Ultrasound biomicroscopic images of the anterior chamber angle of a patient with posterior polymorphous dystrophy

EDITOR,—Posterior polymorphous dystrophy (PPD) is a hereditary corneal dystrophy that is typically asymptomatic and non-progressive. It rarely results in severe visual dysfunction. Posterior polymorphous dystrophy (PPD) is a hereditary corneal dystrophy that is typically asymptomatic and non-progressive. It rarely results in severe visual dysfunction. It is described in AIDS patients in 1982. The retinal findings comprise cotton wool spots similar in appearance to those found in diabetes mellitus and immune complex disorders. However, the aetiology of posterior polymorphous dystrophy is generally regarded as a feature seen in patients with laboratory evidence of significant immune deficiency. We report a patient in whom HIV retinopathy was noted during an acute seroconversion illness—a finding which has not been previously described.

CASE REPORT
A 39-year-old woman was examined for progressive loss of vision (30/200) in her left eye and increased foreign body sensation. Slit lamp examination of the left eye revealed diffuse corneal oedema and bullous keratopathy. We examined the anterior chamber angle of a PPD patient with corneal oedema and broad iridocorneal adhesion by using ultrasound biomicroscopy (UBM). The examination indicated a unique iridocorneal adhesion that could not be seen in gonioscopy.

COMMENT
Patients with PPD usually demonstrate normal vision, but endothelial decompensation and/or glaucoma can develop, resulting in visual loss. Intraocular pressure (IOP) elevation in PPD may be explained not only by synechial closure but also by abnormalities of neural crest cell differentiation or the basement membrane. Recording of UBM images on more cases of PPD is needed to clarify mechanism of IOP elevation in this disease.

CASE REPORT

A 44 year old heterosexual white woman presented to the regional infection unit with a 5 day history of myalgia, arthralgia, fever, anorexia, and watery diarrhoea. On examination a diffuse macular rash was noted, there was generalised lymphadenopathy and several cotton wool spots were noted on ophthalmoscopy of the right retina (Fig 1). Her diarrhoea persisted despite the use of enteropaths and she was found to have an inflammatory infiltrate on jejunal biopsy. Despite normal appearances on barium enema and multiple normal colonic biopsies she was thought likely to have mild inflammatory bowel disease and was treated with corticosteroids and mesalazine. Following discharge on this regimen her diarrhoea settled but she continued to lose weight. When readmitted 2 months later the cotton wool spots were again noted on examination. No other disease process liable to cause these was identified; her blood pressure was never higher than 140 mm Hg systolic/80 mm Hg diastolic, erythrocyte sedimentation rate was only 30 mm in the first hour, and autoantibody screen was negative—and in light of her ongoing weight loss, the patient was tested for HIV and found to be antibody positive. Retrospective analysis of stored serum from her first admission showed the presence of HIV antigen with undevelopable antibody, indicating that she was undergoing a seroconversion illness at that time. Her absolute CD4+ lymphocyte count on the second admission was 220 cells ×10⁹/l.

Her later clinical course following the diagnosis of HIV infection showed a rapid progression of the disease with a marked decline in CD4+ cell count and development of AIDS within 6 months of diagnosis (AIDS with the AIDS-defining illness being the AIDS defining illness). Her later clinical course following the diagnosis of HIV infection showed a rapid progression of the disease with a marked decline in CD4+ cell count and development of AIDS within 6 months of diagnosis (AIDS with the AIDS-defining illness being the AIDS defining illness). Her later clinical course following the diagnosis of HIV infection showed a rapid progression of the disease with a marked decline in CD4+ cell count and development of AIDS within 6 months of diagnosis (AIDS with the AIDS-defining illness being the AIDS defining illness).

