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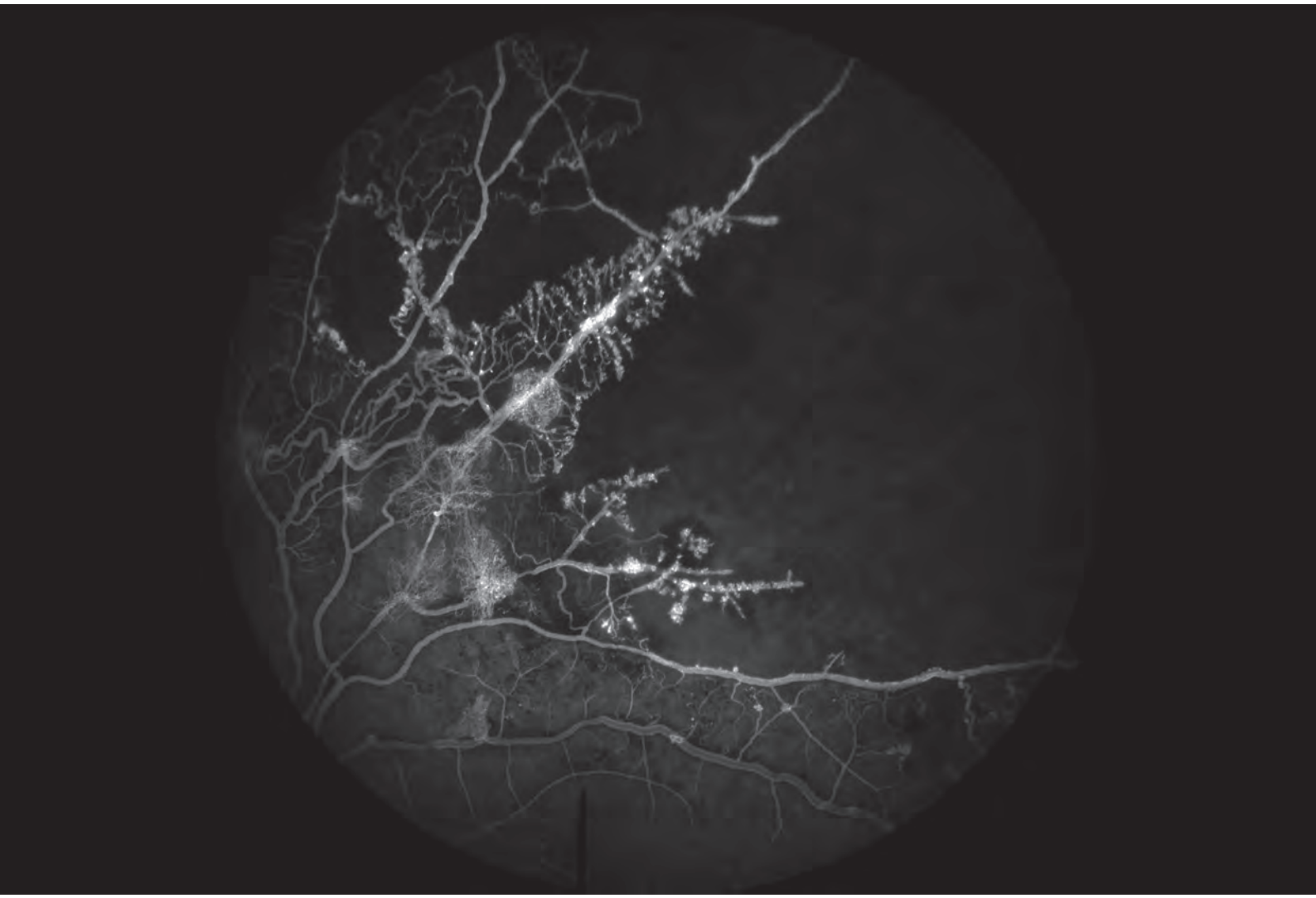

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Sankara Nethralaya – The Temple of the Eye.

It was in 1976 when addressing a group of doctors, His Holiness Sri Jayendra Saraswathi, the Sankaracharya of the Kanchi Kamakoti Peetam spoke of the need to create a hospital with a missionary spirit. His words marked the beginning of a long journey to do God's own work. On the command of His Holiness, **Dr. Sengamedu Srinivasa Badrinath**, along with a group of philanthropists founded a charitable not-for-profit eye hospital.

Sankara Nethralaya today has grown into a super specialty institution for ophthalmic care and receives patients from all over the country and abroad. It has gained international excellence and is acclaimed for its quality care and compassion. The Sankara Nethralaya family today has over 1400 individuals with one vision – to propagate the Nethralaya philosophy; the place of our work is an Alaya and Work will be our worship, which we shall do with sincerity, dedication and utmost love with a missionary spirit.

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Corticosteroids: Time to Put Old Wine in a New Bottle

Jyotirmay Biswas

Director of Uveitis and Ocular Pathology Departments, Sankara Nethralaya

Correspondence to:
Prof. Jyotirmay Biswas, MS, FMRF, FNAMS, FIC Path., FAICO
Director of Uveitis and Ocular Pathology Departments, Sankara Nethralaya,
No. 41, College Road, Chennai,
email: drjb@snmail.org

Inflammatory eye disease is an important cause of blindness in the working age group.[1,2] The incidence of blindness in uveitis can be as high as 35%, with bilateral loss in 10%.[3] Corticosteroid has been the mainstay of treatment of non-infectious ocular inflammation. Corticosteroids are known to inhibit the inflammatory responses by various ways. They inhibit the activity of transcription factors, such as activator protein-1 and nuclear factor-b, which in turn prevent activation of proinflammatory genes. They inhibit the production of cytokines involved in the inflammatory response and prevents leukocyte migration to the area of inflammations. They also exert their anti-inflammatory actions by interfering with the functions of endothelial cells, leukocytes, and fibroblasts.[4]

Corticosteroids are used as topical, periocular, intravitreal and systemically depending on the site and severity of the inflammations. Topical steroids penetrate well into the anterior chamber and has been used for anterior segment inflammations, namely anterior uveitis, episcleritis etc. Use of periocular steroid remained limited to the control of cystoid macular oedema associated with pars planitis. Though it has been tried for treatment of various other ocular inflammation, for example noninfectious scleritis, the role of this route remains controversial. Though in some patients, it can cause increase of intraocular pressure, it can be used safely without any systemic side effects of corticosteroid. Because of the nature of inflammation, most of the time it remains essential to use systemic corticosteroid or immunosuppressives along with this modality of treatment. Oral steroids are most commonly systemically administered by oral route for the management of noninfectious ocular inflammation. However, in sight-threatening conditions like macular serpiginous choroiditis, exudative retinal detachments secondary to inflammations require rapid and more aggressive anti-inflammatory therapy in the form of 'pulsed' (high dose) intravenous corticosteroids. We use 1000 mg of methylprednisolone per day for 3 consecutive days for severe intraocular inflammations. Here it must be kept in mind that though systemic corticosteroid deliver effective anti-inflammatory actions, systemic side effects are present significantly, especially with long-term use, which often outweigh the benefit of this agents. Intravitreal injection of corticosteroids has the advantages here, they deliver the required amount of drug to target tissues without extraocular side effects. Triamcinolone acetonide is a long-acting corticosteroid commercially available in suspension form and proven to be nontoxic to ocular structures when injected intravitreally. Though elimination half-life of this formulation has been estimated 18 to 30 days (in nonvitrectomized eyes, less in vitrectomized eyes),[5] the triamcinolone acetonide has been isolated from aqueous humor and silicone oil, 1.5 years after the intravitreal injection.[6,7] After intravitreal injection, optimal concentrations of triamcinolone would last for approximately 3 months.[5] Over the last decades intravitreal use of triamcinolone acetonide has been increased dramatically especially for the treatment of macular edema secondary to intraocular inflammations. However, it is associated with various ocular side effects, such as cataract, secondary ocular hypertension and infectious, or sterile endophthalmitis. Implantation of devices that provide long-lasting infusion of a corticosteroid formulation is very popular these days. Such implants offer an alternative therapeutic approach to the treatment of intraocular inflammation. Retisert is a nonbiodegradable implant, made up of silicone/polyvinyl alcohol, containing 0.59 mg fluocinolone acetonide which can release the drug slowly (0.3–0.6 µg/day) over a period of 3 years. It is 5 mm long, 2 mm wide and 1.5 mm thick which is inserted into the vitreous cavity and sutured to the sclera through a pars plana surgical incision. In 2005, retisert became the first FDA-approved device for use in the treatment of chronic noninfectious posterior uveitis. Despite the excellent control of intraocular inflammation, the device is not used frequently because of its high complication rate. Multicenter Uveitis Steroid Treatment (MUST) trial compared the relative efficacy of systemic anti-inflammatory therapy and fluocinolone acetonide implant for the treatment of noninfectious cases of intermediate, posterior, and panuveitis. The trial concluded that both treatment groups were effective and neither group was superior to the other in improving visual acuity. According to the trial, in comparison to well-tolerated systemic therapy, the implant group had an increased risk of development of cataracts and elevated intraocular pressures.[8] Ozurdex is a biodegradable implant which contains dexamethasone as the active pharmaceutical ingredient. Dexamethasone has a short half-life compared with other corticosteroids, but it is 20 and 5 times

