

Serous Retinal Detachment Triggered By Eclamptic Attack

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Citation

H Kafali, H Oguz. *Serous Retinal Detachment Triggered By Eclamptic Attack*. The Internet Journal of Ophthalmology and Visual Science. 2002 Volume 2 Number 1.

Abstract

We report two cases of bilateral serous retinal detachment which appeared to be precipitated by valsalva maneuver occurred during eclamptic crisis.

INTRODUCTION

Preeclampsia is classified as a hypertensive disease of pregnancy, which can occur in the absence of other causes of elevated blood pressure and in combination with edema, proteinuria or both (1). This systemic disease can affect almost every organ system in the body and has potentially devastating consequences for both mother and fetus. Ocular involvement has been reported in 30% to 100% of patients with preeclampsia and its ophthalmic manifestations include retinal arteriolar spasm, optic neuropathy, cortical blindness, and serous retinal detachment (1,2). Serous retinal detachment which was first reported by Von Graefe is an unusual complication of hypertensive disorders of pregnancy. It has been reported in 1% to 2% of patients with severe preeclampsia and in 10% of patients with eclampsia (3). Although retinal and choroidal vascular damage has been implicated in the pathogenesis, exact mechanism of retinal detachment associated with preeclampsia is still unclear. We herein report two cases of bilateral serous retinal detachment which appeared to be precipitated by valsalva maneuver occurred during eclamptic crisis.

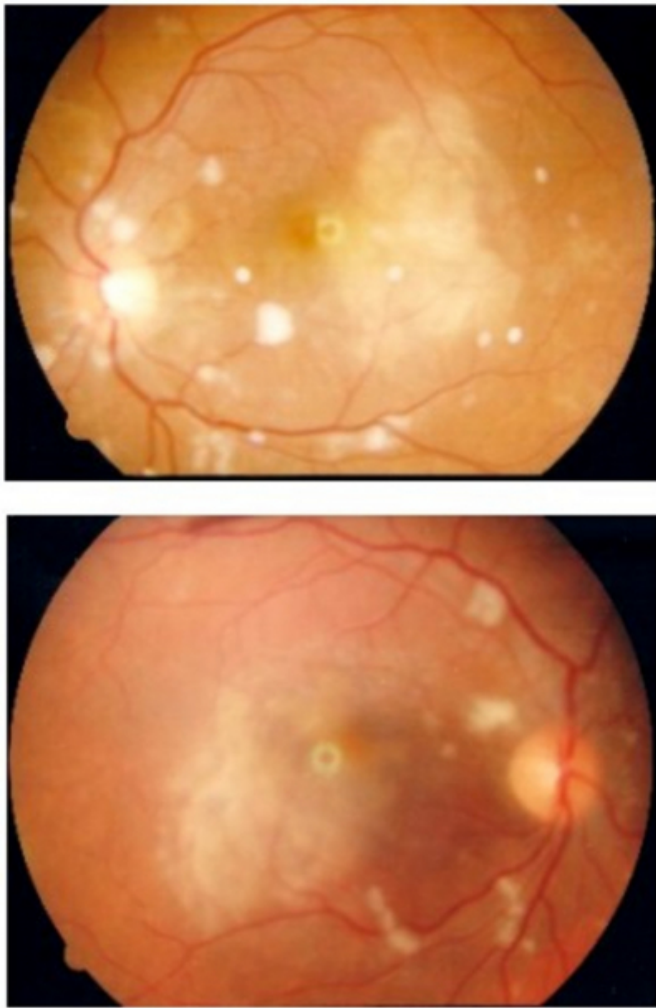
82/min and respiratory rate 16/min. There was a pitting edema of legs and obvious edema of the face, eyelids and conjunctiva. Proteinuria (4+) was noted on a catheter specimen. Funduscopy evaluation revealed normal finding. Standard procedures for management of eclampsia were instituted. Immediate management included an intravenous bolus dose of 6 g magnesium sulfate over 15 min was given then magnesium sulfate was infused at a rate of 1,5 g /h. In spite of initial therapy she had tonic-clonic seizures. She made a slow recovery; three hour later she was alert but complained of blindness. Funduscopy examination revealed bilateral retinal detachment, cotton-wool spot and exudates (Figure 1a, 1b).

CASE REPORTS

CASE 1

A 21- year-old primigravida with 28 weeks of gestation was admitted to local state hospital with severe headache. After initial examination she was referred to our clinic with a diagnosis of severe preeclampsia and intrauterine exitus. The history obtained from her relatives on admission, which revealed that she had had no prenatal care, and there was no past medical history of hypertension, seizure disorders, and any major disease. On admission she was edematous, agitated. The blood pressure was 160/110 mmHg, pulse rate

Figure 1



Labor was induced with 200µg misoprostol (Cytotec®, Ali Raif, Turkey); and 850g, ex, female fetus was delivered. During postpartum period, her vision returned slowly. By the 7th post operative day, she could see and read without any difficulty and her retinal detachment was no longer present.

