Case report

Rare atypical presentation of phacolytic glaucoma as non-resolving microbial keratitis with endophthalmitis

Rinky Agarwal, Manasi Tripathi, Anuja Patil, Namrata Sharma

Ophthalmology, Dr Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi, India

Correspondence to

Dr Rinky Agarwal,
rinky.1990@gmail.com

Accepted 28 August 2019

SUMMARY

Phacolytic glaucoma (PLG) is a rare complication of hypermature senile cataract. Delayed presentation of PLG may make its diagnosis and management difficult and worsen its prognosis. A woman aged 75 years complaining of sleep disturbing pain and inaccurate projection of rays oculus dextrus (OD) was referred to our centre for management of non-resolving microbial keratitis with endophthalmitis. Ultrasound biomicroscopy revealed 360° peripheral anterior synechiae, swollen crystalline lens and hyperechoic granules filling anterior chamber. A diagnosis of PLG and lens-induced uveitis was made and cataract was extracted after control of intraocular pressure (IOP). The patient was left aphakic. There was complete resolution of pain after surgery and at 3 months follow-up the IOP was controlled without any antiglaucoma medications. Rarely, secondary corneal opacification from long-standing pathological changes in PLG may mimic non-resolving microbial keratitis with endophthalmitis and requires a high index of suspicion for appropriate diagnosis and management.

BACKGROUND

Lens-induced glaucoma and uveitis are a rare complication of hypermature senile cataract (HMSC).^{1–5} Microbial keratitis (MK) is a potentially vision-threatening condition that results from microbial colonisation of cornea.⁶ Delayed presentation of all these conditions may make their diagnosis and management difficult, and worsen their prognosis.

MK has rarely been described as a differential diagnosis of phacolytic glaucoma (PLG). Here, we describe an elderly patient presenting with lens-induced glaucoma and uveitis primarily suspected to have MK with endophthalmitis due to secondary corneal changes induced by the pathological process.

CASE PRESENTATION

A systemically healthy woman aged 75 years was referred to our centre with suspected MK and endophthalmitis not responding to topical and systemic antibacterial (fortified cefazolin and tobramycin) and antifungal (natamycin and voriconazole) agents. The patient complained of sudden-onset pain, redness, photophobia and diminution of vision oculus dextrus (OD) 2 months back for which she did not seek medical help due to rural

background and low socioeconomic status. The patient did not give any history of ocular trauma or previous ocular surgery. Two weeks after onset of these symptoms, she had a slowly progressive corneal opacification and sleep disturbing pain over a period of 2 months. At the time of presentation to our centre, her visual acuity was inaccurate projection of rays OD and hand motion oculus sinister (OS). Slit lamp biomicroscopy revealed circumferential congestion, central corneal opacification with irregular margins (figure 1A,B), shallow peripheral anterior chamber (AC) filled with yellow exudates and raised intraocular pressure (IOP) (assessed digitally) OD and a clear cornea, deep AC, HMSC and normal IOP OS.

INVESTIGATIONS

Anterior segment optical coherence tomography (ASOCT) revealed presence of diffuse hyper-reflective retrocorneal granules with uniformly increased corneal thickness and hyper-reflective stroma without an associated localised stromal lesion (figure 2). B-scan ultrasonography showed few mild-to-moderate amplitude spikes in the anterior and mid vitreous cavity suggestive of vitreous haemorrhage/exudates (figure 3A). Ultrasound biomicroscopy (UBM) revealed 360° peripheral anterior synechiae (PAS), swollen crystalline lens and hyperechoic granules filling AC (figure 3B).

DIFFERENTIAL DIAGNOSIS

Acute angle closure glaucoma, open-angle glaucoma with uveitis and glaucoma secondary to trauma are the conditions which can mimic PLG. While presence of AC exudates ruled out primary angle closure glaucoma in our case, existence of 360° PAS and absence of ocular trauma ruled out open-angle glaucoma and traumatic glaucoma, respectively. Also, prolonged course of disease, onset of pain prior to corneal opacification, slowly developing corneal opacification and lack of epithelial defect and stromal infiltrates made diagnosis of MK unlikely in our case.