more potent than fluocinolone and triamcinolone, respectively. It contains 0.7 mg dexamethasone, provides peak doses for 2 months and releases the drug up to 6 months. Ozurdex, a rod-shaped 6.5 × 0.45 mm pellet, is placed intravitreally through the pars plana with the help of an injector with a 22-gauge needle. Initially approved for the treatment of macular edema associated with retinal vein occlusion, this device, in September 2010, became the second FDA-approved therapeutic agent for the treatment of noninfectious posterior uveitis. In a 26-week, multicenter, double-masked, randomized clinical study, the device has showed promising results in controlling intraocular inflammations and the rate of complications were less compared with the other intravitreal implants.[9,10]

Thus in the treatment of the noninfectious uveitis, corticosteroids remain the mainstay. Even in the infectious uveitis, corticosteroids are often used in conditions like toxoplasma infection and acute retinal necrosis. The tapering of corticosteroid remains an art. There are several principles like the one suggested by Dr Douglas Jab and they primarily depend on the severity of the inflammation. It needs an art to taper the oral steroid and to find exact dosage for a particular patient. Though immunosuppressive agents, biologicals have emerged as new modalities of treatment, corticosteroids still plays an important role in the management of intraocular inflammation especially in Indian scenario.

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Regional Ophthalmic Anaesthesia: An Update

V.V. Jaichandran

Department of Anaesthesia,
Sankara Nethralaya,
Chennai, India

Correspondence to:
V.V. Jaichandran,
Senior Consultant,
Department of Anaesthesia,
Sankara Nethralaya,
Chennai, India.
Email: drvvj@snmail.org

Introduction

Ophthalmic regional anaesthesia have moved a full circle from the ancient days of 'no-anaesthesia' couching, to Koller's topical cocaine, through general anaesthesia, Knapp's local anaesthesia and now to topical and no-anaesthesia phacoemulsification.

Today, most surgeons throughout the world use local anaesthesia for cataract surgery, though topical anaesthesia is gaining popularity. There are few contraindications and the techniques have both a high-success rate and a wide margin of safety. However, serious life threatening complications may occur and it is essential that those involved with the care of these patients have a thorough knowledge of the anatomical, pharmacological and practical aspects of the techniques. This review article outlines the relevant anatomy, describes preoperative preparation, monitoring, discusses the commonly used agents, current choices of ocular anaesthesia, compare their efficacies and inherent complications.

Preoperative systemic assessment and preparation

A routine preoperative consultation should be carried out for every patient, including detailed medical, anaesthesia, surgical and medication history. An appropriate physical examination should be conducted. Certain basic investigations like blood sugar estimation and ECG especially in patients aged 60 years and above can be carried out as a routine on all patients and to let clinical judgment guide the need for more extensive investigations. More specific investigations may be required in patients who have a positive risk factor identified while taking history or during physical examination.[1] Patient should be referred to primary care or specialist physicians to manage new or poorly controlled diseases. Preoperative optimization of medical conditions (control of blood sugar, blood pressure, etc.) is required before they could be taken up for planned surgery. Antiplatelet (aspirin) or anticoagulant (warfarin) can be continued for patients posted for cataract surgery (least risk for haemorrhage). Patients on anticoagulants such as warfarin should have International Normalized Ratio (INR) measured, close to the time of surgery, ideally on the same day and the INR should be within the recommended therapeutic range. For non-cataract ocular and orbital surgery (intermediate high risk of haemorrhage) then in the interest of total patient care there should be discussion between anaesthesiologist, surgeon and patient regarding the risks and benefits of continuing/discontinuing anticoagulants and antiplatelet drugs and to agree an acceptable approach such as bridging therapy.[2, 3]

Both the anaesthetic and surgical procedure must be clearly explained to the patient. ECG, blood pressure and pulse oximeter are connected. Intravenous line must be inserted before embarking on a needle block. Patients should receive oxygen 2 l/min via nasal catheters placed near the patient's nostrils.[4]

Preoperative ophthalmic evaluation

Elements of ophthalmic evaluation include history of previous ophthalmic surgery, glaucoma, know about the axial length, presence of any staphyloma and the relationship between globe and orbit. Buckling surgery alters globe dimensions, contour and results in significant scarring within the orbit and so increases the potential of perforation[5]. The axial length and the presence and the location of staphyloma can be known from B-scan echography, which is usually done, before cataract surgery, for diopter power calculation. If the axial length is not available, the spherical equivalent in the patient's eye glass prescription should be reviewed. High myopes tend to have exceptionally long eyes and patients who have axial lengths ≥27 mm are at risk for posterior staphylomas. After the patient's eye length has been determined the relationship of the eye within the orbit should be examined. Knowledge of this relationship is used to determine the angle of the block needle as it enters the desired orbital space in order to avoid penetrating the sclera.

Orbital: globe spatial relationship

The orbital axis is the bisection of line between medial and lateral orbital walls, while visual axis is the position of the eye in primary gaze. Both the axis diverge at an angle of 23°. Normally, the equator of the globe is at or slightly anterior to the lateral orbital rim and the spatial relationship between them is assessed by measuring the distance the globe (top of the cornea) extends over the infraorbital rim and this distance is generally about 8 mm.[6]

Forward set globe

The globe extends quite forwardly over the infraorbital rim (>8 mm) and associated eye lids will be lax with wide palpebral fissure and high brows. Here, the structures in the apex of the orbit are vulnerable to get injured with needle blocks.

Deep set globe

In this condition, there is high chance for the needle to come in contact with the globe. Associated eyelids will be short and tight. From the point of insertion, at the inferolateral quadrant, the needle must not be angulated more than 10° elevations from the transverse plane.

Different types and techniques of regional anaesthesia

The provision of ophthalmic regional anaesthesia for cataract surgery varies worldwide. These may be chosen to eliminate eye-movements or not and both akinetic and non-akinetic methods are widely used.[7–9]

Akinetic techniques

1 Needle-based technique

Retrobulbar block

The aim is to block the oculomotor nerve before they enter the four rectus muscles in the posterior intraconal space. In the modern retrobulbar block, 23 G, 31 mm long needle, with bevel facing the globe, is inserted through the skin in the infero-temporal quadrant as far laterally as possible, just above the junction of inferior and lateral orbital walls, see Figure 1.[10, 11] The atkinson's or classic insertion site, i.e. the junction of medial two-third and lateral one-third of the lower orbital margin, is no more recommended. The reasons cited are: the needle being nearer to the globe, inferior rectus muscle and also closer to the neurovascular bundle supplying the inferior oblique. Several cases of diplopia owing to iatrogenic needle injury to inferior rectus and oblique muscles following needle entering at this site have been reported in literature.[12, 13]

From the extreme corner, it is easier to stay far away from the globe and would prevent any needle injury to the inferior rectus muscle or its neurovascular bundle. The initial direction of the needle is tangential to the globe. Once past the equator, as gauged by the axial length of the globe, the needle is allowed to go upwards and inwards.[14] With the eye in primary gaze, 4–5 cc of local anaesthetic agent is injected.

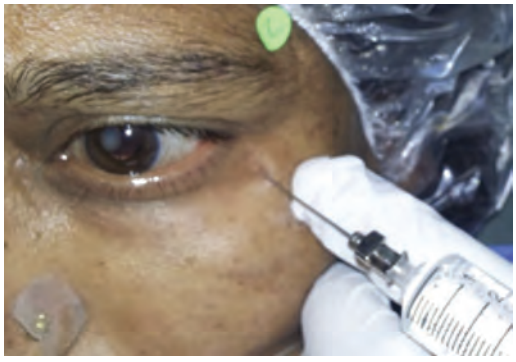

Figure 1 Needle entering at the junction of inferior and lateral walls of the orbit (extreme inferotemporal).