In the patient described cotton wool spots were ultimately attributed to her HIV disease and had been present since her presentation during a seroconversion illness. This finding has not been previously reported. Although she had a significantly depleted CD4+ cell count when it was first measured (several months after seroconversion) no measurement of her lymphocyte subsets was made at the onset of her illness and the degree of immunodeficiency associated with the acute seroconversion is therefore unknown. It is recognised that patients can progress rapidly from seroconversion to profound immunodeficiency and AIDS* and that seroconversion itself can be associated with a marked fall in CD4+ cell count. The latter may have been relevant in our patient as lymphadenopathy noted on initial presentation reflected the severity of her seroconversion and the concomitant CD4+ lymphocyte depletion. Whether this finding of retinopathy during seroconversion is of any clinical value in aiding diagnosis or as a predictor of the subsequent clinical course is not clear, although it is interesting to note that progression to AIDS was rapid in this case. In the experience of the authors the finding proved helpful in stimulating consideration of the diagnosis in a patient with no obvious risk factors for HIV infection.

COMMENT

HIV retinopathy is a benign feature of HIV disease which is principally recognised in patients with symptomatic disease or significantly reduced CD4+ cell counts. The aetiology is poorly understood and has variously been suggested to be due to circulating immune complexes or to direct infection of the retina by the human immunodeficiency virus.

In the patient described cotton wool spots were ultimately attributed to her HIV disease and had been present since her presentation during a seroconversion illness. This finding has not been previously reported. Although she had a significantly depleted CD4+ cell count when it was first measured (several months after seroconversion) no measurement of her lymphocyte subsets was made at the onset of her illness and the degree of immunodeficiency associated with the acute seroconversion is therefore unknown. It is recognised that patients can progress rapidly from seroconversion to profound immunodeficiency and AIDS* and that seroconversion itself can be associated with a marked fall in CD4+ cell count. The latter may have been relevant in our patient as lymphadenopathy noted on initial presentation reflected the severity of her seroconversion and the concomitant CD4+ lymphocyte depletion. Whether this finding of retinopathy during seroconversion is of any clinical value in aiding diagnosis or as a predictor of the subsequent clinical course is not clear, although it is interesting to note that progression to AIDS was rapid in this case. In the experience of the authors the finding proved helpful in stimulating consideration of the diagnosis in a patient with no obvious risk factors for HIV infection.

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Deficiencies.

Regular review and screening for possible absorption and storage of essential vitamins.

Presence of ocular surface abnormalities, the correct treatment.

Resulting in an unnecessary delay in instituting therapy is demonstrated by this case. While the early ocular manifestations of hypovitaminosis A are readily reversible, the late changes cause permanent corneal damage and visual loss. In addition, there is an increased childhood morbidity and mortality associated with vitamin A deficiency which can be reduced by restoring vitamin A levels to normal.

Night blindness had been present for at least 3 years and the initially mild lid and conjunctival changes were misinterpreted as being secondary to allergy, possibly because the patient’s skin condition (itself the result of atopy). Although the clinical presentation was entirely consistent with vitamin A deficiency, the diagnosis was not initially considered because it is so infrequently encountered in developed countries, resulting in an unnecessary delay in instituting the correct treatment.

A history of previous bowel resection, in the presence of ocular surface abnormalities, should raise the possibility of inadequate absorption and storage of essential vitamins. Patients with short bowel syndrome need regular review and screening for possible deficiencies.

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Table 1 Investigation results

<table>
<thead>
<tr>
<th></th>
<th>Pretreatment</th>
<th>Post-treatment (2 weeks)</th>
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<tr>
<td>Haemoglobin</td>
<td>11.0 g/dl</td>
<td>12.6 g/dl</td>
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<tr>
<td>Vitamin A (25.8–48.7 µg/dl)</td>
<td>&lt;20 µg/dl</td>
<td>216 µg/dl</td>
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<tr>
<td>Vitamin E (1.15–35 mmol/l)</td>
<td>Undetectable</td>
<td>0.3 mmol/l</td>
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<td>25 Hydroxy vitamin D (40–195 mmol/l)</td>
<td>7 mmol/l</td>
<td>2.46 mmol/l</td>
</tr>
<tr>
<td>Calcium (2.12–2.65 mmol/l)</td>
<td>1.81 mmol/l</td>
<td>2.46 mmol/l</td>
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<tr>
<td>Albumin (3–50 g/l)</td>
<td>30 g/l</td>
<td>48 g/l</td>
</tr>
<tr>
<td>Prothrombin time (10–14 seconds)</td>
<td>32 seconds</td>
<td>Normal</td>
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Visual impairment due to bilateral corneal endothelial failure following simultaneous bilateral cataract surgery

VOROTON—Although present day cataract surgery has a high success rate, simultaneous bilateral cataract surgery is not routinely performed. The main cause for concern in patients undergoing simultaneous bilateral surgery is the possibility of visual impairment due to serious complications affecting both eyes. The potential problem that is most frequently highlighted in the literature is the risk of bilateral endophthalmitis. We report a case of bilateral poor vision following simultaneous bilateral phacoemulsification and intraocular lens implant due to secondary corneal endothelial failure. To our knowledge this has not been previously reported.

