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RESEARCH ARTICLE

PEDIATRIC BILATERAL ENDOGENOUS ENDOPHTHALMITIS POST MENINGITIS: A CASE REPORT WITH LITERATURE REVIEW

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Abstract

Pediatric endogenous endophthalmitis is a rare disease that can cause serious ophthalmic damage. We report and discuss the diagnostic aspects and the clinical outcome of pediatric endogenous endophthalmitisdue to bacterial meningitis in a immunocompetent 3 year old infant with a three days history of, photophobia, purulent discharge, redness, corneal edema, hypopyon, poor red reflex and nausea.His parents brought him to our emergency department. He was diagnosed as having bilateral endogenous endophthalmitis associated with bacterial meningitis.Intravenous broad-spectrum antibiotic therapy was initiated with Cefotaxime 200 mg / kg per day in 4 slow IV injections and vancomycin15mg/kg per day in 4 IV injections. Intravitreal antibiotic (vancomycin and ceftazidime) injections were performed in both eyes. Two weeks post presentation, the best-corrected visual acuity in both eyes improved to 0.4, and inflammation of the anterior chamber and vitreous cavity was decreased. We recommend that when endogenous endophthalmitis is suspected along with meningitis, or if it is known to be present, Intravitreal and intravenous antibiotics should be promptly administered to preserve vision.

Introduction:-

Endogenous endophthalmitis is a rare, and frequently devastating, ophthalmic disease without exogenous origin. Endogenous endophthalmitis frequently occurs in patients who are immunocompromised, these categories account for over 90% of cases.[1,2] Endogenous endophthalmitis in immunocompetent patients is rare, and endophthalmitis as the initial presentation of bacterial meningitis is unusual.[3]. Here we describe a case of pediatric bilateral endogenous endophthalmitis as the initial presentation of bacterial meningitis in immunocompetent, non-diabetic 3-year-old patient.

Case Report:

A 3-year-old boy was broughtto our ophthalmic emergency department due to a three days history of visual disturbance, photophobia, purulent discharge, redness. He also complained of a headache and nausea. The child had previously been healthy, was using no medication or other pharmaceutical substances, and had no ocular history of disease, trauma, or prior surgeries.

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Upon examination his body temperature (39,6℃), heart rate (96 beats/minute), and respiration rate (30/minute) were measured. The neurologic exam revealed nuchal rigidity, and laboratory studies demonstrated leukocytosis (white blood cell count: 17,730/mm3 and CRP count 259 mg/L).

On ophthalmic examination, the best-corrected visual acuity (BCVA) was hand motion in both eyes. Intraocular pressure (IOP) was 13 mmHg in both eyes. Slit-lamp examination disclosed marked conjunctival hyperemia, corneal haziness, and small hypopyon with iridocrystalline synechia in both eyes **[Figure1.A** and **B]**. Fundoscopic examination demonstrated grade IV vitreous opacity in both eyes**[Figure 1.C** and **D]**.

Cerebrospinal fluid (CSF), blood, and urine samples were obtained for culture. Lumbar puncture revealed a cloudy CSF with 10500 WBC (89% neutrophils), 0,48 g/L glucose (0,72 g/L serum glucose), 0,46 g/L protein and 2,27g/l albumin. Cerebral computed tomography was normal **[Figure 2]**. Ocular ultrasound demonstrated vitreous debris in both eyes **[Figure 3].**

Under the assumption of bacterial meningitis with bilateral endogenous endophthalmitis, immediate intraocular stains and cultures (anterior chamber aqueous humor 0.3 mL) and intravitreal antibiotic injection (vancomycin 1mg/0.1 mL and ceftazidime 2mg/0.1 mL) were performed. Intravenous antibiotic therapy (Cefotaxime 200 mg / kg per day in 4 slow IV injections and vancomycin 15mg/kg per day in 4 IV injections) was initiated. Gram stains of intraocular samples demonstrated no microorganisms, and urine, blood, cerebrospinal fluid, and eye cultures were negative.

One week following the intravitreal antibiotic injection, the intraocular pressure was 10 mmHg in both eyes. Inflammation of the anterior chamber, conjunctival injection, and chemosis was improved. Vitreous opacities had improved in both eyes. The intravenous and topical therapies (ciprofloxacin eye drops hourly and 1% atropine eye drops twice a day in both eyes) were stopped after seven days.

Ten days after treatment, IOP was 10 mmHg in both eyes, and the BCVA was 0.4 in the right eye and 0.3 in the left eye. Inflammation of the anterior chamber and vitreous was reduced. Two weeks after initiation of therapy, the BCVA was 0.4 in both eyes. The anterior chambers were clear, and mild vitreous opacities were present in both eyesThevitrous debris and membranous debris were not found in the ocular ultrasound **[Figure 4]**.