Advantages

- It produces excellent anaesthesia and akinesia.
- The onset of the block is quicker than with peribulbar.
- Low volume of anaesthetic result in a lower intraorbital tension and less chemosis than with peribulbar blocks.

Disadvantages

The main disadvantage is that the complication rate is higher than for peribulbar block.

Orbital complications

- Optic nerve injury due to inadvertent trauma from needle.
- Retrobulbar haemorrhage.
- Globe perforation.

Systemic complications

- Inappropriate large dose.
- Accidental intravascular injection.
- Intra-arterial injection with retrograde flow.
- CSF spread within the dural cuff around the optic nerve–brain stem anaesthesia.

Other disadvantages

All extraocular muscles can be paralyzed by this technique except superior oblique as it is supplied by the trochlear nerve (IV nerve), which lie outside the muscle cone. Akinesia of the eye lids may be incomplete. A separate eye lid block, using 2.5 cc of 1% lignocaine solution injected 0.5 cm below (lower lid) and 1.0 cm above (upper lid) the middle of the canthus, might be required.[15]

Peribulbar block

The principle of this technique is to instill the local anaesthetic outside the muscle cone and avoid proximity to the optic nerve. In the modern peribulbar block 23 G, 25 mm long needle is inserted as far laterally as possible in the infero-temporal quadrant. Once the needle is under the globe, it is directed along the orbital floor, passing the globe equator to a depth controlled by observing the needle/hub junction reaching the plane of the iris.[16] After negative aspiration for blood, with the globe in primary gaze, 4–5 cc of local anaesthetic agent is injected.

Advantages

- By this procedure, all extraocular muscles including superior oblique can be paralyzed.

- Thus, effective analgesia and akinesia of the globe.
- The risk of local and systemic complications associated with this technique is low.
- The local anaesthetic solution diffuses through the orbital septum and orbicularis muscle can also be paralyzed. Thus, a separate eye lid block might not be required with this technique.

Disadvantages

- Onset of action is slower compared with retrobulbar technique.
- More amount of LA volume might be required.
- Increase in IOP is more compared with retrobulbar anaesthesia.

Following a fractionated inferotemporal injection, intraocular pressure increases significantly and this may lead may lead to vitreous loss during intraocular surgery.[17] Hence, adequate compression of the globe either by using digits or Honan Intraocular pressure reducer should be done.

Mechanism of ocular compression

The ocular compression helps in decreasing IOP by the following suggested mechanism:

1 Decreasing the volume of the vitreous, which is about 50% water in the elderly patients.

2 Decreasing the volume of the orbital contents other than the globe by increasing the systemic absorption of orbital exracellular fluid, including, presumably, injected fluids such as anaesthetics.

3 Increasing the aqueous outflow facility mechanism.

4 Emptying the choroidal vascular bed.

Technique of ocular compression

The ocular digital compression is done gently with the middle three fingers placed over a sterile gauze pad on the upper eye lid with the middle finger pressing directly down on the eyeball. For every 30 s, digital pressure is released for 5 s to allow for the vascular pulsations to occur (Intermittent digital pressure).[18] The ocular compression device most commonly used is Honan's balloon which is applied for 10–20 min with the pressure set at 30 mm Hg. After adequate compression, if significant movement of the eye still persists, then supplementary injection, medial peribulbar block is administered. The administration of this supplementary injection depends upon the type and duration of the procedure to be performed, the experience of the ophthalmologist and the preference of the anaesthesiologist.

Medial peribulbar block

It is given using 26 G, ½″ disposable needle. With the bevel facing the medial orbital wall, needle is passed into the blind pit, between the medial caruncle and canthus (Figure 2). It is passed backwards in the transverse plane, directed at 5° angle away from the sagittal plane and towards the medial orbital wall. If the medial wall is contacted, the tip is slightly withdrawn and needle is redirected to a depth of 15–20 mm and after negative aspiration for blood, 3–5 cc of local anaesthetic solution is injected.[14]

Advantages

- Helps in the supplementation of the primary intra- or extraconal blocks.
- This extraconal space is an excellent site for administering local anaesthesia, as it communicates freely with the intraconal space. Also, with this injection, eyelids may fill with the anaesthetic solution which provides excellent orbicularis akinesia too.
- Given as primary injection in patients with high axial length or those with posterior staphyloma.

Disadvantages

- Orbital cellulitis or abscess.
- Haemorrhage.
- Globe perforation has been reported.

2 Cannula-based injection technique

Sub-Tenon's block

It was introduced into clinical practice in the early 1990s as a simple, safe, effective and versatile alternative to needle block.[2, 19] It is also known as parabulbar, pinpoint anaesthesia[20] or episcleral block.[21] Local anaesthetic eye drops are instilled onto the conjunctiva. Under sterile conditions, at the inferonasal quadrant, 3–5 mm away

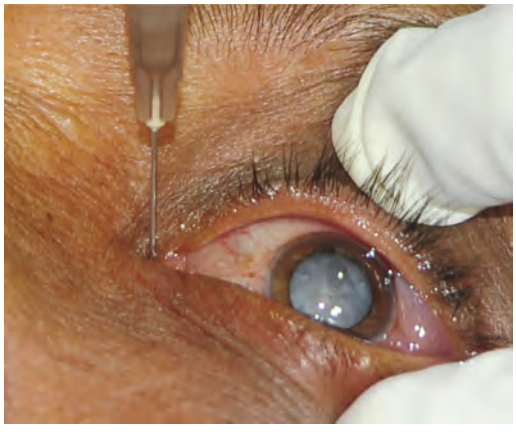

Figure 2 Needle entering between the medial caruncle and medial canthal angle for performing medial peribulbar injection.

from the limbus, the conjunctiva and Tenon capsule are gripped with non-toothed forceps and a small incision is made through these layers with Westcott scissors to expose the sclera. A blunt curved posterior sub-Tenon's cannula mounted on to a 5 ml syringe with local anaesthetic is inserted through the hole along the curvature of the sclera. Injection of local anaesthetic agent under the Tenon capsule blocks the sensation from the eye by action on the short ciliary nerves as they pass through the Tenon capsule to the globe. Akinesia is obtained by direct blockade of anterior motor nerve fibres as they enter the extraocular muscles. Two percentage lidocaine is the most commonly used local anaesthetic agent.[22]

Advantages

- It can be performed as a primary block technique or as a supplementary block to augment needle block performed as a primary method of anaesthesia.
- Simple, safe and effective alternative technique to needle block.
- Very minimal chance for optic nerve getting injured especially smaller volume of LA is injected anteriorly.

Disadvantages

- Chemosis and sub-conjunctival haemorrhage can hinder the surgical field, it can also stimulate postoperative scarring later.[14]
- Not a suitable technique in patients who had multiple surgeries owing to scarring.
- Leakage of local anaesthetic solution from the injection site which decreases the effectiveness of the block.
- Akinesia is variable and volume dependent.
- Orbital and retrobulbar haemorrhage, globe perforation, the central spread of local anaesthestic and orbital cellulitis have been reported to occur.[23–25]

Non-akinetic technique

1. Topical anaesthesia

It can be achieved either by instilling local anaesthetic eye drops (0.5% Proparacaine Hydochloride or 2–4% lignocaine)[26] or application of lignocaine gel[27] and found to be useful for cataract, glaucoma surgery like trabeculectomy[28] and secondary intraocular lens implantation. Topical anaesthetic agents block trigeminal nerve endings in the cornea and conjunctiva, leaving the intraocular structures in the anterior segment unanaesthetized. Thus, manipulation of the iris and stretching of the ciliary and zonular tissues during surgery can irritate the ciliary nerves, resulting in discomfort. A modified technique consists of combining topical anaesthesia with 0.5 ml of 1% lignocaine (preservative free) injected through the side port incision after evacuation of aqueous (intracameral anaesthesia).[29] It provides sensory blockage of the iris and ciliary body and thereby relieves discomfort experienced during intraocular lens placement.

Advantages

- Topical anaesthesia not only avoids the systemic and local complications associated with eye blocks but it is also the most cost and time efficient.
- No fear or pain of injection of the needle.

Disadvantages

Apart from surgical skill, the patient should be cooperative enough for successful completion of eye surgery under topical anaesthesia.

In addition, the duration of anaesthetic effect is typically less than an hour. Even in uncomplicated cases, there may be a loss of effect by the end of a case. For vitreo-retinal surgeries, corneal surgeries, etc. topical anaesthesia would not be appropriate.

