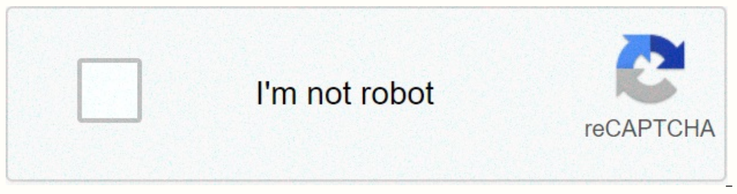


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# Pseudoxanthoma elasticum case report

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adulthood. Small yellowish papillary lesions in linear or reticulous patterns are found in the neck, axillary, groin, and bent folds. [10] As the disease progresses, the skin becomes loose, loose, redundant, and droops with a plinth that creates a typical plucked chicken appearance. The theology of PXE is characteristic: skin lesions show agglomerated and fragmented elastic fibers with calcium deposits in the dermal of medium and deep meshes. Similar changes occur in elastic fibers in the membranes, blood vessels, endometrium and other organs of the eye bruch. Abnormal calcium deposits can be demonstrated by von Cossa staining of connective tissue. Initially, mineralization of elastic fibers is seen as the central nucleus of electron density in electron microscopy, after which the core density increases as mineralization progresses. The mineralizing area of the dermis indicates the deposition of irregularly diameterd filamentous substances and collagen fibrifibria. Ultra-structurally, extracellular matrix components such as proteoglycans, fibronexin, vitronektin, and lesioned skin were found to accumulate. Rising levels of glycosaminoglycan were seen in affected skin and urine in some patients with PXE[12]. Eighty-seven percent of patients with pseudo-emphysema elasticity were found to have angioid muscles (AS), which are seen as irregular radiation from ring-shaped areas around the disc and extend deep into the eye. Angioid muscles are seen as dark red to brown bands, among which they are variableVascular muscles arise from cracks-like rifts in bruch membranes due to an abnormal structural composition predisposition to these local areas of rupture. During fluorescence angiography, the endovascular muscle reveals an increase in the visibility of the choroid due to local defects in the membrane of the bruff and an increase in fluorescence in the early stages at a later stage due to leakage from adjacent villi. The similarity of frequent clinical associations and histopathological changes in vascular muscle and intervertebral degenerativity in the macular region indicates some etiological links between them. Vision loss and macular involvement usually appear after the age of 40. The endovascular muscles may progress slowly or remain stationary for years. Two possibilities have been suggested to be the cause of endovascular muscles: [1] changes in bruch membrane dilation associated with several other pathological conditions. [2] In other tissues of the body, primary deformation of bruch membranes that are bilateral and have elastic tissue deformation. Cerebral glandular defects are more susceptible to choroidal neovascular formation, which can lead to subcutaneous renal hemorrhage and ultimately disk-like dilation. Loss vision due to visual disc dolzene has been reported in some patients with pseudo-emphysema elastics with vascular gland muscles. [13] The prognosis is often very poor due to choroidal rupture and renal hemorrhage, which can occur in patients with pseudocardioma elasticity due to microocular trauma. Choroidal new angioplasty (CNV) can treat surgery, photocoagulation, and photodynamic therapy with various successes.[15] Intracellular vascular endothelia growth factor (anti-VEGF) agents should be considered for patients with choroidal recitation. Intra-vitrial injections of the off-label use of vigitumab or bevacizumab in afribercept or ranibizumab seem to maintain vision [15]. Cardiovascular symptoms include calcification and intermittent claudication in the elastic tissues of the intestinal membrane, the medium of blood vessels leading to coronary artery and cerebrovascular disease [10]. The valve membrane change can exist mainly, mitral valve detached. Early PXE-related coronary artery disease is often severe, and most cases are presented as early angina pectoris or myocardial infarction. In some cases, coronary artery disease led to sudden death. Strokes can also occur as a result of ischemic or hemorrhagic cerebrovascular disease. Gastrointestinal bleeding is often dramatic and recurrent. The earned form of PXE has been mentioned in many case reports as PXE with skin and eye symptoms, which, like hereditary PXE, does not involve ABCC6 mutations with no genetic basis. This form of PXE may be associated with autoimmune thyroiditis, congenital anemia such as sickle cell disease, and other conditions such as cases of spherocytosis. In combination with PXE-like skin lesionsβ and calcified elastic fibers were detected in patients with thalassemia. No disease-causing variants have been found in the ABCC6 gene, indicating that this is pxe phenosis. All these conditions associated with the earned PXE indicate the deformation and fragmentation of elastic fibers infiltrated with calcium, resulting in revealing clinical and histological changes. If you damage elastic fibers under the above conditions, it can result from focus, mechanical, and biochemical stimuli on connective tissue that induce foreign body reactions and lead to their deformation[6][18]. Differential diagnosis includes papillary skin adenolysis, papillary elastolex, puncture pseudo-venomous cell tumor elasticity, severe actinous damage to the end of the neck, penicillamin therapy and skin relaxation. Serious eye lesions such as PXE can occur in some cases. The generated vascular muscle in hemochromaptosis can be a reflection of iron deposits. [19] Vascular gland muscles were also shown in patients with bone paget disease and hyperphosphatemia tumor carcinopathy due to calcium deposits in relatively normal Bruch membranes. Dietary restrictions on calcium have been tried with limited results, but there is no treatment to directly interfere with this multifaceted disorder. In excessive areas of the skin, plastic surgery may be necessary. Avoidance of head injuries and severe tension are necessary to prevent renal bleeding. [20].