Posterior lens capsule abscess due to
Propionibacterium acnes and Staphylococcus epidermidis following extracapsular cataract extraction

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Abstract
A case of posterior lens capsular abscess occurring many months after an extracapsular cataract extraction is presented. This was caused by a mixed infection involving Propionibacterium acnes and Staphylococcus epidermidis. The significance of Staph epidermidis after such a long postoperative period is uncertain, but the case shows features typical of secondary endophthalmitis due to P acnes, including a long delay in onset and a grumbling course not brought under control by medical treatment. It supports the theory that the nidus of infection is localised in the posterior lens capsule by showing development of a visible capsular abscess with associated vitreous involvement. The subsequent removal of the capsule and vitreous, despite leaving the intraocular lens in place, led to complete resolution of the inflammation. Both organisms have previously been found to be sequestered in the posterior lens capsule by histological and microbiological examination of excised capsular specimens. It is important to consider them as possible causative agents in the formation of a postoperative capsular abscess.

There is an increasing awareness that the differential diagnosis of chronic, indolent, postoperative endophthalmitis, especially following extracapsular cataract extraction, should include the anaerobe Propionibacterium acnes. This is a ubiquitous organism found on skin and conjunctiva and was generally previously dismissed as a contaminant. Propionibacteria are anaerobic, Gram-positive rods, and the organism may occur as a mixed infection as shown in the case presented here. It is highly sensitive to penicillin but less so to cephalosporins and resistant to gentamicin and vancomycin. P acnes has also been reported as causing intralenticular abscess and postoperative endophthalmitis following trabeculectomy and keratoplasty.

Staphylococcus epidermidis is an aerobic, Gram-positive organism of low virulence, commonly found in normal eyes and causally associated with postoperative endophthalmitis since 1964. Generally the delay between surgery and postoperative infection is a matter of days and is thought to be due to antibiotics and steroids quelling the initial response. Latent periods of several months are rare.

We report here a case of delayed postcataract extraction endophthalmitis due to a mixed infection of P acnes and Staph epidermidis. There are aspects of this case which suggest that the chronic infective source of the organisms in the eye was in the lens capsule.

In April 1989 a 79-year-old woman was admitted for left cataract extraction. Two years earlier a right extracapsular cataract extraction with posterior chamber lens implant had been performed, and her visual acuities then were 6/12 in the right eye and hand movement in the left with appropriate myopic correction.

An uneventful left extracapsular cataract extraction and posterior chamber intraocular lens insertion was performed under local anaesthesia. The procedure included a corneal section, needle can-opener capsulotomy, nucleus expression by external pressure, irrigation and aspiration with the Simcoe cannula, and insertion of an American Medical Optics posterior chamber, 22 dioptre intraocular lens, model PC57B, under viscoelastic substance which was subsequently aspirated. The section was closed with five 10/0 monofilament nylon sutures and the eye padded. The patient was discharged on the second postoperative day on chloramphenicol eyedrops 0.5% and dexamethasone eyedrops 0.1% four times a day.

The eye initially settled well following surgery, and five weeks postoperatively a visual acuity of 6/9 was achieved with appropriate correction. The eye appeared quiet and at this stage the posterior lens capsule was clear. All treatment was stopped.

Five months later the patient attended the casualty department complaining of pain and watering in the left eye for two days. On examination the left visual acuity was 6/12 with glasses. Conjunctival injection was present, with a quiet anterior chamber. The posterior lens capsule was noted to be slightly thickened. A diagnosis of conjunctivitis was made and treatment with chloramphenicol eyedrops 0.5% four times a day was started again...

On review one week later the eye was still painful and a moderately severe anterior uveitis was found. Therefore dexamethasone eyedrops 0.1% and cyclopentolate eyedrops 1-0% were started, and over the next month the inflammation gradually settled and topical treatment was eventually stopped. However, within one week the patient attended again with a severe recurrence of the inflammation. Posterior synechiae and corneal oedema were seen, and once again a thickened posterior lens capsule was noted. A conjunctival swab at this stage showed no growth. Intensive topical steroids and cycloplegics was restarted, and during the next month
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![Image](http://example.com/image1.png)

Figure 1 Abscess of the posterior lens capsule extending posteriorly into the vitreous.

the anterior chamber had settled sufficiently to allow a clear view of the lens capsule, which was seen to have developed a localised, whitish, dense opacity just inferotemporal to the visual axis. There was infiltration extending back from this into the vitreous (Fig 1).

A diagnosis of endophthalmitis secondary to a posterior lens capsule abscess was made. Two days later a vitrectomy and capsulotomy were performed. An attempt was made to remove all abnormal looking capsule, leaving a peripheral annulus in which the lens was stable (Fig 2). During the procedure two specimens were taken: (1) capsular fluid, (2) vitreous. Intracameral antibiotics were not administered. A subconjunctival injection of cefuroxime 125 mg was given, and postoperative treatment was cefuroxime eyedrops 50 mg/ml two hourly, dexamethasone eyedrops 0.1% four times a day, and cyclopentolate eyedrops 1% four times a day. Oral fluoroacillin 250 mg and pivampicillin 500 mg four times a day and metronidazole 400 mg three times a day were given for seven days. All medication was tailed off over the next month. The eye has since remained quiet for six months and has a final visual acuity of 6/9.