Retained visual sensations that include seeing light, colours, movements and instruments during surgery are expected to occur more frequently under topical anaesthesia because optic nerve function is not affected. Although majority of patients feel comfortable with visual sensations they experience, a small proportion find the experience unpleasant or frightening[30]. Preoperative counselling and IV Midazolam are known to alleviate the fear caused by intraoperative visual images seen.[31]

Pharmacological considerations

Local anaesthetic agents

Lidocaine hydrochloride: It is available as 2 and 4% solution for the injection and topical use. Its onset of action is quick, but the duration of action is relatively short. The action can be prolonged by the addition of adrenaline. Dosage to be used 5–7 mg/kg body weight and 3–5 mg/kg body weight with and without adrenaline, respectively.

Bupivacaine hydrochloride: It has higher lipid solubility and protein binding, and is therefore more potent and has a longer duration of action than lidocaine, see Table 1. It offers excellent postoperative analgesia as well. A common practice is to use combination of 1:1 mixture of 2% lignocaine with 0.5% bupivacaine. Dosage to be used 2–3 mg/kg body weight.

Ropivacaine and levobupivacaine are some of the newly marketed amide group of local anaesthetic agents that have been found to be useful for eye surgery.[32, 33]

Ropivacaine: A long acting, pure S-(-)-enantiomer, amide local anaesthetic similar to bupivacaine in duration. It is prove to be less

Table 1 Pharmacological properties and pharmacodynamics of different local anaesthetic agents.

Local anaesthetic agents	Lipid solubility	pKa	Protein binding	Onset time	Duration of action
Lignocaine	2.9	7.7	65	Rapid	Medium
Bupivacaine	8.2	8.1	96	Slow	Longer
Ropivacaine	8	8.1	93	Slow	Longer

cardiotoxic and has a significantly higher threshold for central nervous system toxicity than bupivacaine.

Levobupivacaine : It is a pure S(-)-enantiomer of racemic bupivacaine. Because of findings that cardiotoxicity observed with racemic bupivacaine, although infrequent, is based on entantioselectivity, the S enantiomer, levobupivacaine, was developed for use as a long acting, local anaesthetic that shows reduced cardiotoxicity.[34]

Adjuvants

Adrenaline: It is commonly mixed with local anaesthetic solution to increase the intensity and the duration of block and minimize bleeding from small vessels.[35]. A concentration of 1:200,000 has no systemic effect.[36]

Hyaluronidase

It is an enzyme which reversibly liquefies the interstitial barrier between cells by depolymerization of hyaluronic acid to a tetrasaccharide, thereby enhancing the diffusion of molecules through tissue planes. It has been shown to improve the onset and enhance the quality of retrobulbar, peribulbar and sub-Tenon's block.[37, 38] Though varying amount of hyaluronidase (5–150 IU/ml) have been used by authors, it is better to limit the concentration to 15 IU/ml.[14]

pH value

Local anaesthetics are weak bases. At higher pH values, greater proportion of local anaesthetic molecules exist in the non-ionized form, allowing more rapid influx into the neuronal cells. Also, the nociceptor receptors are also less sensitive to the non-ionized form of the drug.[39] Thus, alkalinization has a proven impact in decreasing onset time, prolonging the duration of action and also decreasing the pain experienced by the patient during injection of the local anaesthetic.[40]

Do's and Don'ts for a safe ophthalmic regional anaesthesia

For a safe and effective ophthalmic regional anaesthesia, the following are some of the important practical points to be remembered.

- Bevel should face the globe, to reduce the chance of snagging of the globe.
- Eye should be in primary gaze position, to prevent any iatrogenic trauma to optic nerve.
- Aspirate before injecting local anaesthetic solution.
- Injection should be done slowly, to decrease the pain perceived by patients during injection.
- Always feel the tension of the globe with fingers of the non-blocking hands.
- Stop injecting the local anaesthetic solution when there is any atypical pain, any resistance felt or if there is any abnormal movement of the globe seen during injection.
- Always withdraw the needle along the line of insertion.
- Injection at superomedial quadrant should not be administered. Superomedial quadrant is more vascular in nature when compared with the remaining other three quadrants resulting in more chances of haemorrhage to occur in the lid and as the globe is closer to the roof than to the floor, superomedial block *per se* can result in perforation of the globe.[41]
- Once the local anaesthetic has been injected the most important thing do be done immediately is the gentle digital ocular massage for a period of 2 min.
- Assess the block before giving any supplementary injections.

Summary

Regional blocks provide excellent anaesthesia for eye surgery and success rates are high. Although rare, orbital injections may cause severe local and systemic complications. A thorough knowledge of the orbital anatomy and training are essential for the practice of safe ophthalmic regional anaesthesia.

Given the choices for ocular anaesthesia today, no single mode of anaesthesia can serve as a universal choice for all patients and all surgeons. The literature reveals that each of the major modes of ocular anaesthesia—retrobulbar, peribulbar, sub-Tenon's and topical are essentially equally effective in controlling patient pain and allowing a surgeon to have a successful surgical outcome. The choice of the technique should be individualized-based upon specific needs of the patient, the nature and extent of eye surgery, and the anaesthesiologist's and surgeon's preferences and skills.

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Electrophysiology in Neurophthalmology

Parveen Sen

Department of Vitreoretinal Diseases, Sankara Nethralaya, Chennai, India

Correspondence to:
Parveen Sen,
Senior Consultant,
Department of Vitreoretinal Diseases,
Sankara Nethralaya,
Chennai, India,
email: drpka@snmail.org

Optic nerve disorders have varied clinical manifestations and neurophthalmologists depend on multiple investigations including electrophysiology to reach at a correct diagnosis. Electrophysiological tests not only help in differential diagnosis but also in follow-up of many of these neuropathies. The most commonly used electrophysiological test in neurophthalmology setting is the visual evoked potential (VEP). The newer tests that are also being increasingly used include pattern electroretinogram (PERG), multifocal VEPs (mfVEP) and multifocal ERG (mfERG).

Here, we try to outline the role of these investigations in a clinical setting. Full-field ERG is the most commonly used electrophysiological investigation by ophthalmologists. However, it does not comment on the function of the ganglion cells and the optic nerve. VEP evaluates the response of the visual system to light. The generator site of VEP is at the peristriate and the striate occipital cortex. This response can be in response to a bright flash of light (flash VEP) or in response to a pattern (pattern VEP). Flash VEP is commonly used to assess the visual status in subjects with poor visual acuity, in opaque media or in infants and children. It may help to prognosticate the outcome in these situations by grossly picking up the optic nerve function. Pattern VEP may be in response to pattern reversal or pattern onset and offset. Pattern reversal VEP is used more commonly because its results are more reproducible. Indications of pattern reversal VEP include optic neuritis, multiple sclerosis, compressive optic nerve disease, unexplained visual loss, amblyopia, cortical blindness, traumatic optic neuropathy and measuring visual acuity in non-cooperative individuals. Pattern onset/offset VEP is preferred over pattern reversal in subjects malingering a loss of vision and in patients with significant nystagmus.

Interpretation of VEP

Flash VEP

The most important parameter in the analysis of flash VEP is the P2 latency and amplitude. Since high degree of intersubject variability is seen in flash VEP parameters in the normal population, an interocular comparison may be more informative.

Pattern VEP

On pattern reversal VEP P100 is the most important parameter for analysis because of its narrow latency range in normal subjects. Pattern VEP may be more sensitive in picking up an optic nerve disorder in certain situations and is useful even in chronic optic neuritis when the MRI has become normal. Also, it is more economical for the patient especially when repeated investigations are required to know the progress of the disease.

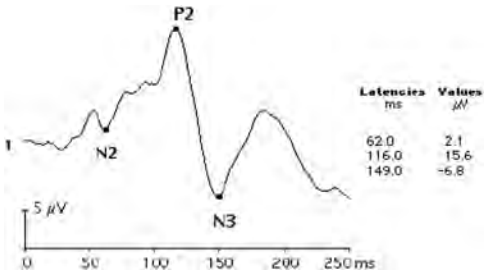

Figure 1a Flash VEP with Normal P2 latency and amplitud.

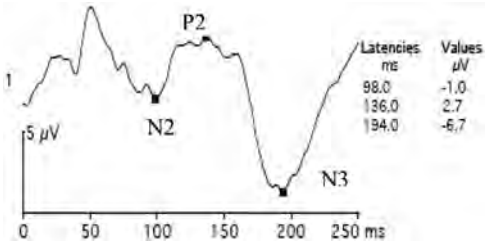

Figure 1b Flash VEP with Delayed P2 latency with reduced amplitude.