CASE REPORT

A 76 year old white woman was referred to our cornea clinic with complaint of poor vision. She had undergone an uncomplicated simultaneous bilateral phacoemulsification with posterior chamber intraocular lens implant in March 1995 at another hospital. A few months before her surgery the visual acuity had been noted to be 6/24 in either eye. She had bilateral cataracts and the corneas were reported as normal. Following the surgery her vision gradually deteriorated in both eyes over 6 months to 3/60 right and hand movements left. This was due to bilateral diffuse corneal oedema secondary to endothelial failure. She underwent a left penetrating keratoplasty in September 1995 and subsequently the same operation was performed in her right eye in August 1996. Unfortunately the right corneal graft failed in the postoperative period.

On presentation to us in September 1997 her vision was 1/60 right eye and 6/36 left. Examination revealed a right failed corneal graft with vascularisation in one quadrant and a clear corneal graft in the left eye (Fig 1). Intraocular lens implants were in situ. The fundus appeared grossly normal in the right eye. Early retinal pigment epithelial changes were noted at the left macula. She was offered a repeat right corneal graft with a guarded prognosis but she decided against it.

COMMENT

Previous studies of patients undergoing simultaneous, bilateral modern cataract surgery have reported no bilateral, vision threatening postoperative complications. Even so the possibility of rendering the patient temporally or permanently blind cannot be completely ruled out. In a recent consultation section on simultaneous bilateral cataract surgery the main cause of concern among surgeons was the potential problem of bilateral visual loss due to routine simultaneous bilateral cataract surgery. There was, however, no mention of bilateral secondary endothelial failure resulting in poor vision.

Secondary endothelial failure accounts for approximately 25% of patients requiring corneal grafts and they have a higher rate of graft failure and rejection. The visual prognosis is also poorer in this group of patients and it can take up to a year to reach an optimum level.

Therefore, patients requiring corneal grafts for bilateral secondary endothelial failure following simultaneous bilateral cataract surgery can potentially be rendered visually handicapped for a long time.

We are not aware of the reasons for our patient having simultaneous bilateral cataract surgery. Unfortunately, despite an apparently normal corneal examination she still developed bilateral secondary endothelial failure resulting in severe visual impairment for a long time.

This case therefore demonstrates that the possibility of bilateral visual loss due to secondary endothelial failure is another strong argument against routine simultaneous bilateral cataract surgery. We suggest that patients who are being offered this surgery should be made aware of the risks and consequences of secondary endothelial failure. Preoperatively, a meticulous examination of their corneal endothelium should be undertaken. If significant corneal endothelial pathology is noted, than only unilateral cataract surgery should be performed. The second eye should have the cataract surgery only after the first eye has been successfully rehabilitated.

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Figure 1 Shows right failed corneal graft with vascularisation in one quadrant and a clear corneal graft in the left eye.
Symptomatic acute raised IOP following haemodialysis in a patient with end stage renal failure

EDITOR.—We report a case of a 45 year old man with chronic renal failure presenting with symptomatic bilateral acute raised intraocular pressure (IOP) following haemodialysis. The pressures were successively reduced with a topical β blocker and following the commencement of regular topical treatment his symptoms were controlled with no further record of raised IOPs.