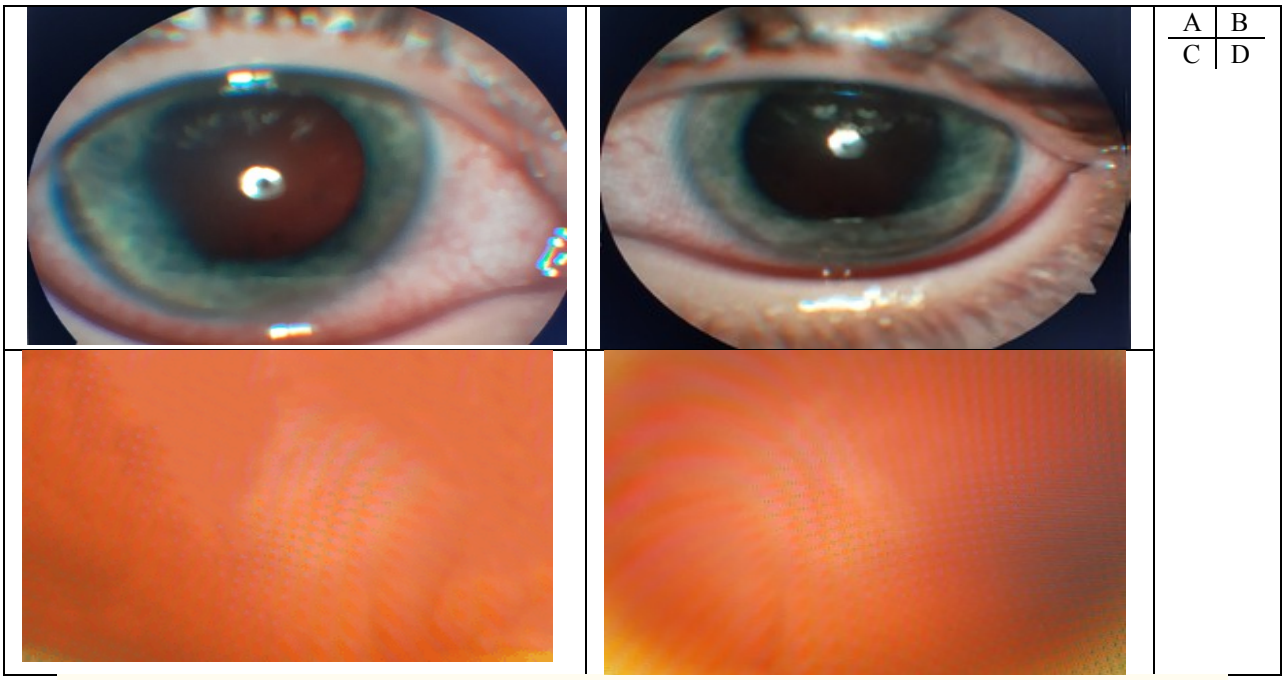

Figure 1:- (A, B) Slit lamp photography revealed marked conjunctival hyperemia, corneal haziness, and mild hypopyon with iridocrystalline synechia of both eyes. (**C and D**) Fundoscopic examination demonstrating grade IV vitreous haze in both eyes.

