Case report:

22-year-old male presented to KLES Hospital with sudden loss of vision in both eyes. He had history of generalized weakness, diarrhea and swelling of the body. There was no history of pain with eye movement, trauma or recent vaccination. Patient was not a known hypertensive or diabetic.

Ophthalmic examination:

- Visual Acuity- No light perception in both eyes.
- Pupils both eyes- 4mm, symmetrical, sluggishly reacting to light, consensual absent in the other eye.
- Fundus examination of both eyes- Disc edema with splinter hemorrhages near the disc margins, mild macular edema, arteries narrowed and veins dilated.

Systemic Blood Pressure was 150/100 mm of Hg.

Laboratory data included serum hemoglobin 11 g/dl, total count- 9590 cells/cu mm., Differential count- Neutrophils 77, Lymphocytes 14, Eosinophils 04, monocytes 05, Erythrocyte sediment rate- 28 mm in 1 hour, blood urea nitrogen-463 mg/dl, Serum creatinine- 21.3mg/dl, Random Blood sugar- 100 mg/dl, HIV- Non Reactive, HCV- Negative, VDRL- Non Reactive, HbsAg- Negative. CT- Scan of the Brain was normal.

Results of the Ultrasound Abdomen were Bilateral Grade I Renal Parenchymal changes. Neurologic examination revealed no deficits. The patient was started with immediate hemodialysis and intravenous MethylPrednisolone 125mg TID for 5 days. Hemodialysis was repeated after one day. Patient’s vision improved the next day after instituting treatment to Finger counting one and half meter in left eye and finger counting one meter in right eye. The disc edema reduced after three days.

Discussion:

A young patient presenting with sudden bilateral loss of vision, sluggish pupillary reactions and disc edema in both the eyes; we suspected either papillitis or optic neuropathy. There was no neurological deficit or any other systemic disorder detected on clinical examination. Blood pressure was150/100 mmHg, Blood Urea - 463 mg/dl, USG- Bilateral Grade I Renal Parenchymal changes suggestive of Acute Renal Failure . Correlating uremia with the disc edema and loss of vision, a diagnosis of Uremic Optic Neuropathy was made. Hemodialysis and intravenous methyl Prednisolone was started. A higher dose could not be given as the patient was in renal failure. Such ocular morbidity may result from Diabetes, Hypertension, anemia, hypotension, DIC, the uremic state itself, toxicity of concurrent medications, complications of dialysis or other intercurrent illnesses(6). In our patient, the optic neuropathy was from ischemia due to systemic hypertension or uremic toxins themselves. Prompt improvement in vision following hemodialysis and corticosteroids correlate with previous studies [5,7,8]. In the setting of uremia, anemia, systemic hypertension and other yet unknown factors associated with azotemia, it is possible that even a young patient may suffer an atypical and unusually severe episode of non-arteritic ischemic optic neuropathy [6]. Close collaboration among nephrologists, ophthalmologists and neurologists is important in this interdisciplinary emergency.

References:


Contact Details
J N Medical College & KLE Hospital, Belgaum, Karnataka.