CASE REPORT

A 45 year old white man was referred to the eye casualty department by the renal physi- cian complaining of bilateral blurred vision and a dull frontal headache following haemodialysis. The blurred vision resolved spontaneously within 2 hours of onset but the headache persisted. The headaches had been recurrent following every haemodialysis which he had undergone and could last up to 10 hours. The blurred vision was a less consistent feature, only occurring occasionally. He had end stage renal failure due to glomerulonephritis and could last up to 10 hours. The headaches had been recurrently within 2 hours of onset but the headache may be due to a decrease in outflow facility and an osmotic influx of water into the eye because of hyperosmolality of intraocular fluids following dialysis. In all the studies, the raised IOP was of questionable clinical significance. All except one patient who had a history of narrow angle glaucoma were asymptomatic. To our knowledge this is the first case reported of symptomatic acute raised intraocular pressure following haemodialysis in a patient who had previously healthy eyes. Carbonic anhydrase inhibitor is relatively contraindicated in renal failure. Systemic carbonic anhydrase inhibitor was therefore unwise with levobunolol 0.5% eye drops alone. We treated the raised ocular pressure with a topical β blocker and following the commencement of regular topical treatment his symptoms were controlled with no further record of raised IOPs.

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COMMENT

Symptomatic raised IOP following haemodialysis is rarely diagnosed. Asymptomatic raised IOP following haemodialysis has been reported in the medical literature. Several studies have shown that raised IOP follows haemodialysis in a significant number of patients while others have failed to show this relation.2 4 The prevalence of this phenomenon among patients undergoing haemodialysis is not known and the pathophysiology involved is not certain. The elevation of IOP may be due to a decrease in outflow facility and an osmotic influx of water into the eye because of hyperosmolality of intraocular fluids following dialysis. In all the studies, the raised IOP was of questionable clinical significance. All except one patient who had a history of narrow angle glaucoma were asymptomatic. To our knowledge this is the first case reported of symptomatic acute raised intraocular pressure following haemodialysis in a patient who had previously healthy eyes. Carbonic anhydrase inhibitor is relatively contraindicated in this condition as it can precipitate severe metabolic acidosis. Regular topical β blocker can be used to control this condition.


Episcleral melanoma without conjunctival or uveal involvement

EDITOR.—Melanocytic lesions of the episclera include Axenfeld nerve loop, episcleral melanocytosis, ochronosis, conjunctival naevus, cellular blue naevus, melanocytoma, conjunctival melanoma with deep extension, extraocular extension of uveal melanoma, or metastatic melanoma.4 5 The occurrence of an episcleral melanoma without conjunctival, uveal, or skin involvement is extremely rare. We report an unusual case of malignant melanoma occurring as an isolated tumour on the episcleral surface.

CASE REPORT

A 36 year old healthy man developed a pigmented epibulbar lesion in the left eye over a 1 year period. There was no history of ocular trauma, cutaneous melanoma, or dysplastic naevus syndrome. Ocular examination revealed visual acuities of 6/6 in both eyes. In the left eye, there was an episcleral pigmented mass located 2 mm from the limbus at the 10 o’clock position, measuring 4.0 × 3.5 mm in size (Fig 1). The conjunctiva was freely mobile over the lesion. Anterior segment examination was otherwise normal with no sign of conjunctival naevus, primary acquired melanosis, or malignant melanoma. There was no evidence of an intraocular melanoma by funduscopy. On transillumination, blockage of light transmission by the epibulbar lesion was noted. The differential diagnosis included an

Figure 1 Anterior segment photograph of the left eye showing the epibulbar pigmented lesion (arrow).

Figure 2 Photomicrograph demonstrating the deep subconjunctival lesion containing epithelioid melanocytes with prominent nuclei, consistent with malignant melanoma (haematoxylin and eosin, original magnification ×150).
Intraocular metastasis of endodermal sinus tumour

Editor,—Ocular metastases were previously thought to be rare.1 However, after having been extensively studied in patients with cancers, the incidence of ocular metastatic tumours increased.1,4 Haematological malignancy—including leukaemia, lymphoma, and multiple myeloma—is the most common primary cancer whose ophthalmic and pulmonary carcinomas are regarded as the most frequent intraocular metastatic carcinomas.1,4 We report an intraocular metastatic endodermal sinus (yolk sac) tumour which, to our knowledge, has not yet been described.

CASE REPORT
A 45 year old man presented with blurred vision in the left eye. Funduscopic study suggested a posterior choroidal tumour causing retinal detachment. Additional physical examination disclosed a 10 cm mass in the anterior wall of the chest.