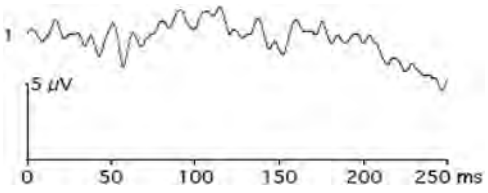

Figure 1c Non-recordable flash VEP.

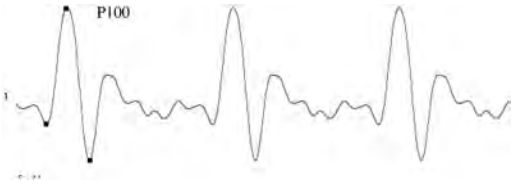

Figure 2a Typical pattern VEP waveforms seen in normal subjects.

Salient features on VEP in the commonly seen optic nerve disorders are tabulated below.

S. No.	Optic neuritis	ION	Compressive lesions
1	Marked latency delay	Latency delay not seen	Latency delay seen
2	Amplitude reduction seen which recovers	Predominant decrease in amplitude	Amplitude reduction seen
3	Waveform morphology maintained	Waveform morphology maintained	Distortion of the waveform seen
4	Changes in scalp distribution uncommon	Changes in scalp distribution uncommon	Abnormal scalp distribution seen
5	VEP changes seen during the course of the disease	Monophasic	VEP changes seen during the course of the disease

Flash and pattern VEP responses in optic neuritis
Please refer Figure 3.

A normal VEP practically rules out an optic nerve disease anterior to the chiasma. However, chiasmal and retrochiasmal disorders may be missed until multichannel VEP recording is done. Also, conventional VEP elicits global response and hence is unable to detect subtle or local pathology. It can be influenced by macular pathology as well since the majority of optic nerve fibers are from the macula.

To differentiate between the reduced VEP because of optic nerve disease or macular disorder newer investigation protocol like the pattern ERG is being increasingly used. PERG is a retinal biopotential that is produced when a stimulus pattern of constant mean luminance is viewed. Transient PERG has two components: P50 (positive component appearing at 50 ms) which shows macular function and N95 (larger negative component appearing at 95 ms) which shows ganglion cell function. In some patients, an early small negative wave called the N35 is also seen. PERG, however, has lower amplitude than ERG and so special recording techniques need to be used to differentiate it from noise. Also, fixation is critical for a good PERG record; hence, it cannot be used in patients with poor visual acuity.

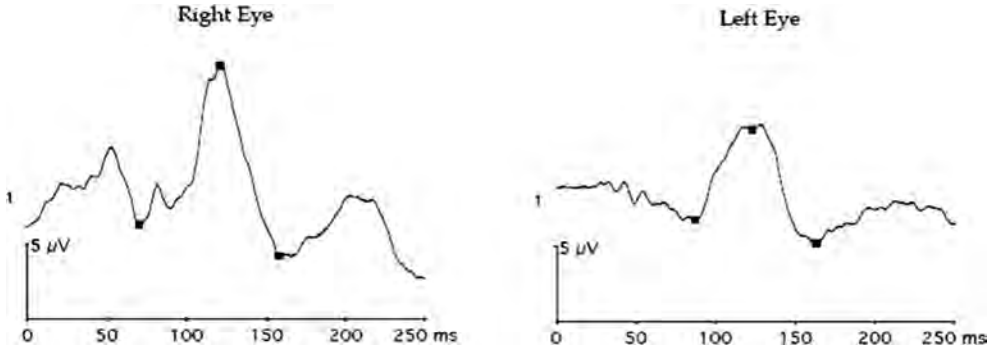

Figure 3a Flash VEP showing delayed P2 component in the left eye.

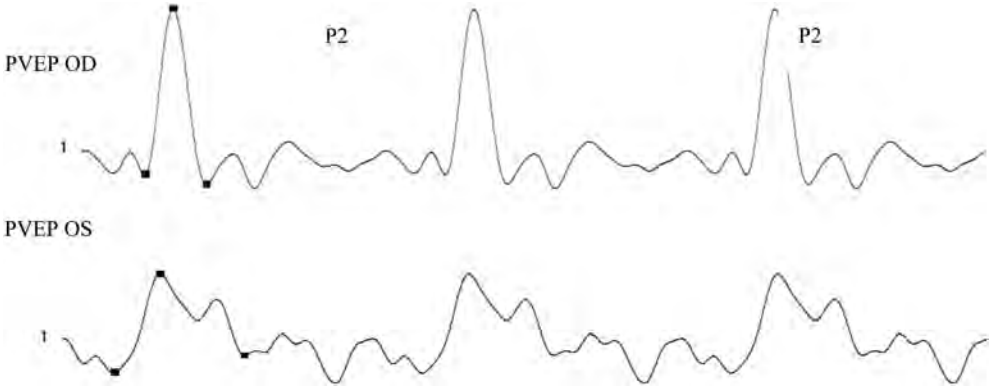

Figure 3b Pattern VEP in optic neuritis: The right eye shows PVEP with normal latency and amplitudes whereas the left eye reveals markedly delayed waveform with reduced amplitude.

VEP responses in ischemic optic neuropathy: Please refer Figure 4.

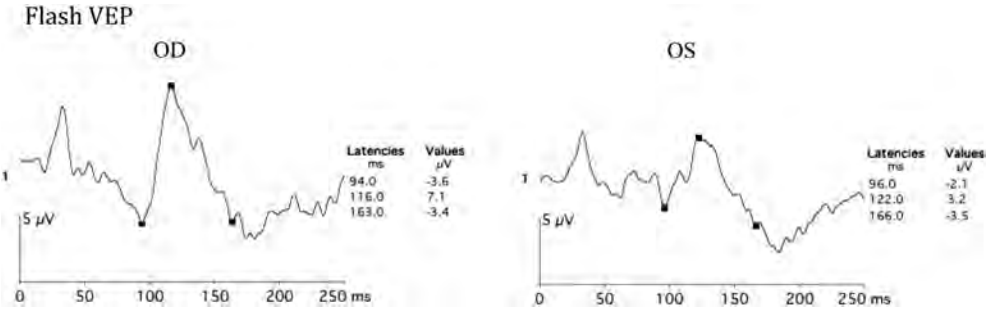

Figure 4a Flash VEP showing decrease in amplitudes in OS.

Figure 4b PVEP showing a predominant decrease in amplitudes in a subject with ischemic optic neuropathy of the left eye.

Flash and pattern VEP responses in compressive optic neuropathy: Please refer Figure 5.

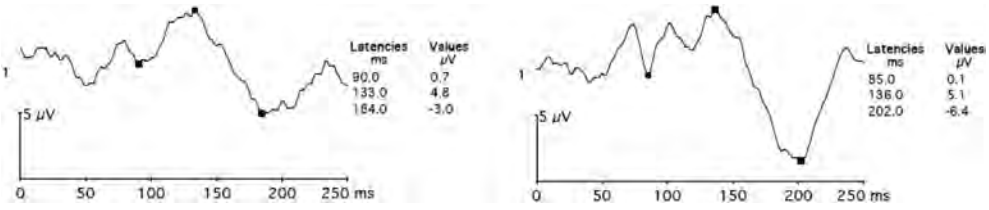

Figure 5a Flash VEP in 27-year-old female with bitemporal hemianopia; MRI revealed a Pitutary adenoma.

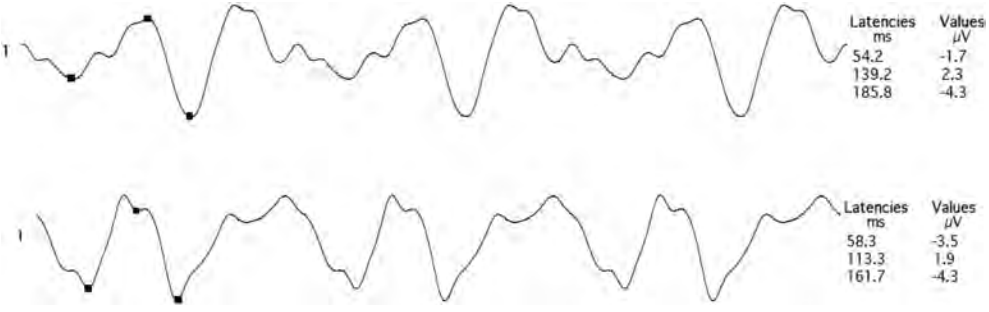

Figure 5b Pattern VEP of the same subject. OD reveals a distorted waveform reduced amplitude (gross RAPD with optic disc pallor was seen in right eye).

