Chorea - A Presenting Feature of Giant Cell Arthritis

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Introduction

Chorea occurs as a hereditary or non-hereditary disorder. Non-hereditary chorea most commonly occurs in rheumatic fever (Sydenham’s Chorea) but may also be encountered in other metabolic and systemic disorders. Green House and Albuquerque listed more than forty different causes of chorea. The etiology of non-rheumatic chorea may cause diagnostic difficulties, due to its rarity. We report a 75 years old patient with giant cell arteritis presenting as chorea. Introduction Chorea occurs as a hereditary or non-hereditary disorder. Non-hereditary chorea most commonly occurs in rheumatic fever (Sydenham’s Chorea) but may also be encountered in other metabolic and systemic disorders. Green House and Albuquerque listed more than forty different causes of chorea. The etiology of non-rheumatic chorea may cause diagnostic difficulties, due to its rarity. We report a 75 years old patient with giant cell arteritis presenting as chorea.

Case Report

A seventy-five years old non-smoker male presented with malaise, bodyache, headache mid feeling unwell. He was treated with Tab. Diagesic (Dextropropoxyphene+Paracetamol). A month later, he complained of binning of vision in the right eye and abnormal movements on the left side of the body. History did not reveal any movement disorder in the family and he did not take any medications except Diagesic. There was no history of diabetes, hypertension or alcohol intake. On examination, the pulse was 86/min regular, all the pulses were intact and the temporal arteries had a granular feel on palpation. Blood pressure was 170/90 mmHg. Chorea form movements were noted on the left side of the body. Right disc appeared pale and a formal ophthalmological opinion confirmed Ischemic Papillitis on the right side. Left optic disc was normal. Respiratory system did not reveal any abnormality and the rest of the systemic examination was also unremarkable. Right temporal after biopsy was cell arteritis may suggest a ‘Malignant Course’ possibly due to extensive cerebral vessels involvement and the performed on the same day and 80 rug prednisolone was given daily in divided doses investigations at this stage included: ESR, 96mm at the end of is hr. and haemoglobin 12 Gm%. Total and differential leucocyte counts were normal, Blood sugar 7m mol/L, se mm calcium 2.4 mmol/L. ASO titre, ANF, T3, T4 and TSH levels were normal. VDRL was negative. ECG and echocardiography revealed no abnormality. Chest and skull x-rays and CT scan of the skull were normal. Histological changes in temporal artery showed giant cell arteritis. Three weeks later, he developed choreiform movements on the right side as well, followed by complete blindness in the left eye. It was again confirmed to be due to ischemic papillitis. The patient died two months later with disabling chorea.

Discussion

In 1960, Paradise suggested that chorea may be expected though not documented in conditions like polyarteritis and deramatomyositis, i.e., conditions causing arteritis. Chorea has been reported in SLE, Henoch-Schonlen purpura and in association with central retinal artery occlusion. Chorea occurring in cases of giant cell arteritis is unusual. Giant cell arteritis is a disorder resulting in systemic vasculitis particularly involving the cranial- vessels. The sudden onset and stepwise pattern of chorea in this
patient was most likely due to cerebral vessels involvement. Giant cell arteritis does not affect the life span\textsuperscript{4} but in this patient the course was quickly progressive, inspite of steroids. Thus chorea in giant disease may be resistant to conventional doses of steroids.

References