CASE 2

A 24-year-old, primigravida was admitted to hospital at 37 weeks gestation. She complained of a one-week history of progressive anorexia, right upper quadrant abdominal pain. She also noted mild swelling of her ankles, feet and hand. She denied having a headache, scotoma or vaginal bleeding. Her antepartum course had been otherwise uncomplicated. Physical finding on admission included a blood pressure of 170/100, 3+ pedal edema, abdomen with a term-sized fundus, and normal fundusoscopic examination. Management of eclampsia followed routine procedures. Because the patients had a cervix unfavorable for induction as previous case, cesarean section was performed and a 2600 g male

fetus delivered. Despite of fact that patient was under treatment of magnesium sulfate, two eclamptic attacks were occurred at postoperative period. Three hour later she awoke and complained of blurred vision. After fundusoscopic examination, bilateral serous retinal detachment was observed. The patient made a rapid recovery and her vision was normal within 72 hours.

DISCUSSION

Wide ranges of ocular abnormalities resulting from preeclampsia are reported. The most common abnormality seen in the visual system is spasm and narrowing of the retinal vessels reported in 70% of cases of toxemia (⁴). The degree of retinal narrowing seems to parallel the severity. At first, focal areas of spasm may be observed, which progress to more generalize narrowing as the preeclampsia worsens. Other changes including retinal hemorrhage, papilledema, cotton-wool spots, and arteriosclerosis can also be observed. Although these background changes are rare in patients with “pure” preeclampsia and their occurrence usually associated with systemic disease such as chronic hypertension and diabetes, one of our patients has demonstrated cotton-wool spot, and exudates despite of not having underlying such a systemic disease. Retinal hemorrhage and papilledema in preeclampsia characterize patients with an acute, severe rise in blood pressure. Our patients did not have blood pressure greater than 170/110 mm Hg. if they had had more markedly increased blood pressure, it is likely that they would have manifested these evidences of retinal vascular decompensation.

Temporary blindness associated with preeclampsia has been reported as originating from diffuse, generalized spasm of retinal arterioles, thrombosis of the central artery, papillophlebitis, cerebral spam and edema (⁵). Perhaps one of the best known, although rare, causes of loss of vision associated with preeclampsia is serous retinal detachment (⁶). Serous retinal detachment as a complication of preeclampsia can occur at antepartum or postpartum period even in the presence of only mild hypertension and in absence of significant retinal vascular abnormalities; however it is usually present in patients with severe preeclampsia or eclampsia (^{4, 6}).

Serous retinal detachment of our cases occurred in the presence of severe hypertension but in the absence of significant fundusoscopic abnormalities.

The pathophysiology of serous retinal detachment in preeclampsia is not fully explained. Choroidal and retinal

vascular damage has been postulated as the cause of retinal detachment. The theory that vascular damage causes retinal detachment is supported by experimental work by Collier who produced reversible retinal detachment in 14 of 23 cats by inducing choroidal ischemia with latex microsphere suspension (⁶). Cogan reported similar findings of serous retinal detachment secondary to thrombotic occlusion of choriocapillaries in patients with disseminated intravascular coagulation (⁶). The events in such cases have been suggested to occur as a result of localized ischemic injury to retinal pigment epithelium with the resultant accumulation of subretinal fluid at a rate exceeding the capacity of the epithelium to remove it.

Ocular pathologies resulting from Valsalva maneuvers were coined by Duane in a series of patients with hemorrhagic retinopathy following an abrupt rise of intra-thoracic or intra-abdominal pressure. Several reports of sudden loss of vision following coughing, vomiting, heavy weight lifting have been reported (⁷). Valsalva retinopathy has been also reported in a pregnant woman (⁸). In the present study both of our patients had a virtually normal fundusoscopic finding before their eclamptic episode and they have developed bilateral retinal detachment after the convulsions. Therefore, we speculate that pre-existing retinal or choroidal capillary destructions as a result of ischemia combined with sudden increase intraocular pressure as a result of Valsalva maneuver might be the predisposing factors for the retinal detachment.

Although such a phenomenon has not been reported previously, it is important to note that a Valsalva maneuver can cause different pathologies in the eye including a large central preretinal hemorrhage, diffuse retinal hemorrhage,

Purtscher retinopathy and blindness associated with a hematoma of upper and lower lid of eye, probably caused by bleeding in the sheaths of the optic nerve (⁹). Since not all patients experiencing eclamptic episodes develop retinal detachment we believe that it may be only a trigger factor for retinal detachment in the presence of previous retinal and choroidal capillary damage.

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