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REVIEW ARTICLE

GLAUCOMATOCYCLITIC CRISIS REVIEW

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ABSTRACT

Glaucomatocyclitic crisis (GCC) was first reported by Posner and Schlossman in 1948 as unilateral ocular hypertension associated with mild anterior segment inflammation (uveitis), and few clinical symptoms. The exact etiology is not clear although there are several theories proposed, ranging from autoimmune to infectious. It is a self-limited condition with recurrent episodes, and during intervals between attacks the patient is asymptomatic. Treatment management is based on controlling the intraocular pressure and decreasing inflammation. Glaucomatocyclitic crisis (GCC), was described as a rare, recurrent and typically unilateral inflammatory ocular hypertensive disease.1 It generally affects one eye at a time, and its recurrence usually afflicts the same eye. Bilateral and simultaneous involvement is very uncommon. The individual attacks may last from a few hours to a few weeks, but rarely persist over two weeks. Episodes may occur with varying frequency and without any apparent cause. It can affect adults of all ages (reports range from 23 to 67 years), especially between the third and sixth decade of life

Keywords: PosnerSchlossman, glaucomatocyclitic crisis, intraocular pressure, secondary glaucoma,

INTRODUCTION

Acutely increased intraocular pressure (IOP) and nongranulomatous anterior chamber inflammation are the hallmarks of Posner-Schlossman syndrome (PSS), also, called glaucomatocyclitic crisis. Most frequently, secondary inflammatory glaucoma is used to describe it. The condition is characterized by reduced vision, high intraocular pressure, open anterior chamber angles, normal appearance of the optic nerve, and normal visual fields, despite the unclear origin. Upon physical examination, PSS typically exhibits mild pain or discomfort in the eyes as well as unilateral impaired vision. The patient may occasionally experience no discomfort at all. A person who is impacted could also report seeing halos rather than blurry vision. But both are linked to corneal edema brought on by a high IOP. [1].

Etiology

Many etiologies, such as autoimmune and allergy (as suggested by Theodore and Kraupa, who defined this entity before Posner and Schlossman), have been put forth as causes of PSS, while the precise etiology is still up for debate. Skolic, Raitta, and Vannas suggested vascular and developmental anomalies as the disease's etiology, while Posner and Schlossman suggested autonomic dysregulation. [2].

Herpes simplex virus (HSV), varicella-zoster virus (VZV), and cytomegalovirus (CMV) are implicated in the more plausible infectious explanation of PSS pathogenesis. Helicobacter pylori is another potential culprit. The findings of certain studies about the effective treatment of PSS with antiviral drugs that target CMV provide the strongest evidence for a causal relationship between PSS and CMV. CMV was first identified as the infectious agent by Bloch-Michel et al., and this conclusion has been supported by additional research. The polymerase chain reaction (PCR)

used to identify CMV DNA and antibodies in the aqueous humor serves as proof for this. [3] .

Pathophysiology

PSS's pathophysiology is still unknown. According to reports, individuals with PSS had considerably lower levels of endothelium-dependent flow-mediated vasodilation in comparison to controls, which has led some authors to propose a putative aberrant vascular mechanism. One established risk factor for both normal tension glaucoma and primary open-angle glaucoma (POAG) is vascular endothelium cell dysfunction [4].

It's possible that inflammation in the anterior segment, specifically in the trabecular meshwork, is impairing aqueous outflow, which would explain the patients' occasional elevated IOP during acute phases. This proinflammatory process, which has been shown to be greater in aqueous humor during acute stages of elevated IOP in individuals with PSS, may be mediated by prostaglandins, most likely prostaglandin E. [5].

Studies have reported altered cytokine profiles in the aqueous of patients with PSS, which include higher levels of interleukin (IL)-6, IL-8, monocyte chemoattractant protein-1 (MCP-1), tumor necrosis factor (TNF), and vascular endothelium growth factors (VEGF). Elevated IOP in acute phases can give rise to temporarily reduced blood flow to the optic nerve head and retina, which tends to return to normal without leaving permanent damage in most cases.[6]

PSS seems to be in the range of acute uveitic disorders, and the trabecular meshwork may be involved. Possible causes of increased IOP in cases of anterior chamber CMV infection include: Trabecular outflow blockage by persistent inflammatory cells and pigments; trabeculitis, which results in an edematous and dense trabecular meshwork. Eventually, secondary angle closure evolves from peripheral anterior synechiae (PAS)[7]

It's unclear where the first infection occurred. The iris, trabecular meshwork, and corneal endothelial cells are among the potential sites. The pathogenesis of PSS may also be influenced by genetics, vascular endothelial dysfunction, and the cytokine composition of the aqueous humor. In addition, PSS and human leukocyte antigen (HLA) polymorphism are linked in the Chinese population. PSS has revealed polymorphism of both classical and nonclassical HLA classes. [8, 9].