Flash VEP responses in traumatic optic neuropathy: Please refer Figure 6.

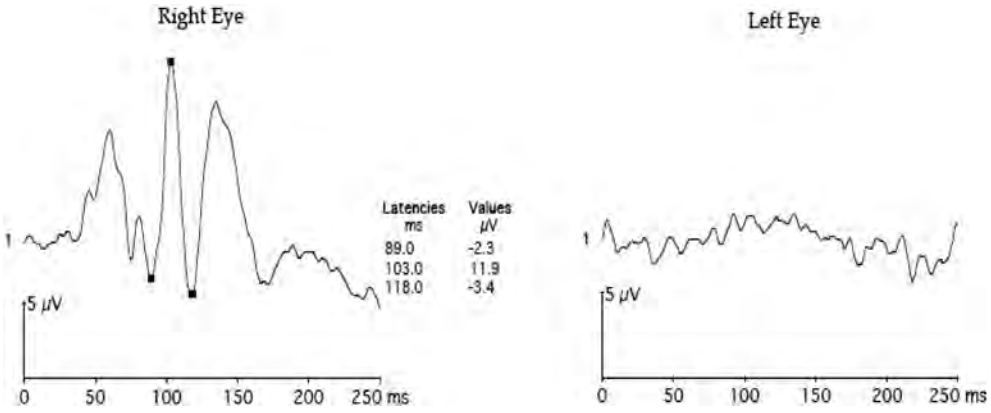

Figure 6 Flash VEP shows normal waveform in the right eye while the left eye flash VEP is non-recordable.

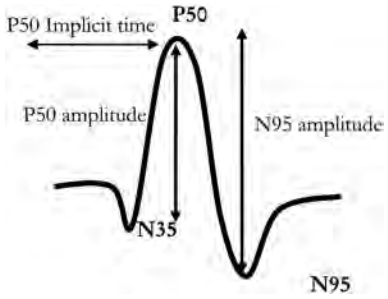

Figure 7 A normal PERG waveform.

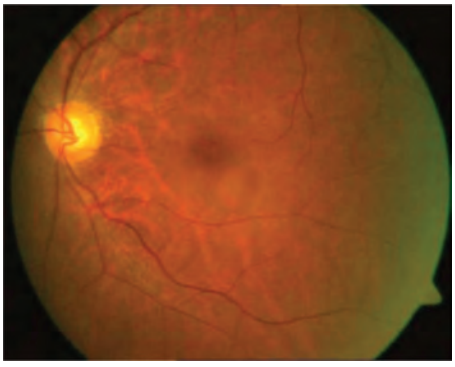

Figure 8a Fundus photograph showing mild temporal pallor.

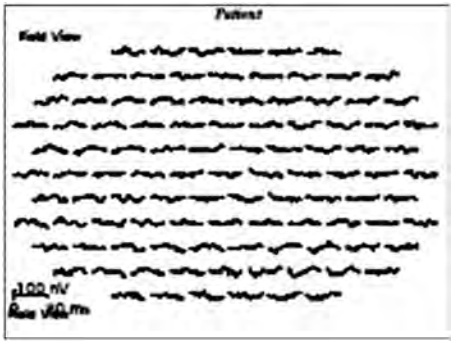

Figure 8b Reduced mfERG amplitude in all the Rings suggestive of a cone dystrophy.

P50 component of PERG which reflects the macular function is complementary to full-field ERG. Normal ERG with a decrease in the P50 amplitude depicts macular dysfunction while an abnormal ERG with an abnormal PERG is a pointer towards a generalized retinal disorder. Extinct PERG may occur in macular dysfunction but is rarely seen in optic nerve dysfunction. Selective affection of the N95 component with a near normal P50 points towards optic nerve pathology. N95/P50 ratio could also be very useful in differentiating between the macular and optic nerve dysfunction. It remains unaltered in macular disease but decreases in optic nerve disorders. Acute phase of optic neuritis shows a loss of visual acuity; hence, PERG waveforms can be non-recordable. In chronic phase of optic neuritis P50 recovers while N95 abnormality persists with significant reduction in the N95:P50 ratio. Though PERG can differentiate between a retinal and an optic nerve disorder it does not give us the topography of the disease process.

For the topography of retinal and optic nerve disorders, multifocal techniques like the mfVEP and the mfERG are used. Most important application of mfERG in neurophthalmology is to rule out macular pathology as a cause of poor vision. A near normal fundus with a poor amplitude chart on mfERG and an abnormal VEP suggests macular pathology as the cause of poor vision.

A mild temporal pallor of the discs with near non-recordable waveforms on mfERG across the plot points towards a cone dysfunction rather than optic neuritis.

mfVEP can be particularly useful in picking up distribution of the optic nerve dysfunction and

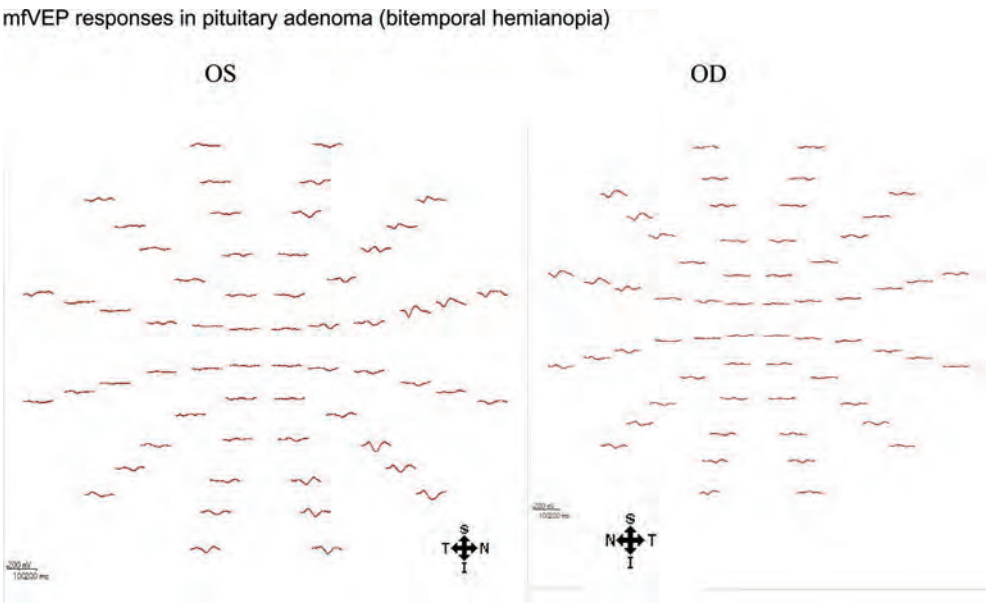

Figure 9 MfVEP responses showing non-recordable temporal field waveforms in a 48-year female presented with bilateral gradual progressive decrease in vision and difficulty in seeing temporal field of vision. Her BCVA was 6/7.5 in right eye and 6/18 in left eye. The fundus examination revealed bilateral disc pallor. Visual field examination revealed bitemporal hemianopia and MRI imaging revealed pituitary adenoma.

may be a pointer to the underlying pathology. For example, an altitudinal defect seen on mfVEP points towards an AION. MfVEP follows the visual fields but can also be effectively done in some subjects not cooperative for HVF 30-2 and in children. Just like macular pathology can affect the VEP, retinal disorders can also affect mfVEP.

We have seen that no single test can give us the complete information and one has to rely on a combination of investigations to reach a diagnosis. Even though newer and better imaging techniques particularly MRI may have limited the use of electrophysiology in clinical setting, these tests still give useful information about the physiology of disease whereas the imaging modalities essentially give information on the structural damage. In most of the circumstances, these could be complimentary to patient care if intelligently used.

Suggested reading:

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How to Write a Case Report

Bikramjit P. Pal[1] and Jyotirmay Biswas[2]

[1]Senior Resident, Department of Vitreoretinal Diseases, Sankara Nethralaya, Chennai, India
[2]Director, Ocular Pathology and Uvea, Sankara Nethralaya, Chennai, India

Correspondence to:
Bikramjit P. Pal, Senior Resident, Department of Vitreoretinal Diseases, Sankara Nethralaya, Chennai 600006, India, email: vrnuts@gmail.com

"There are two kinds of writers: those that make you think, and those that make you wonder"
Brian Aldiss

Introduction

Case reports are no different from writing a good story: the only difference between a good and a bad one is how well it is narrated. Case reports are the oldest documented form of medical communication and the first line of evidence in health care.[1] In recent times, the values of case reports have dwindled. Some citing case reports to be only anecdotal evidence while others argue it to have a low level of general application, but the value of a well-researched and a well written case report cannot be undermined. Preparing and writing a case report not only helps in the development of ones thought process but also stimulates research. The aim of this article is to provide the budding researchers and clinicians with a basic idea of writing a well-structured case report.