**Bacteriological findings**

Microscopy showed Gram-positive cocci and rods together with pus cells in the capsular fluid but no organisms in the vitreous.

Culture showed a mixed growth from both the capsular fluid and vitreous of Propionibacterium acnes and a single strain of coagulase negative Staphylococcus which showed colonial variation. The former was sensitive to penicillin and the latter to cloxacillin.

**Discussion**

The many reported cases of postoperative *P. acnes* endophthalmitis show a characteristic clinical course. The delay in onset is several months, and the inflammation then has a grumbling, insidious nature, often partially responding to antibiotics and steroids but recurring once topical treatment is stopped, as this case showed.° The significance of the *Staph epidermidis* is difficult to assess. It is known to cause postoperative endophthalmitis, but a delay of five months before onset of symptoms is atypical.

The vector by which the organism gains entry to the eye is thought to be the intraocular lens. Vafidis et al° showed that, when sterile intraocular lenses were placed on the conjunctival flap during cataract surgery, 28% became contaminated with organisms, mainly *Staph epidermidis*. They suggested that minimising contact of the lens implant with the eye before insertion would reduce the chances of intraocular infection. Griffiths et al° showed in vitro that *Staph epidermidis* can adhere to the plastic surface of the intraocular lens by producing a polysaccharide glycocalyx which acts like glue. Unfortunately this substance also seems to confer some resistance to antibiotics.

The intraocular lens used in this case was a one-piece, biconvex lens, posteriorly vaulted to an angle of 10° with no laser ridge. Thus it is assumed that the posterior capsule was apposed to the back surface of the lens with no intervening space. The presence of an IOL has been shown to increase the intensity and duration of *P. acnes* infection in rabbit eyes, and possible explanations are that the IOL itself acts as a nidus for infection or that increased trauma during lens insertion may damage the blood- aqueous barrier, which will prolong the inflammatory response to intraocular bacteria.°

The case presented here showed clearly that the continuing infective site was the posterior lens capsule. Exudative material on the capsule and IOL occurring in the active stage of an indolent, granulomatous iridocyclitis has been previously reported by Meisler et al°. Since the infection is thought to be associated with the lens capsule, it is more commonly found following extracapsular cataract extraction. Piest et al° examined capsule sacs that had been removed from cases of persistent, smouldering endophthalmitis and found pleomorphic bacilli in large numbers in the residual lens cortex and often in folds of capsule. In one case cultures were performed and *P. acnes* grown. They thought that the capsule acted as a protective barrier preventing a full blown disseminated endophthalmitis, which was reported following a Nd-YAG laser capsulotomy. *Staph epidermidis*

![Image](http://example.com/image2.png)

Figure 2 Postcapsulectomy and vitrectomy showing clearance of infective nidus and retention of intraocular lens.
has also been reported to cause endophthalmitis following Nd-YAG capsulotomy in a previously quiet eye five months after extracapsular cataract extraction. It was assumed that the low virulence micro-organisms had been sequestered in the capsular bag and released into the vitreous following laser treatment.

Ormerod et al. have found experimentally in rabbits that the local environment in the vitreous just posterior to the lens has an oxidation-reduction potential (ERh) and an oxygen pressure suitable for the growth of anaerobic organisms. They produced P. acnes endophthalmitis which was self-limiting. Many factors may have suppressed these infections including the rise in ERh with inflammation above the limiting value for the anaerobic bacteria. Administration of 100% oxygen to the anterior chamber has thus been put forward as a treatment for this condition.

Clinically, when topical treatment fails to control the inflammation, further intervention is required. Zambrano et al. have suggested that less severe cases can be treated with intravitreal vancomycin (with or without topical and intravenous antibiotics) and have shown successful results. They suggest vancomycin as the initial choice since it is effective against P. acnes and also coagulase negative staphylococci. Alternative antibiotics include methicillin and the cephalosporins. However, if the condition is initially severe or fails to respond to medical management, they recommend capsulotomy and pars plana vitrectomy together with intravitreal vancomycin. Our patient underwent such surgery without any antibiotics and the eye had improved dramatically within two days.

The intracocular lens alone does not appear to be the nidus of infection, since Meisler showed that whether or not the lens was removed in addition to the capsulotomy and vitrectomy made no difference to the successful outcome in their series of six cases. Driebe et al. found only one out of 57 cases of pseudophakic endophthalmitis in which the eye could not be sterilised while the intraocular lens was retained. Bacteria could have remained on the surface of the lens, but alterations in the local environment, such as loss of substrate cells and exposure to host defences by removal of the lens capsule, may have prevented regrowth. These theories are supported by the course in our patient who has retained her intraocular lens in a quiet eye for six months following surgery.

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