TREATMENT

We made a diagnosis of secondary corneal opacification due to long-standing PLG and lens-induced uveitis and planned extraction of crystalline lens under peribulbar anaesthesia. After controlling IOP with intravenous mannitol, oral acetazolamide and

© BMJ Publishing Group Limited 2019. No commercial re-use. See rights and permissions. Published by BMJ.

To cite: Agarwal R, Tripathi M, Patil A, et al. *BMJ Case Rep* 2019;**12**:e231616. doi:10.1136/bcr-2019-231616

Figure 1 Slit lamp photograph showing opacified cornea and exudates in anterior chamber (A) and shallow peripheral anterior chamber (B).

topical timolol and brimonidine, the surgery was performed under sterile conditions. As soon as a side port entry was made, there was release of thick white fluid (figure 4A) with slight improvement in corneal clarity. A yellow colour mass similar to nucleus of a cataractous lens in colour and consistency was seen inside AC (figure 4B). The wound was extended and the lens was delivered (figure 4C). Anterior vitrectomy was done and the patient was left aphakic. The aqueous aspirate obtained during surgery was sent for cytopathological and microbiological examination.

OUTCOME AND FOLLOW-UP

While the smear and culture were negative for any organism, cytopathological examination of the aspirate revealed macrophages, leucocytes and cortical matter. Postoperatively, topical moxifloxacin 0.5%, homatropine 0.5% and prednisolone phosphate 0.5% were given four times per day. The steroids were tapered over a period of 1 month and all medications were stopped at 1-month follow-up. In the immediate postoperative phase and till 3 months follow-up (figure 4D), the patient had a dramatic relief in signs and symptoms despite no visual improvement. The IOP was controlled till last follow-up without the need for any antiglaucoma medications.

DISCUSSION

Lens-induced glaucomas can be either open-angle or closed-angle depending on the underlying mechanism. PLG is a rare type of secondary open-angle glaucoma that can occur due to leak of soluble lens proteins through microscopic defects in the capsule of a hypermature cataract.¹ In PLG, either macrophages laden with phagocytised lens protein or high molecular weight lens protein themselves are speculated

Figure 2 Anterior segment optical coherence tomography showing oedematous hyper-reflective stroma with hyper-reflective retrocorneal granules.

Figure 3 B-scan ultrasonography showing mild-to-moderate amplitude spikes in anterior and mid-vitreous cavity (A) and ultrasound biomicroscopy showing peripheral anterior synechia, exudates in anterior chamber and swollen cataractous lens (B).

to block the trabecular meshwork to produce elevated IOP.^{3,4} Phacogenic or lens-induced uveitis is a type of anterior uveitis (with or without vitreous inflammation) directed against lens proteins released or retained in the AC.⁵ These proteins can be released in the AC either after traumatic rupture of lens capsule or spontaneous leak can occur from capsular microleaks in a hypermature cataract.² MK is a potentially vision-threatening condition that results from microbial colonisation of cornea.⁶ These infections are characterised by the presence of white or yellowish infiltrates in the corneal stroma with or without signs of overlying epithelial defect, and signs of inflammation. In later stages, MK may also cause endophthalmitis. All these conditions mandate prompt diagnosis and management to prevent their untoward outcomes.¹⁻⁶

Various conditions mimicking PLG include acute angle closure glaucoma, open-angle glaucoma with uveitis, neovascular glaucoma and glaucoma secondary to trauma.⁷ MK has rarely been reported as a differential diagnosis of PLG.⁸ In 2011, Sahu *et al* reported a single case of PLG primarily presenting as deep corneal abscess. However, their case had a localised deep corneal lesion, clear surrounding cornea without any associated endophthalmitis, was approached primarily as MK and diagnosis established intraoperatively. PLG associated with secondary corneal changes severe enough to mimic MK with endophthalmitis, diagnosed preoperatively with the aid

Figure 4 (A) Leaked thick white fluid (red arrowhead) admixed with blood; (B) improved corneal clarity and cataractous lens (red arrowheads); (C) delivered crystalline lens (red arrowhead); (D) slit lamp photograph at 1-month follow-up (compare with figures 1A and 4A).

of UBM, to the best of our knowledge, is being reported for the first time.^{8 9}