When to write a case report

Rarity itself is an insufficient ground for publication. Case report is an important platform for providing new ideas and knowledge which adds to the already existing understanding of a clinical condition. Case reports can be considered in the following scenarios:[2, 3]

1 To report an unusual or an unknown clinical entity.

2 To illustrate unusual etiology for a case.

3 To provide new insights into pathogenesis of a disease.

4 To report a unique or rare features observed during imaging.

5 To report a novel therapeutic or interventional technique.

6 To describe and report new adverse reactions to an drug already in use.

7 To make readers aware of an new complication of an therapeutic procedure in vogue.

A thorough literature search is essential before writing a case report. One may not report a case if it has been reported earlier unless there is something new to offer. Literature search forms a major backbone of any research and hence ample amount of time should be spent in doing so.

How to write a case report

The basic structure of a case report is no different from writing any other scientific article. The following paragraphs deals with the headings under which a case report should be prepared.

(1) Title

Ideally a title should be short, descriptive yet catchy. It should be informative enough to hold the interest of the reader.

(2) Abstract

An abstract is a gist of the entire case report and hence should contain the most important and salient features. Only the key results obtained should be highlighted. The abstract should summarize as to how it contributes to the medical literature and should end with a clear take home message.

(3) Introduction

As the name implies "Introduction" introduces the readers to the topic to be discussed. It begins by describing the condition in short while providing salient features of the condition already known. A brief review of literature pertaining to the case should be presented. The introduction should always end with the author's citing the purpose of documenting and reporting thecase.

(4) Describing the case

In this section, the case is presented in a chronological order. The description should include all the major positive history and clinical findings, while also highlighting the significant negative results. The imaging and the various diagnostic tests used should be clearly explained. The description of the case should end with the authors presenting their clinical diagnosis. The author's inferences should not be included in this section. The most important thing while preparing a case report is "Patient Confidentiality". At no point should the details of the patient leading to their identification be divulged.

(5) Discussion

This part of case report involves presenting the author's point of view. The purpose is to explain "how" and "why" certain inferences were made. These should be then compared with previously cited similar literature and points which are

unique be highlighted. In short discussion involves revealing "what is known" and presenting "what is new" in the context of the case. Discussion should also provide readers with a logical management option and an insight into how it might be helpful as a future research topic.

(6) References

Articles which are peer reviewed are generally chosen as a reference. Relevant quotes from books may also be cited as a reference.

(7) Figures/tables/illustrations

Case reports become interesting if accompanied by self-explanatory tables and figures. A good quality clinical photograph is an asset to a well-structured case report.

(8) Authorship

According to the international committee of medical journal guidelines[4] one is considered an author if one has:

a provided substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data;

b drafted the article or revised it critically for important intellectual content;

c given final approval of the version to be published.

Anyone else who might have helped in the process of making the report can be acknowledged at the end of the report.

Where to publish

Ideally the decision of selecting the journal should be made at the start of the write up. A discussion with the concerned consultant and colleagues might help in choosing the ideal journal. Whichever journal is chosen it is important to follow the instructions and prepare it accordingly.

What if the journal rejects the case report

- Never get disheartened.
- A rejection is also a learning experience. Reviewer's comments can be used in a constructive way to make ones case report better.

Limitations and pitfalls of a case report

Although case reports are an invaluable tool, they do have their limitations.[2, 3, 5, 6] Since the clinician has no control over the patient's clinical settings as in a well-controlled study, the validity of the results obtained are often questioned. Case reports involve one or two patients and therefore the results cannot be generalized. Ideally a case report should provide something new rather than being merely a tool to boast ones Curriculum Vitae.

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Bacillus cereus

J. Malathi, K. Lily Therese and H.N. Madhavan

L&T Microbiology Research Centre,
Vision Research Foundation,
Chennai, India

Correspondence to: J. Malathi, L&T Microbiology Research Centre, Vision Research Foundation,
Chennai, India.
Email: drjm@snmail.org

INTRODUCTION

The genus *Bacillus* consists of aerobic bacilli which are Gram positive, large, rod-shaped bacteria, some are facultative aerobes. All the *Bacillus* species are capable of forming heat-resistant endospores. It is a soil-dwelling motile bacteria with low virulence[1] and is considered as an opportunistic pathogen in humans. It is the second most important *Bacillus* group of organisms capable of causing destructive, localized infections in humans, the first being *Bacillus anthracis*.

BACILLUS CEREUS AT A GLANCE

General characteristics of *B. cereus*

It is a soil organism, but the spores produced by them are present in the environment ubiquitously in dust, water and air. It grows well at temperature ranges of 15 °C–45 °C and is a facultative anaerobic organism. It produces well-marked beta-haemolytic grey colonies on sheep or horse blood agar, and the colony size is variable ranging from 2 to 7 mm in diameter, varying in shape from circle to undulate with crenate or fimbriate edges and matt or granular texture. The consistency of the colony is butyrous, which can be pulled as a string/standing peak with the inoculation loop.

Characteristic features of *B. cereus*

Bacillus cereus is large bacterium (the diameter of *B. cereus* is more than 1·0 µm in size with rounded or square ends). It is motile with peritrichous flagella. It is prone to be decolourized easily to appear as Gram variable or Gram-negative bacilli. The endospores produced by *B. cereus* are non-bulging, sub-terminal and ellipsoidal in shape. It is a non-fastidious organism, grows readily on ordinary/basal culture media. PEMBA (polymyxin B, egg yolk, mannitol, bromothymol blue agar), MEYP (mannitol, egg yolk, polymyxin B) and penicillin agar are used to selectively grow *B. cereus* from various specimens like food, water, rice or any food item that is suspected to be the source of infection.

The typical biochemical reactions for laboratory identification are: it ferments glucose trehalose, salicin, starch, and glycogen with only acid. Voges Proskauer test is positive. It produces lecithinase enzyme demonstrated by growing it on egg yolk agar (broad opaque deposit around the colonies). In addition, it is capable of hydrolyzing casein, gelatine and starch. It is characteristically resistant to penicillin, ampicillin and cephalosporin, but sensitive to gentamicin and other aminoglycosides like vancomycin and also to clindamycin and chloramphenicol.

Genome

The molecular studies conducted have given valuable information into genetic profiles of *B. cereus* group. Genome analysis shows that the organism is closely associated with *Bacillus anthracis*.[2] The full genome of *B. cereus* has been sequenced in the year 2002. There are six genotypes of *B. cereus*. The genome has 5,411,809 nucleotides in length and has a circular chromosome. It comprises 5481 genes, 5234 protein coding, 147 structural RNAs and 5366 RNA operons.

Infections

Bacillus cereus is an opportunistic human pathogen.[1] It is primarily a food poison causing opportunistic pathogen with production of two types of enterotoxins. Immunocompromised patients are susceptible to bacteraemia, endocarditis, meningitis, pneumonia and endophthalmitis.[3] Its potential to cause systemic infections are of current public health and biomedical concerns.[3] *Bacillus cereus* is an important opportunistic pathogen associated with ocular infection, especially in traumatic endophthalmitis, introduced into the eye through foreign bodies.

Virulent factors involved in pathogenic mechanisms

The important virulent genes present in the genome are those that code for non-haemolytic enterotoxins, channel-forming type III haemolysins, phospholipase C, a perfringolysin O (listeriolysin O) and extracellular proteases. Food poison causing enterotoxins are produced by the *hbl* operon, an RNA transcript of 5·5 kb. The gene is also required for virulence of *B. cereus* and is often targeted for newer drug design.