Computed tomography of the thorax exhibited a mass, 13 × 11 × 8 cm, in the anterior mediastinum. The lesion with solid and cystic components extended into the right lung and anterior wall of the chest. Left enucleation was subsequently performed.

Grossly, the left eyeball revealed a grey white mass, 2 × 1.5 × 1.5 cm, with cystic degeneration in the posterior choroid. It protruded into the vitreous chamber (Fig 1). Microscopically, the tumour was composed of glandular structures of various sizes lined by low columnar cells having large, oval, and basophilic nuclei with coarse granules of chromatin and inconspicuous nucleoli. Numerous eosinophilic hyaline globules were noted (Fig 2, upper). Schiller–Duval bodies, however, were not observed. Immunohistochemical stainings revealed strong cytoplasmic positivity to a fetoprotein (Fig 2, lower), 

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\text{α1-antitrypsin, epithelial membrane antigen, and cytokeratin. Stainings for S-100 protein, HMB-45, GFAP, NF, HCG, and CD30 were negative. The diagnosis was endodermal sinus tumour (EST). Subsequently, biopsy of the chest wall lesion consisted of a few pieces of grey tissue, 0.5–1 cm in greatest dimension, was obtained. Sections revealed round vacuolated tumour cells forming nests and glands. Immunohistochemical study showed the same result as previously described in the ocular lesion. Furthermore, a high serum α fetoprotein level of 6850 IU/ml was detected. The final diagnosis was primary anterior mediastinal EST showing prominent glandular differentiation with metastasis to the left eyeball.
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COMMENT
EST has several histological features such as reticular, polyvesicular-vitelline, hepatoid, endometrioid-like, intestinal, and mixed variants.6 Although Schiller–Duval body is the diagnostic hallmark of EST, it is not found in all cases.7 In our example, the diagnosis of EST was based on the morphology of the tumour cells, numerous hyaline globules, and identification of a fetoprotein in neoplastic cells as well as in serum. This pattern of EST with prominent glandular differentiation is similar to that originally described in the ovary by Cohen et al.8 A small sized ocular tumour compared with the mediastinal one as well as absence of any previous report on primary ocular EST makes us believe that the mediastinal EST is the primary cancer that metastasises to the left eyeball. Malignant melanoma should be in differential diagnosis because it has diverse histopathological patterns and is common intraocular neoplasm in the West. However, hyaline globules are not the feature of malignant melanoma and there is no immunohistochemical finding to support. Moreover, the uveal melanoma is extremely rare in Thailand.9

In summary, we present an intraocular metastatic EST in which we suggest to spread from the anterior mediastinal EST. Although metastatic cancer to the eye is currently believed to be the most common ocular malignancy,9 EST has not yet been recorded to metastasise intraocularly.

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**Fluorescein and indocyanine green angiography in arteritic anterior ischaemic optic neuropathy**

**EDITOR.**—Anterior ischaemic optic neuropathy (AION) is the most common cause of visual loss in giant cell arthritis (GCA). However, other presentations have been described including posterior ischaemic optic neuropathy, choroidal ischaemia, retinal artery occlusion, branch retinal artery occlusion, cilioretinal artery occlusion, and occult central retinal artery infarction. We present the first indocyanine green angiography (ICGA) findings in a case of GCA with simultaneous optic nerve and choroidal ischaemia.

**CASE REPORT**

A 65 year old woman was admitted because of bilateral blindness. She had had complete, painless visual loss in her right eye 72 hours before admission followed 48 hours later by visual loss in the left. One week earlier she had noted jaw claudication and neck pain. Ophthalmic examination revealed no light perception (NLP) with disc oedema in both eyes. Western-greyn erythrocyte sedimentation rate (ESR) was 76 mm in the first hour; brain magnetic resonance imaging was normal. She was given 50 mg/day oral prednisone, with no improvement. One week later she was referred to our institution for a neuro-ophthalmological evaluation. Visual acuities were still NLP with mid-dilated, unreactive pupils; funduscopic examination showed pale disc oedema in both eyes. ESR was 30 mm in the first hour. Fluorescein angiography (FA) (Fig 1) showed marked delay in optic nerve and choroidal filling; mild optic nerve leakage, and peripheral hyperfluorescent spots with RPE mottling were seen in the late angiographic phases in the left eye. ICGA (Fig 2) confirmed the severe ischaemia of the optic nerve especially on the temporal side and highlighted staining of several peripheral choroidal vessels. Temporal artery biopsy was positive for GCA. Prednisone was increased to 100 mg/day but there was no recovery of visual function at follow up.