Clinical Presentation

During active bouts of GCC, patients usually report with symptoms varying from mild ocular or orbital irritation to colored halos or blurred vision due to corneal edema brought on by high IOP. Severe corneal edema and discomfort may cause a substantial reduction in visual acuity in individuals with very high IOP. Significantly high intraocular pressure (IOP), open anterior chamber angles as revealed by gonioscopy, mild cyclitis manifesting as infrequent aqueous inflammatory cells, and a few tiny keratic precipitates are common clinical findings. In rare circumstances, IOP may rise to 70 mm Hg or more. Unless IOP is extremely high, conjunctival hyperemia is typically absent; in such case, moderate congestion may be observed. Posterior synechiae are usually absent due to the mild anterior chamber inflammatory response. Peripheral anterior synechiae are likewise lacking in this regard. When tissue ischemia and sustained IOP increase cause either diffuse or sectoral iris atrophy, the affected eye may seem light-colored, a condition known as heterochromia.[10].

Generally, optic discs are normal, and no visual abnormalities are observed, particularly during the early stages of GCC. However, typical glaucomatous damage might also result from protracted or recurrent episodes. Furthermore, two cases of GCC attack-related non-arteritic anterior ischemic optic neuropathy (NAAION) have been reported. One theory suggested that a "disc at risk" (i.e., crowded disc and minimal physiological cup) and risk factors like hypertension and diabetes, in addition to GCC-induced elevated IOP, contributed to a decreased ocular disc perfusion, which in turn caused vision loss and optic nerve atrophy.[11].

Evaluation

The evaluation of viral DNA using PCR analysis of aqueous humor tap is a crucial step in the characterization of PSS. CMV, HSV, and VZV are the common viruses that were evaluated [12]. Additionally, serum titers in a lab for the same viruses can be examined. Planning a course of treatment may depend significantly on the virus's presence being confirmed[13].

To evaluate a glaucomatous pattern of visual field loss suggestive of optic nerve damage, central visual field testing (Humphrey visual field, 24-2 or 30-2) is crucial. Stereoscopic optic disc photography is also useful for monitoring and diagnosing glaucoma. Following an acute episode

of sharply increased IOP, the discovery of increasing optic disc cupping and disc bleeding may indicate irreversible damage to the optic disc. [12].

During an acute episode, segmental iris ischemia, vascular congestion, and leakage may be seen on iris angiography. Optic nerve scanning laser Doppler flowmetry also shows reduced optic nerve perfusion during an acute assault. The optic nerve's structure, the retinal nerve fiber layer, and the density of peripapillary vessels can all be learned about through the use of optical coherence tomography (OCT) and OCT angiography (OCTA) of the optic disc. A decline in these metrics could indicate long-term glaucoma damage. [14] .

An uneven and highly reflecting endothelial cell layer is seen in the anterior segment OCT of the cornea in eyes with PSS linked to endothelins[15]. Furthermore, confocal scanning microscopy of the corneal endothelium layer reveals massive endothelial cells with low reflection haloes surrounding high reflection nuclei, resembling an owl's eye. It is determined that these "owl eye" cells are corneal endothelial cells infected with CMV that have an intranuclear inclusion body. [16].

Differential Diagnosis

Understanding the differential diagnosis for PSS is crucial since they impact the course of treatment and outlook. Among the most widely recognized differential diagnosis are common glaucomas:[17]
Both acute and long-term ACG
ocular hypertension
POAG [18]
Glaucoma in the eyes
Heterochromia Fuchs iridocyclitis
Iridocyclitis herpes [19]
Additional viral uveitis [19]

Distinguishing PSS from other frequent causes of elevated IOP requires effort. Due to its acute character, unilateral nature, significant increase in intraocular pressure, and presence of ocular inflammation, ACG is ranked highly on the list. Peripheral iridectomy was done in accordance with a case report that involved misdiagnosing PSS as ACG. Narrow or closed angles, a more intensely painful red eye, a fixed dilated pupil, nausea and vomiting, and PAS, on the other hand, distinguish ACG from PSS. The presence of PAS will be a differentiating factor in chronic ACG.[20]