There were multiple clinical findings that favoured a diagnosis of PLG in our case. An episode of pain, photophobia, redness and diminution of vision 2 months before presentation to our centre was suggestive of acute episode of signs and symptoms classically seen in PLG.¹⁻³ Delay in seeking treatment for cataract in the affected eye is commonly seen in patients with PLG due to good vision in the other eye.¹⁻³ In our case, delay in seeking ophthalmic help, despite having poor vision in the other eye, could be attributed to the patient's rural background and low socioeconomic status. Older age, lack of ophthalmic treatment until and presence of HMSC in the other eye also suggested presence of untreated advanced cataract in the affected eye. Onset of pain 2 weeks prior to onset of corneal lesion in our case was against MK as these usually occur simultaneously or immediately sequentially in MK. Clinical presence of diffuse stromal opacification and oedema (suggestive of corneal decompensation) along with AC exudates (confirmed on ASOCT and UBM), without an associated corneal infiltrate and epithelial defect also disfavoured diagnosis of MK in our case. As both cellular and humoral immune response have been implicated in the pathogenesis of PLG, we believe that both the AC exudates and diffuse corneal decompensation seen in our case were secondary to chronic inflammation from long-standing lens-induced uveitis. Unlike PLG that is associated with open angles without any anterior or posterior synechia, presence of 360° PAS and shallow AC in our case could be due to permanent changes in the AC secondary to chronic inflammation from lens-induced uveitis.³ We believe that chronic inflammation due to long-standing uveitis could have led to permanent changes in the AC resulting in 360° PAS and few mild-to-moderate amplitude spikes in the anterior vitreous cavity of the affected eye.² It is also likely that these ultrasonic vitreous spikes must have led to a clinical suspicion of endophthalmitis by the treating physician elsewhere.

Anterior chamber paracentesis is a useful technique of establishing causative agent in infectious keratitis, particularly when the infiltrate is deep.^{10 11} This can also help in cytopathological examination of aqueous aspirate in suspected PLG cases. However, this is an invasive method and carries its own complications. In the case reported by Sahu *et al*, AC paracentesis was undertaken to confirm diagnosis of MK and to determine its aetiological organism.⁸ However, the affected individual in their case had a deep AC and reasonably good corneal clarity. While in our case, directly lens extraction was planned without need for primary AC paracentesis as UBM proved to be a useful non-invasive method of diagnosis of PLG by demonstrating a swollen crystalline lens. UBM helped us in diagnosis of PLG and circumvented need for unnecessary AC paracentesis in an eye with already compromised corneal clarity, high IOP and 360° PAS.⁸

Dramatic symptomatic improvement and a controlled IOP, without the need for any antimicrobial or antiglaucoma medications, after surgery also confirmed diagnosis of PLG in our case. Nevertheless, the diagnosis can also be confirmed by cytopathological examination, as seen in our case, which revealed macrophages, leucocytes and cortical matter. However, waiting for cytopathological assessment results may cause unnecessary delay in appropriate treatment of the patient. This makes detailed history and thorough clinical examination the most crucial steps in establishing diagnosis of PLG and importance of non-invasive modalities, such as ASOCT and

UBM, in establishing correct diagnosis of atypical PLG cases is overemphasised again.

Urgent control of IOP with antiglaucoma medications is the first step in management of PLG and steroids are used to relieve the uveitic component. However, these are only temporary measures and the definitive treatment involves extraction of cataract with or without intraocular lens (IOL) implantation.^{12 13} Keeping this in mind, lens extraction was planned in our case to provide symptomatic relief to the patient and waive off unnecessary antiglaucoma medications. The placement of IOL, either in the posterior chamber or in the anterior chamber, may depend on numerous factors such as the status of posterior capsule, iris integrity and corneal endothelium and AC depth.^{12 13} Diffusely decompensated cornea with limited visualisation of posterior capsule details and presence of 360° PAS precluded placement of IOL in our case and the patient was left aphakic.

The prognosis of PLG depends majorly on the duration of signs and symptoms and may be guarded if the primary presentation is delayed or if there is an associated optic atrophy, uveitis or corneal oedema.¹³ Patients with defective projection of light perception may also have a chance of good visual recovery provided they present early.¹² Nevertheless, nil visual prognosis is not a contraindication for cataract surgery in these patients as crystalline lens extraction is expected to provide symptomatic relief from a painful blind eye as seen in our case.