**COMMENT**

The association of choroidal ischaemia and AION is particularly suggestive of GCA as indicated by Hayreh.1,2 Mack et al.1 and Siatkowski et al.3 performed FA in GCA and found significant delay of choroidal filling in comparison with either normal subjects or patients with non-articular AION. We report a case of optic nerve and simultaneous choroidal ischaemia in GCA. Choroidal hypoperfusion was more severe on the temporal side suggesting a distinct involvement of the lateral posterior ciliary arteries (PCAs); FA also highlighted areas of RPEatrophy and pigmentary migration in the peripheral retina. Similar abnormalities of the outer retina in GCA were attributed to arteritic involvement of the PCA supply to the choroid.4


This is the first ICGA study of choroidal circulation in GCA. ICGA clearly demonstrated the choroidal ischaemia but also showed staining of some peripheral vessels, probably related to an inflammatory infiltration of their wall not visible with ophthalmoscopy and FA. Even though no conclusions can be drawn from a single case, ICGA may be a valuable diagnostic tool for differentiating arteritic from non-arteritic AION and even an interesting way to monitor the disease.

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Intraocular and extraocular bleeding after intracameral injection of tissue plasminogen activator

EDITOR—During the early postoperative period after glaucoma filtration surgery the sclerostomy can be blocked by haemorrhage or fibrin clot.1 In these cases tissue plasminogen activator (tPA) can be injected into the anterior chamber after paracentesis or subconjunctivally. It works rapidly so that within 3 hours the effect is usually apparent. This report describes a patient who had massive ocular bleeding after intraocular injection of tPA.

CASE REPORT

A 76 year old white man with uncontrolled advanced primary open angle glaucoma in the left eye underwent trabeculectomy with mitomycin C. Past ocular history was relevant for trabeculectomy with 5-fluorouracil, 8 years earlier, and a combined mitomycin C trabeculectomy, phacoemulsification, and intraocular lens implantation 2 years before. Medical history regarding bleeding or coagulation disorders was negative, although tests to exclude abnormalities in the coagulation system were not done. The patient did not take coagulation inhibitors before or after surgery.

The surgery was uneventful. One day after surgery the intraocular pressure (IOP) was 10 mm Hg and there was a large superotemporal filtering bleb. One week later the IOP was 30 mm Hg, with a very vascularised low bleb and a deep anterior chamber. Laser suture lysis (two sutures) and digital ocular compression did not lower the IOP. An intracameral injection of 15 µg of tPA was done. The following day the patient had a large (40%) hyphaema and a dense subconjunctival haemorrhage extending to the eyelids and the orbital rim (Figs 1 and 2). A mild vitreous haemorrhage was also present. Vision was hand movements and IOP was 5 mm Hg. Ocular trauma had
not occurred. The blood resorbed over 3 weeks, and the function of the bleb remained satisfactory.

COMMENT

Recombinant tPA is a serine protease with clot specific fibrinolytic activity. tPA has been used successfully to lyse blood, fibrinous clots, and/or membranes after pars plana vitrectomy, cataract surgery, and glaucoma surgery. A dose of up to 25 µg of tPA is used for ophthalmic procedures. Hyphaema is the most frequent complication of intracameral tPA injection after glaucoma surgery (up to 36% of cases). Lundy et al suggested that a dose of 6–12.5 µg may be equally effective and reduces the risk of hyphaemias.

In this patient the bleeding source was probably intraocular, which extended to the subconjunctival space through the fistula (functioning after the tPA injection), and to the preseptal periorcular tissues because of the large volume of the haemorrhage. It is not known whether previous surgeries and/or ocular scarring might have contributed to the intensity of the bleeding. The use of mitomycin C and laser suture lysis were probably not related to this complication. The singular aspect of this case was the severity of the bleeding, and its extraocular extension.

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