POAG patients are typically older, may have a known family history of the disorder, and do not exhibit acute intraocular inflammatory symptoms. IOP peaks may occur in cases of pigmentary glaucoma and pseudoexfoliation glaucoma; nevertheless, both conditions present clinically with important diagnostic indicators and symptoms. Typically, pigmentary glaucoma manifests as iris transillumination abnormalities, trabecular meshwork pigmentation, and corneal endothelium pigment deposits (Krukenberg spindle). In addition to iris pigment loss that might result in iris transillumination deficiencies, white, flaky material on the lens anterior surface and at the iris pupillary border are typically present in cases of pseudoexfoliation glaucoma.[21]

A crucial class of illnesses related to PSS are the uveitic glaucomas. The intraocular inflammation in uveitic glaucoma is frequently more fulminant. Iris heterochromia, which appears late in the disease, fine aberrant anterior chamber angle vessels, fine diffuse keratic precipitates, and posterior subcapsular cataracts are characteristics that distinguish Fuchs heterochromic iridocyclitis from PSS. Viral infections, including as herpetic iridocyclitis caused by HSV and VZV, can be the cause of uveitic glaucoma. A more severe anterior chamber reaction, less noticeably raised intraocular pressure (IOP) than in PSS, and sectorial or diffuse iris atrophy are characteristics that can be used to distinguish uveitic glaucoma from PSS.[22].

In addition to vesicular rash and dendritic ulcers, sectorial iris atrophy is suggestive of HSV or VZV. Other potential causes of ocular inflammation, such as HLA B27 uveitis, sarcoidosis, HSV, Vogt Koyanagi Harada (VKH) illness, and human T lymphotropic virus type 1, should be taken into consideration in the clinical situation of high IOP and uveitis. Suspicion of PSS should be raised in the event of a significantly increased IOP that is out of proportion to moderate iridocyclitis, lack of PAS, and posterior synechiae [23].

Aqueous humor sampling and qualitative PCR analysis may be required if the diagnosis is unclear. A patient may have more than one virus. Concurrent cases of HSV-positive keratouveitis in one eye and CMV-positive PSS in the other have been reported.[24].

Keratitis with corneal stromal opacities and edema leading to scarring, neovascularization, and hypoaesthesia are additional distinguishing

indicators of HSV. Maculopapular rash and vesicles, which can appear at the nose's tip, side, or root as well as occasionally on the forehead, are the symptoms of VZV. Rashes at the tip of the nose are referred to as "Hutchinson's sign" and are a reliable indicator of VZV intraocular involvement [25].

Treatment / Management

Reducing intraocular inflammation, or IOP, is the goal of treating PSS. Antiviral therapy has been utilized recently to target the virus that is causing the infection. Medical and surgical procedures can be used to accomplish PSS treatment goals..

Medical Therapy

Often employed in the treatment of glaucoma, topical beta-blockers, alpha agonists, and carbonic anhydrase inhibitors are the first-line interventions for lowering intraocular pressure (IOP) during acute attacks. A satisfactory response can be obtained when using these groups of topical treatments, or a combination of therapies. Systemic drugs, including oral carbonic anhydrase inhibitors, may be necessary in certain situations to manage the sudden increase in intraocular pressure. Topical prostaglandin analogs should be avoided since they may worsen inflammation. Topical ocular hypotensives may be stopped in between attacks if the IOP returns to normal, particularly if the patient has a minimal risk of developing glaucomatous optic neuropathy (GON) or other eye problems. It is not advisable to use topical pilocarpine as it may worsen trabeculitis.[26]

Topical steroids like prednisolone or dexamethasone can be used to treat intraocular inflammation, which is typically moderate. To effectively manage low-level intraocular inflammation, topical fluorometholone has also been employed. If necessary, cortisone topical therapy can be used four times a day; after that, it can be rapidly discontinued. Nonsteroidal anti-inflammatory medications (NSAIDs), both oral and topical, can reduce inflammation. Since an increase in aqueous prostaglandin levels has been linked to attacks, taking NSAIDs can prevent possible steroid-induced glaucoma and also have a desired antiprostaglandin effect. [27]

Daily patient follow-ups are necessary to track IOP during an acute episode and make sure it is lowered to a safe level. Once steroid and ocular hypotensive are weaned, fewer visits (weekly or monthly, as needed) can be implemented. Give instructions to see an ophthalmologist as soon as an attack is suspected. In the absence of clearly severe corneal edema, baseline optic nerve measures (as previously mentioned) must be documented when treatment is started. When starting topical ocular hypotensives and steroids, it is important to monitor the patient's response and gradually reduce the dosage. An aqueous tap should be used to check for anterior chamber CMV infection if the patient does not show any signs of improvement after maximum medication, or if there are several recurrences. If CMV DNA is detected, treatment with an antiviral drug should be taken into consideration [28].