To conclude, our case report highlights the importance of early presentation in ensuring timely diagnosis of PLG. In eyes with delayed presentation where corneal haze limits visualisation of intraocular structures, UBM may play an indispensable role in its diagnosis and management.

Patient's perspective

I had complete relief of symptoms and non-dependence on medications after surgery. I thank the doctors at Dr RP Centre for taking good care of me.

Learning points

- ▶ Phacolytic glaucoma (PLG) is a rare type of glaucoma associated with leak of soluble lens proteins through microscopic capsular defects in a hypermature senile cataract.
- ▶ Secondary corneal opacification and anterior chamber exudates from long-standing pathological changes in PLG and lens-induced uveitis may mimic non-resolving microbial keratitis with endophthalmitis and high index of suspicion should be maintained for all elderly patients with similar clinical presentation.
- ▶ Ultrasound biomicroscopy may serve as a useful non-invasive modality in diagnosing PLG in eyes with compromised corneal clarity.
- ▶ Delayed presentation of PLG may make its diagnosis and management difficult and worsen its prognosis. Yet, despite nil visual prognosis, cataract extraction may be required in these patients to provide symptomatic relief from a painful blind eye.

Contributors RA: manuscript writing; MT: data collection; AP: patient care; NS: supervision and manuscript editing.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Flocks M, Littwin CS, Zimmerman LE. Phacolytic glaucoma; a clinicopathologic study of one hundred thirty-eight cases of glaucoma associated with hypermature cataract. *AMA Arch Ophthalmol* 1955;54:37–45.
- 2 Luntz MH, Wright R. Lens-induced uveitis. *Exp Eye Res* 1962;1:317–IN28.
- 3 Epstein DL, Jedziniak JA, Grant WM. Identification of heavy-molecular-weight soluble protein in aqueous humor in human phacolytic glaucoma. *Invest Ophthalmol Vis Sci* 1978;17:398–402.
- 4 Epstein DL. Diagnosis and management of lens-induced glaucoma. *Ophthalmology* 1982;89:227–30.
- 5 Gujjula C, Kumar S, Varalakshmi U, et al. Study of the incidence, mechanism, various modes of presentation and factors responsible for the development of lens-induced glaucomas. *Albasar Int J Ophthalmol* 2015;3:56–62.
- 6 Upadhyay MP, Srinivasan M, Whitcher JP. Diagnosing and managing microbial keratitis. *Community Eye Health* 2015;28:3–6.
- 7 Allingham RR, Damji K, Freedman S, et al. chapter 18 Glaucomas associated with disorders of the Lens. *Sheilds' Textbook of Glaucoma*. 5th edn. Philadelphia, PA, USA: Lippincott Williams & Wilkins:323–4.
- 8 Sahu SK, Keswarani S, Mittal R. Unusual presentation of phacolytic glaucoma: simulating microbial keratitis. *Case Rep Ophthalmol Med* 2011;2011:1–3.
- 9 Papakostas TD, Yonekawa Y, Chee YE, et al. Ultrasonographic biomicroscopy in lens-induced glaucoma. *JAMA Ophthalmol* 2015;133:112.
- 10 Sridhar MS, Sharma S, Gopinathan U, et al. Anterior chamber tap: diagnostic and therapeutic indications in the management of ocular infections. *Cornea* 2002;21:718–22.
- 11 Murphy GE. Acute phacolytic glaucoma with primary intraocular lens implantation after intracapsular cataract extraction. *J Am Intraocul Implant Soc* 1981;7:266–7.
- 12 Mandal AK, Gothwal VK. Intraocular pressure control and visual outcome in patients with phacolytic glaucoma managed by extracapsular cataract extraction with or without posterior chamber intraocular lens implantation. *Ophthalmic Surg Lasers* 1998;29:880–9.
- 13 Pradhan D, Hennig A, Kumar J, et al. A prospective study of 413 cases of lens-induced glaucoma in Nepal. *Indian J Ophthalmol* 2001;49:103–7.

Copyright 2019 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <https://www.bmj.com/company/products-services/rights-and-licensing/permissions/>
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

Customer Service

If you have any further queries about your subscription, please contact our customer services team on +44 (0) 207111 1105 or via email at support@bmj.com.

Visit casereports.bmj.com for more articles like this and to become a Fellow