Panophthalmitis in the acquired immune deficiency syndrome (AIDS) patient presenting as an acute glaucoma: a case report.

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The acquired immune deficiency syndrome (AIDS) is caused by the human immune deficiency virus (HIV). The ophthalmological manifestations are of special interest. The reported ophthalmic manifestations of AIDS included cotton wool spots, conjunctival Kaposi’s sarcoma, retinal periphlebitis and CMV retinitis. One of our patients had an acute unilateral glaucoma secondary to endogenous endophthalmitis and subsequently progressed to panophthalmitis. His HIV antibody was positive. Unfortunately, no proven causative organism was detected in our patient. The patient was treated with antiglaucoma drugs and parenteral antibiotics. The infection was controlled and subsided but the eye became atrophic. The patient was lost to follow up. The ocular infection in association with secondary glaucoma in AIDS patients were reviewed.

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ในปัจจุบัน กลุ่มอาการกลุ่มคู่ที่บกพร่องทางการยืดหยุ่นได้รับความสนใจอย่างมาก ทั้งในงานแพทย์และประชาชน ทั่วโลก อาการขาดแคลนอาหารพิษทำให้เกิด และผู้ป่วยอาการหนัก คนไข้กลุ่มอาการน้ำมันบางส่วน ผู้ป่วยได้รับการรักษา ซึ่งมีความยากลำบากของการรักษาด้วยทีมทีมกีฬาทีมกีฬา สามารถทำความเข้าใจได้ ดูตัวอย่างการรักษา ผู้ป่วยได้รับการรักษาที่มีการปฏิรูปความรักษาที่มีการปฏิบัติชีวิตอย่างดี แต่การควบคุมอาการที่ดี และการป้องกันการเกิดอาการที่ดี สามารถควบคุมการเพิ่มขึ้นและทำให้ง่ายต่อการรักษาด้วย
The acquired immune deficiency syndrome (AIDS) caused by the human immuno deficiency virus (HIV), is a severe disorder of the immune system affecting previously healthy individuals of age below 60 years. Almost all patients have evidences of severe defects of immunoregulation, usually associated with the T-cell component of the immune system. Life-threatening opportunistic infections or Kaposi’s sarcoma, or both, occur in persons with no underlying immunosuppressive therapy. At least eight groups have been found to be at special risk of acquiring AIDS(1).

The illness effects on the body are widespread, of special interest are the ophthalmological manifestations. The actual case fatality rate approaches 100%. No effective treatment for the illness is currently available.

The reported ophthalmoscopy manifestations of AIDS included cotton wool spots, conjunctival Kaposi’s sarcoma, retinal periphlebitis and CMV retinitis.

One of our patients had an acute unilateral glaucoma secondary to endogenous endophthalmitis and subsequently progressed to panophthalmitis.

A case report

A 30-year-old Thai man came in with a complaint of one day history of severe ocular pain, nausea, vomiting and inability to see the light in the left eye. His ocular past history was unremarkable.

He had been an intravenous drug (heroin) abuser for more than 10 years and had a history of sharing the needles with his partners. He denied homosexuality, transfusions, or known exposure to patients with AIDS.

The physical examination showed a cachetic man in no acute distress with normal vital signs.

The initial visual acuity was: R.E.: 20/40 and L.E.: no light perception. Intraocular pressure by applanation tonometry was R.E.: 18 mm.Hg. and L.E.: 50 mm.Hg. Slit-lamp examination of the left eye disclosed diffuse microcystic oedema of the cornea with marked ciliary injection, blepharospasm, lacrimation and steamy cornea. The anterior chamber was shallow peripherally, deep centrally with mild cell and flare. The pupil was 2 mm. in diameter and nonreactive. Iris bombe configuration was not present. There were no posterior synechiae, hypopyon or conjunctival discharge. The right eye was unremarkable except for a reversed Marcus Gunn pupil. The corneal haziness precluded gonioscopy and ophthalmoscope examination of the posterior pole.

An immediate standard B-scan ultrasonography was undertaken and showed moderate vitreous opacity, diffuse choroidal and scleral thickening of the left eye and unremarkable finding on the right eye.

The diagnosis was endogenous endophthalmitis with forward displacement of the lens-iris diaphragm causing secondary glaucoma.

The patient was treated with oral glycerine, oral dexamethasone, topical atropine, timolol and intravenous penicillin, cloxacillin and gentamicin.

12 hours later, the patient had acute left periorbital oedema, proptosis, a markedly chemotic left eye and complete ophthalmoplegia. (Fig. 1,2) Computerized tomography scan showed marked proptosis, soft tissue swelling of the lid, diffuse thickening of the sclera, increased density of vitreous and irregular enhancement of all orbital structures on the left side. The sinuses were normal. Despite aggressive intravenous antibiotic therapy a ring-shaped peripheral corneal abscess in association with a central abscess developed. The result of the haemoculture and conjunctival culture revealed no bacterial growth. Antibody to HIV was positive.

Figure 1,2 The patient had acute left periorbital edema, proptosis, a markedly chemotic left eye and complete ophthalmoplegia.
After five days of the above therapy the proptosis and chemosis decreased (Fig. 3.4). Evisceration was refused. The left eye became atrophic, the patient was lost to follow up.

**Discussion**

Ocular disease is a frequent feature of AIDS. A variety of ocular disorders in association with this syndrome was described\(^{(1,2)}\). Among the prominent ocular abnormalities seen have been cotton wool spots and cytomegalovirus retinitis\(^{(3-6)}\).

Grossniklaus et al\(^{(9)}\) had reported two young male intravenous drug abusers who were treated for acute glaucoma in the left eye and who proved to have Bacillus cereus panophthalmitis within 24 hours. The recent article by Ullman et al\(^{(10)}\) reported an AIDS related ocular manifestation in a drug abuser, Bacillus cereus panophthalmitis.

Our patient had a combination in some parts of the reported cases., A drug abuser who had AIDS, presented with acute glaucoma secondary to endophthalmitis in the left eye and progressed to panophthalmitis in 24 hours.

**Summary**

A 30-year-old Thai male intravenous drug abuser, with positive antibody to HIV, presented with an acute glaucoma secondary to endogenous endophthalmitis in his left eye and rapidly deteriorated into panophthalmitis. No definite causative organism was detected. The infection and inflammation could be controlled with medications. However the eye eventually developed phthisical eye and the patient was lost to follow up.

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**References**


