ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 07 | July - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

NAVIGATING THE MAZE WHEN BOTH EYES SUFFER : A CASE REPORT OF VALPROIC ACID INDUCED BILATERAL CRVO IN A YOUNG PATIENT WITH HYPERHOMOCYSTINEMIA

Ophthalomology

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ABSTRACT

Central retinal vein occlusion (CRVO) is one of a common cause of visual impairment. It commonly affects men and women equally and occurs predominantly in persons over the age of 65 years. In this population, there may be associated systemic vascular disease, including hypertension and diabetes. Younger individuals who present with clinical picture of CRVO may have underlying hypercoagulable or inflammatory etiology. Sodium valproic acid is an antiepileptic drug extensively used for treating partial and generalised seizures, acute mania, and prophylaxis for bipolar disorder. VPA causes hepatotoxicity which in turn causes hypercoagulability. Drug induced liver injury(DILI) persists as significant issue related to fatal outcomes by VPA. Here is a 35 yr old young patient with h/o intake of sodium valproate for 10 yrs, presented to our opd with B/L CRVO with hyperhomocysteine levels.

KEYWORDS

INTRODUCTION

CRVO(Central Retinal Vein Occlusion) is a retinal vascular condition that causes significant ocular morbidity. Younger individuals who present with clinical picture of CRVO may have underlying hypercoagulable or inflammatory etiology. Population based studies report the prevalence of CRVO at <0.1 to 0.4%. (Mitchell P, 1996) (Rogers S, 2010).

CRVO is usually a unilateral disease; however, annual risk of developing a CRVO in fellow eye is approximately 1% per year, and it is estimated that upto 7% of persons with CRVO may develop eye within 5yrs of onset in the first eye. Individuals with CRVO demonstrate significant decrease in the vision- related quality of life as well as increased illnesss burden with increased healthcare costs and resource use. (Natural history and clinical management of Retinal vein occlusion, 1997) CRVO may impact a person's ability to perform activities of daily living, especially in cases of bilateral CRVO or when ocular disease limits vision in the fellow eye.

CASE REPORT

A 37 year old male patient with no comorbidities came to opd with c/o Diminution of Vision for far distance that was insidious in onset and gradually progressive in nature since 2 weeks. He is k/c/o Bipolar disorder since 20 yrs and h/o epilepsy since 10yrs, on T.Divalproex. No other complaints.

On Examination:

Vn- CF@1m (BE), not improving with Pinhole, BCVA is CF@1m

On Dilated Fundus examination:

Patient was found to have intraretinal hemorrhages-both superficial flame shaped and deep dot and blot type(classic blood and thunder appearance), with dilated, tortuous retinal vein. Cotton-wool spots, and macular edema.

DISCUSSION

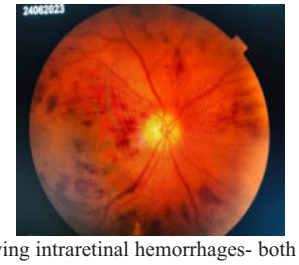
Hemodynamic alterations may produce stagnant flow and subsequent thrombus formation in the central retinal vein, including diminished blood flow, increased blood viscosity, and altered lumen wall(Virchow triad). Occlusion of both retrolaminar central retinal artery and central vein, posterior to the lamina cribrosa and prior to branching of collateral channels from the main trunk, was required to produce the clinical appearance of hemorrhagic(ischemic)CRVO (Hayreh SS, 1994). Hence concurrent retinal atery insufficiency or occlusion plays role in ischemic CRVO. Lahey and collegues studies have demonstrated an increased incidence of coagulation cascade abnormalities including protein C deficiency, protein S deficiency, activated protein C resistance, presence of factor V Leiden, presence of antiphospholipid antibodies, hyperhomocysteinemia, prothrombin gene mutations in young patients who reported with CRVO.

In addition, Sodium Valproate/Valproic acid- induced hepatotoxicity may interfere with production of coagulation factors. The mechanism of valproate toxicity is thought to be due to mitochondrial toxicity, by inhibition of beta oxidation. It lowers tissue carnitine levels which may affect mitochondrial function and cause hyperammonemia and microvesicular steatosis. According to the Banerjea study-2002 and Koenig study-2008, it causes coagulopathy by decreasing the Fibrinogen levels and Protein C levels. (Banerjea NC, 2002) (Koenig S).

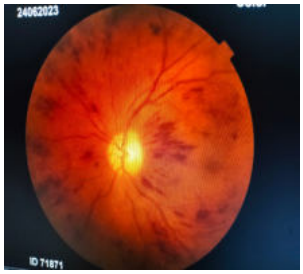
Our patient presented with intake of Sodium Valprote 500mg OD dose for 10 years which may have been the predisposing factor for CRVO. Increased Homocystein level – 22.60µmol/L (Normal is 5.46-16.2 µmol/L), LFT showed raised ALT levels, normal USG abdomen. OCT showed Spongiform macular edema in both eyes. He was given monthly injections of Anti-VEGF Ranibizumab for 3 months, and improvement in the vision upto 6/18 was observed. He was also prescribed T.Folic Acid OD dose for a month.

CONCLUSION

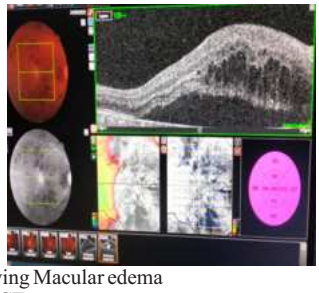
Valproate hepatotoxicity varies in severity from minimal and asymptomatic to severe liver injury. Monitoring of symptoms and LFT levels is recommended. In this case the increased intake of Sodium Valproate could probably be the reason for CRVO in addition to increased homocysteine levels. In majority of the cases, the prognosis of CRVO is good provided the treatment is taken at regular intervals and with treatment of the underlying systemic disease. Since the bilateral presentation is rare, early intervention, thorough examination and treatment with regular follow ups helps in better vision prognosis and better quality of life.



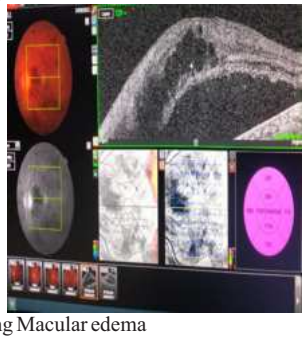
RE Fundus showing intraretinal hemorrhages- both superficial flame shaped and deep dot and blot type (blood and thunder appearance)
Figure 1- RE Fundus



LE Fundus with flame shaped, dot blot hemorrhages
Figure 2 -LE Fundus



RE OCT – showing Macular edema
Figure 3- RE OCT



LE OCT- showing Macular edema
Figure 4- LE OCT

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