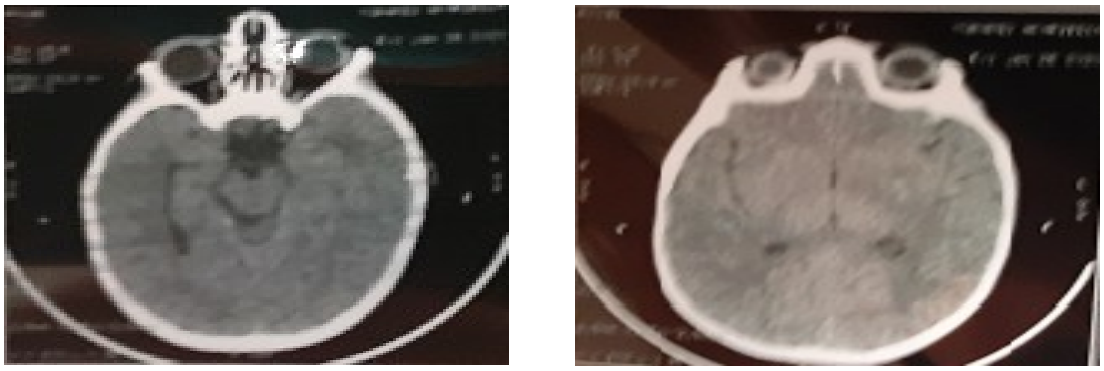

Figure 2:- cerebral computed tomography was normal

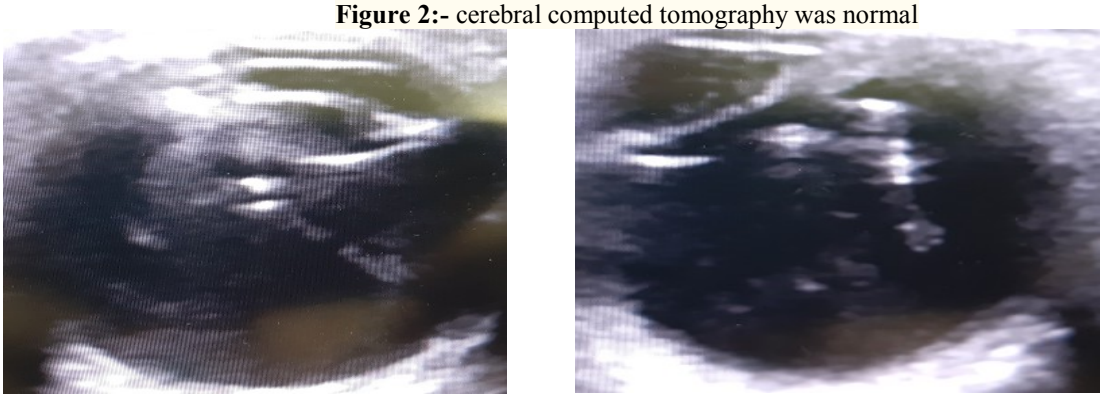

Figure 3:- Ocular ultrasound demonstrated vitreous debris in both eyes

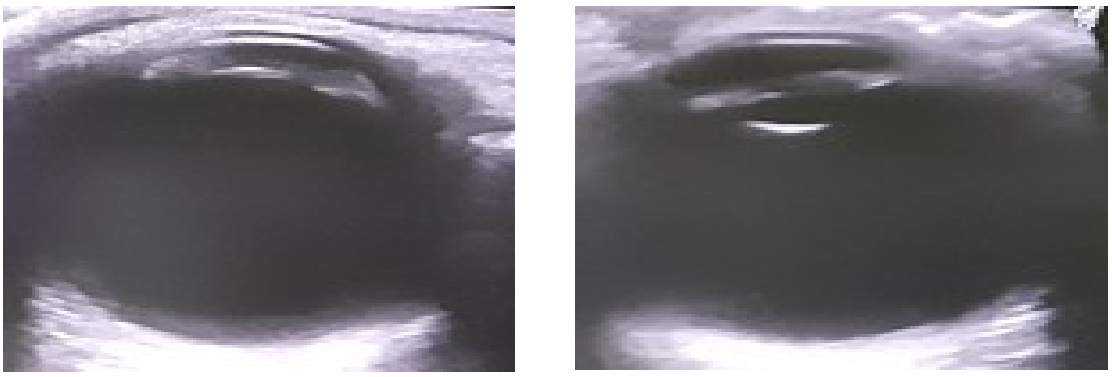

Figure 4:- ocular ultrasound 2 weeks later did not found any debris.

Discussion:-

Endogenous endophthalmitis constitutes a potentially devastating intraocular inflammation, caused by the migration of the pathogen from a distant primary site of infection to the eye where it crosses the blood-ocular barrier. It can occur at any age, ranging from 1 week to 85 years.Bilateral involvement is seen in 14 to 25% of patients and reports have shown a higher incidence of involvement of right eye versus left eye [4] in patients with unilateral disease. Before the widespread use of antibiotics the incidence of endophthalmitis secondary to bacterial meningitis was significant [5], though it has become very rare today.

The majority of patients with endogenous endophthalmitis suffer from an underlying disease. Factors predisposing one to endogenous endophthalmitis are intravenous drug use, immunosuppression, and prolonged intensive care.[6]. Cohen et al.[7] reported that the most common cause of endogenous endophthalmitis was urogenital infection. Greenwald et al.[1] reported that the pathogen was detected 71% of the time in anterior chamber aqueous sampling and 60% of the time at vitreous sampling. However, there were many cases where neither the primary focus nor the specific pathogen was detected. In the current case, we did not identify a specific pathogen, but, the diagnosis of

bacterial meningitis was verified by examination of thecerebrospinal fluid obtained by spinal tap, and the diagnosis of endogenous endophthalmitis was based on the clinical findings and ophthalmic examination. The most common gram positive organisms are group B streptococci, staphylococcus aureus, streptococcus pneumoniae and listeria monocytogenes. The most common gram negative organisms include Klebsiella spp., Escherichia coli, Pseudomonas aeruginosa and Neisseria meningitidis. [8].

visual outcomes following endogenous endophthalmitis have been disappointing because of the aggressive pathogens typically involved with this condition and because of compromised host immunity and delay in diagnosis especially in children. The visual acuity at the time of diagnosis, the causative agent and the degree of vitreous opacity are the main prognostic factors for the outcome.

Prompt administration of intravenous an intravitreal antibiotic therapy is the cornerstone in the acute management of endogenous endophthalmitis [9].

Conclusion:-

The rarity of endogenous endophthalmitis secondary to bacterial meningitis should not diminish our degree of awareness for this condition in immunocompetent patients. the early recognition and immediate treatments by intravitreal injections of antibiotics combined with intravenous antibiotics are essential to preserve vision in cases of endogenous endophthalmitis with bacterial meningitis.

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