Previous studies have utilized oral and topical ganciclovir in combination. In certain situations, the application of topical ganciclovir has led to the resolution of inflammation, allowing for the discontinuation of topical steroids and a decrease in pressure-lowering drops. However, the recurrence of subsequent episodes was not entirely prevented by this treatment. [29].

While the failure rate of oral valganiclovir was found to be lower than that of ganciclovir gel, the recurrence rate was much greater, at 80% as opposed to 57%. For at least two weeks, 900 mg of oral valganciclovir were given twice a day as part of an oral loading dose. After that, 450 mg of valganciclovir were given twice a day as part of maintenance therapy. At this dosage, a positive treatment outcome was observed in this trial. However, once valganciclovir was withdrawn, recurrence happened. Recurrence after stopping valganciclovir has been noted to be quite common. The increased recurrence rate observed in valganciclovir can be explained by its virostatic rather than virucidal properties. Furthermore, the immunological privilege of the eye keeps the CMV infection from being completely eradicated.[30]

It has also been investigated to administer ganciclovir 2.0 mg intravitreally in 0.05 cc with or without oral antiviral medication. Although it has been demonstrated that intravitreal valganciclovir at a dose of 2 mg/0.05 mL can lower aqueous CMV to undetectable levels, the necessity of intravitreal injections in cases of anterior chamber infection has been questioned. Moreover, oral valganciclovir reduces aqueous CMV in a comparable way, while leukopenia is a possible side effect. Therefore, in patients taking oral valganciclovir, the white cell count should be kept under observation.[31]

Surgical Therapy

In patients with PSS, surgery is performed to reduce IOP if medical treatment is ineffective. The treatment of ocular endothelial insufficiency may also need surgery. Trabeculectomy is one surgical method that has been utilized to control IOP. Nevertheless, there is a higher risk of conjunctival scarring and the ensuing failure of the conjunctival filtering bleb as a result of preoperative conjunctival inflammation, which mobilizes fibroblasts and macrophages..[32]

Using an antimetabolite, like mitomycin C (MMC), to complement trabeculectomy is a useful method of managing intracranial pressure. MMC trabeculectomy improves PSS, decreases IOP spikes, and reduces inflammation. This improvement in inflammation following surgery may be explained by inflammatory cells in the aqueous chamber draining out through the filtering bleb, which lowers anterior chamber activity and trabeculitis.[7]

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A comparable problem in PSS can be treated with penetrating keratoplasty (PK) or Descemet's stripping automated endothelial keratoplasty (DSAEK), just like in postuveitic corneal endothelial insufficiency. Furthermore, following corneal surgery in PSS eyes, the rate of graft failure will resemble that of postuveitic cornea grafts (50%) compared to other forms of corneal endothelial insufficiency (13%) in this case [34].

Complications

Although PSS was once thought to be a benign condition, it can cause serious ocular problems. A consequence of this is the development of GON. Topical hypotensives should be used prophylactically to individuals who have a high cup-to-disc ratio, retinal nerve fiber loss (RNFL) damage, or localized neuroretinal rim thinning who may have later optic nerve damage. Filtration surgery may be necessary if the IOP is still uncontrolled and there is evidence of increasing injury to the optic nerve.[7]

In addition, optic atrophy and nonarteritic anterior ischemic optic neuropathy (NAION) can be brought on by PSS. Patients with PSS who have risk factors for ischemic optic atrophy and NAION (such as a small cup-to-disc ratio) may additionally receive topical hypotensives as a preventive measure [35].

Individuals who have PSS are prone to ocular issues. Evidence of a reduced endothelial cell count has been found in eyes with CMV-associated PSS.[7] Corneal endothelial insufficiency, characterized by a significant loss of endothelial cells, arises during recurrent sickness. Steady intraocular pressure also raises the possibility of corneal endothelial cell death and induces corneal epithelial edema. Endotheliitis is another observation in PSS associated with CMV.[36] Endotheliitis is a clinical sign of CMV infection in the anterior chamber in an immunocompetent patient. Two additional negative effects of PSS include cataracts and iris atrophy [37].

CONCLUSION

Closer observation of this patient group is necessary since some PSS patients experience acute attacks more frequently than others. For these patients, it may be necessary to gradually reduce the amount of eye drops, and clear instructions on when to make follow-up appointments should be communicated. In the event that a PSS patient has an attack before their next appointment, providers should make sure they can see them in between. It is equally important to take a planned strategy with evidence-based tactics to maximize treatment plans and reduce side effects. When pharmacists and ophthalmic healthcare providers collaborate to give patients with PSS ongoing assistance and education, the patient outcomes are at their best.

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