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Case Report

Hypercortisolism induced atypical central serous chorioretinopathy in pregnancy

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Introduction

Pregnancy can cause changes in the functioning of the eye in health and in disease, just as it modifies other non-reproductive systems of the body. Central serous chorioretinopathy (CSCR) is a relatively common retinal disease characterized by the accumulation of sub retinal fluid at the posterior pole of the fundus, creating a circumscribed area of serous retinal detachment. It typically affects young and middle-aged men with no previous medical and family history, and no systemic symptoms or signs. However, pregnancy, predisposed by endogenous hypercortisolism, probably represents a risk factor for central serous chorioretinopathy.[1] Many cases of central serous chorioretinopathy have been described during or following treatment with glucocorticoids, administrated by any route, for various systemic or ocular conditions, when the CSCR is generally atypical, bilateral and has less male prelidiction.[2] We present here the nuances of a case of a concomitant endogenous hypercortisolism (pregnancy) and exogenous hypercortisolism (treatment with oral corticosteroids) leading to a unilateral, atypical CSCR with sub retinal exudates in a 26 year old second gravida.

Case report

A 26 year female patient, $G_2P_1L_1$ booked antenatal case in our hospital had an episode of fever with vomiting at 30 weeks Period of Gestation (POG) at her hometown and was treated locally with antibiotics and antiemetics of which no details were available. A day later on 16 Jan 12, she developed mildly itchy reddish brown lesions over the body. The lesions were well defined erythematous papules distributed over face, trunk, limbs, involving palm and soles but preferentially sparing the upper thighs, abdomen and the oro-genital mucosa. The lesions were 2–4 mm in size, non

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tender with no signs of excoriation or secondary infection. She denied history of developing rashes in the previous pregnancy. CBC, blood glucose, ANA, HBsAg, Anti HCV, IgM HEV, IgM HCV, APLA workup, VDRL, ELISA HIV, ASO titre were all normal. C Reactive Protein was positive and LFT was deranged with elevated transaminases. She was diagnosed as a case of drug induced cutaneous vasculitis with a differential diagnosis of Pruritic Urticarial Papules and Plaques of Pregnancy (PUPPP) and treated with Tab Prednisolone 40 mg OD for seven days tapered by 10 mg every five days, supportive therapy and topical steroid cream. She responded to the treatment and the cutaneous lesions regressed (Fig. 4). Obstetric examination and investigations were essentially normal. On 27 Feb 12 (POG 37 5/7) the patient reported sudden, painless diminution of vision in the right eye. She denied pain with eye movement, photopsia, diplopia, tearing, redness, foreign body sensation, systemic weakness, seizure activity, loss of sensation, or difficulty in speaking or swallowing. Best Corrected Visual Acuity (BCVA) right eye (RE) was counting fingers close to face and accurate projection of rays and 6/6 in the left eye (LE). Anterior segment was normal in both eyes and she had no relative afferent pupillary defect (RAPD) RE. The fundus of the right eye revealed an elevated disc, blurred temporal margin of the disc with neurosensory detachment and fluid collection extending from the temporal peripapillary region to and involving the macula. The sub retinal fluid had yellowish-white fibrinoid exudates in the macular area forming a macular star and extending into the pappillomacular area (Fig. 1). The rest of the fundus did not reveal any abnormalities. LE fundus was normal. Optical Coherence Tomography confirmed the above findings and fundus fluorescein angiography was not carried out due to the pregnancy (Fig. 2). VEP revealed a normal latency of P_{100} in both eyes but a delayed amplitude in the right eye (Fig. 3). Morning serum cortisol was 22.21 μg/dL ($N = 4.3–22.4$) and 24 h urine free cortisol was 830 μg/24 h ($N = 28.1–213.7$). Both central and peripheral fields were normal. B scan did not reveal any scleral or choroidal thickening or T sign. She was treated conservatively and put on Gt Nepafenac 0.1%q 8h. The patient went into spontaneous labour on 5 Mar 12 and delivered a live healthy baby. She was kept under follow up, when the fibrinoid exudates were found to be spontaneously thinning and disappearing. The BCVA RE on 1 May 2012 was 6/9 (p-1).

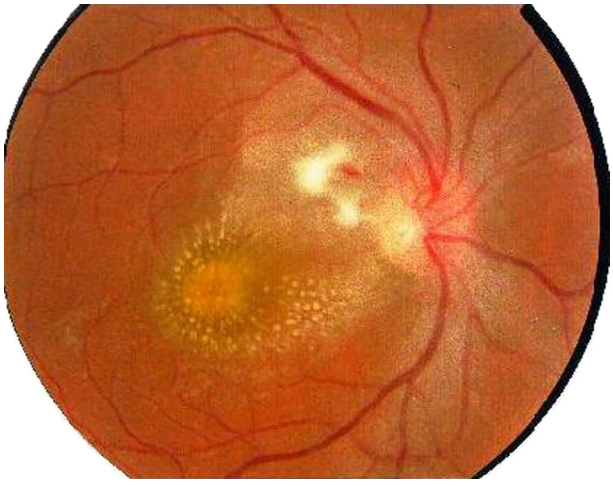

Fig. 1 – Fundus photograph OD, showing swollen disc with blurred temporal margin due serous detachment extending from the optic disc to the macula. The edge of the detached area is lined by fibrinoid exudative sub retinal precipitates.

Discussion

A number of investigators have reported the development of central serous chorioretinopathy (CSCR) during the first, second or third trimesters of otherwise normal pregnancies. In all cases, symptoms resolved and vision recovered following delivery, with only mottling and clumping of the retinal pigment epithelium as evidence that the event had occurred.

Proposed causes for the association of CSR with pregnancy include hormonal alterations and haemodynamic alterations – increased red blood cell volume and cardiac output, changes in permeability along with decreased colloid osmotic pressure and hypercoagulability. Since CSCR is much more common in males than females (10:1), one might not think of the diagnosis of CSCR in a pregnant woman.

The salient points needing discussion in this case are 1) Differential diagnosis 2) Effect of concurrent exo and endogenous hypercortisolism 3) Atypical features of CSCR in pregnancy.

Differential diagnosis

In this case we entertained the diagnosis of a) Posterior Scleritis b) Non Arteritic Anterior Ischaemic Optic Neuropathy (NA-AION) c) Atypical CSCR d) Acute Retinal necrosis e) Toxaemia induced retinopathy f) Retinal vasculitis similar to the drug induced cutaneous vasculitis the patient suffered.

Though more common in pregnancy, Posterior Scleritis was ruled out due to the absence of pain and normal B scan.

NA-AION was ruled out due to the age of the patient, substantial sub retinal fluid collection and absent RAPD. A normal VEP latency with reduced amplitude can occur in both NA-AION and sectoral retinal lesions.

Acute Retinal necrosis due to reactivation of Herpes simplex virus and Herpes zoster has been reported during gestation. This was ruled out by no history of prior herpetic diseases, such as fever blisters or genital herpes, absence of anterior uveitis and central location of the retinal pathology.

A normal blood pressure, no other retinal vascular changes of Toxaemia, unilateral pathology and absence of proteinuria helped exclude retinopathy of toxaemia.

Retinal vasculitis caused by the same auto-immune process which led to the dermal vasculitis was a very tempting diagnosis to make. However the unilateralism of the disease, limitation of most of the pathology to the relatively avascular macular area and normal retinal vasculature everywhere else ruled out this diagnosis.