or three year after onset of dermatomyositis, after that the deposition remain stable and spontaneous resolution has been occasionally reported [5]. The cause and mechanism of calcification are unknown. calcium deposition are ofen in those muscle that were most severly affected during acute phase of disease. Serum calcium, phosphate and urinary calcium values are within the normal range[2] . The calcinosis can be demonstrable both clinically and radiologically. A whole body scan with 99m TC pyrophosphate and CT scan can also identify calcinosis[6]. Aggresive treatment with high doses of prednisolone and physical therapy can decrease the incidence of calcinosis [5] .The use of bisphosphonate in the treatment of soft tissue calcification has varing result[7,8].Two studies shows suppression of GIa synthasis by warfarin sodium may prevent deposition and allow for removal of existing calcinosis. Large and localized mass may be remove surgically[9,10].

Conclusion:

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

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