Bacillus cereus-associated ocular infections

Bacillus cereus can cause ocular infections such as keratitis, endophthalmitis[4–9] (Figure 1A and B) and panophthalmitis. It is the most common agent causing traumatic endophthalmitis and the source is the soil. The main virulence factor in *B. cereus* endophthalmitis is haemolysin BL (HBL) which can result in the detachment of the retina and blindness. This ocular pathogen causes a rapid, fulminant endophthalmitis that invariably leads to blindness within 1 or 2 days. Despite aggressive antibiotic and surgical intervention, *B. cereus* endophthalmitis has a relatively poor prognosis as there is no universal

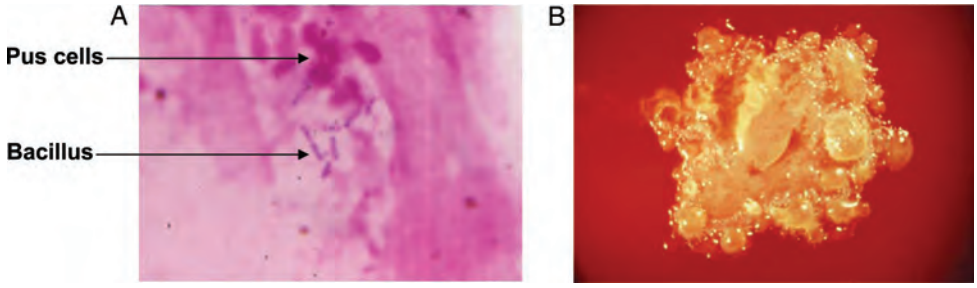

Figure 1 (A) Gram stain smear of the vitreous aspirate from a case of post surgical endophthalmitis (100x magnification under bright field microscopy. (B) Growth of the bacterial colony on blood agar of the vitreous aspirate from the same case (*Bacillus cereus*).

therapeutic regimen available for successful treatment of *B. cereus* endophthalmitis.

Clinicians and researchers have attributed the virulence of *B. cereus* endophthalmitis to toxin production. *Bacillus cereus* being motile migrates throughout the eye in a short period of time, inciting an explosive intraocular inflammatory response. This inflammatory response is likely the result of breakdown of the protective blood ocular barrier in response to infection, the triggers of which are presently being investigated. In addition, during infection, retinal architecture collapses and retinal function drops precipitously. Either *Bacillus* or its toxins (or both) target specific cells in the retina that are involved in protection by the blood ocular barrier (retinal pigment epithelial [RPE] cells) or retinal function itself (Muller cells, photoreceptor cells), leading to the detrimental effects observed during infection. Since the ultimate goal of therapy is to kill offending organisms, arrest inflammation and preserve organ function, an important goal is to develop more effective therapeutic regimens, with newer agents targeting virulence agents to prevent blindness.

Components of the Bacillus cell wall incite intraocular inflammation in sterile endophthalmitis models, but do not affect retinal function. Individual bacterial cell wall-associated constituents (i.e. peptidoglycan, teichoic acid, capsules, S-layer) are being analysed as inducers of acute intraocular inflammation. Bacterial component recognition by retinal cells is being analysed using toll-like receptor knockout mice. It is well established that *B. cereus* elaborates a host of tissue-destructive exotoxins that contribute to the devastating outcomes in endophthalmitis. However, recent investigations into the pathogenesis of *B. cereus*-induced endophthalmitis have identified several other factors that also contribute to the poor outcome of *B. cereus* endophthalmitis. Initially, Beecher et al.[7] suggested that the poor outcome of antibiotic treatment of *B. cereus* endophthalmitis was actually a consequence of continued tissue-destructive activity independent of antibiotic bacterial killing.

Among the elaborated exotoxins incriminated in an experimental rabbit model of destructive endophthalmitis conducted by Beecher et al.[7] were hemolysin BL (a tripartite dermonecrotic vascular permeability factor), a crude exotoxin (CET) derived from cell-free *B. cereus* culture filtrates, phosphatidylcholine-preferring phospholipase C (PC-PLC) and collagenase. The contribution of these factors individually or in concert could account for retinal toxicity, necrosis and blindness in experimentally infected rabbit eyes. The toxicity of PC-PLC was a direct result of the propensity of the secreted enzyme for the phospholipids in retinal tissue, which may also act similarly in human eye retinal tissue, which also contains a significant amount of phospholipids. In a separate study, Callegan et al.[8] showed that the role of BL toxin in intraocular *B. cereus* infection was minimal, 'making a detectable contribution only very early in experimental *B. cereus* endophthalmitis but did not affect the overall course of infection'. Callegan et al.[8] concluded that *B. cereus* endophthalmitis followed a more rapid and virulent course than *S. aureus* and *E. faecalis*, the other two bacterial species. Additionally, *B. cereus* intraocular growth was significantly greater than those of *S. aureus* and *E. faecalis*. Analysis of bacterial location within the eye showed that the motile *B. cereus* rapidly migrates from posterior-to-anterior segments during infection.

This phenomenon was confirmed in a subsequent study using wild-type motile and non-motile *B. cereus* strains, which confirmed that while both strains grew to a similar number in the vitreous fluid, the motile swarming strain migrated to the anterior segment during infection, causing more severe anterior segment disease than the non-swarming strain. Bacterial swarming a specialized form of surface translocation undertaken by flagellated species similar to that exhibited by *Bacillus cereus*. Swarm cells in a population undergo a morphological differentiation from short bacillary forms to filamentous, multinucleate and hyperflagellated swarm cells with nucleoids evenly distributed along the lengths of the filaments. The differentiated cells do not replicate, but rapidly migrate away from the colony in organized groups, which comprise the advancing rim

of growing colonies. Swarming is thought to be a mechanism by which flagellated microorganisms traverse environmental niches or colonize host mucosal surfaces. Moreover, swarming can play a role in host pathogen interactions by leading to an increase in production of specific virulence factors. Ghelardi et al.[9] showed a correlation between swarming and haemolysin BL secretion in a collection of 42 *B. cereus* isolates.

Laboratory diagnosis of *Bacillus cereus*

The organism is identified in the laboratory by their typical morphology Gram-positive reaction, characteristic large broad thick rods with square or rounded ends forming short chains and turbid growth in liquid media. The organism is cultured in 6·5% sodium chloride broth and fermentation of glucose only with acid. The confirmatory and differential identification from *B. anthracis* are the broad zone of beta-haemolytic colonies on sheep blood agar, its ability to produce lecithinase enzyme in the egg yolk medium within 6 h of incubation, motility and resistance to penicillin and cephalosporin group of drugs.

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Erratum

INSIGHT, Year: 2012, Volume: XXX Issue: 3 Page: 40–43

Title: An interesting case of blepharoptosis
Author: Bipasha Mukherjee

Should be read as

Title: An interesting case of blepharoptosis
Authors: V. Akila Ramkumar and Bipasha Mukherjee

The error is regretted

Editor,
Insight

Isolated Necrobiotic Xanthogranuloma: A Rare Clinical Entity

Bipasha Mukherjee[1], Puja Goyal[1], S. Krishnakumar[2] and Jyotirmay Biswas[2]

[1]Department of Orbit, Oculoplasty, Reconstructive and Aesthetic Services, Sankara Nethralaya, Medical Research Foundation, Chennai, India
[2]Department of Ocular Pathology, Sankara Nethralaya, Medical Research Foundation, Chennai, India

Correspondence to: Bipasha Mukherjee, Department of Orbit, Oculoplasty, Reconstructive and Aesthetic Services, Sankara Nethralaya, Medical Research Foundation, Chennai, India, email: drbpm@snmail.org

INTRODUCTION

Xanthogranuloma is a slowly progressive histiocytic disease that is associated with paraproteinemia, commonly monoclonal gammopathy, in 80% of patients.[1] It includes four clinical syndromes: adult-onset xanthogranuloma; necrobiotic xanthogranuloma (NBX); adult-onset asthma with periocular xanthogranuloma and Erdheim-Chester disease. Ophthalmic manifestations affect approximately 50% of cases and include orbital masses, conjunctival involvement, keratitis, scleritis and uveitis.[2, 3] Systemic associations include organomegaly, diabetes mellitus, hyperlipidemia, blood dyscrasias, multiple myeloma, non-Hodgkin's lymphoma and asthma.[4]

CASE REPORT

A 32-year-old middle east Asian male reported with complaints of ocular discomfort, redness and diminution of vision in his left eye since one and a half years. On examination, the best corrected visual acuity was 6/6; N6 and 6/9; N6 in the right and left eye, respectively. The intraocular pressure was 10 mmHg in both eyes. Extraocular movements were full in all gazes in both eyes. There was no visible skin abnormality. On examination, the left globe was displaced laterally. A raised globular, salmon-colored, well defined, subconjunctival lesion was present in the superomedial quadrant of the left orbit. The posterior extent of the lesion could not be visualized. Palpebral fissure was reduced by 1 mm. On palpation, the orbital margins were continuous. The mass was firm and fingers could be insinuated between the orbital margin and the lesion. There was no regional lymphadenopathy. Anterior segment examination was normal. Posterior segment examination revealed a few choroidal folds in the superonasal quadrant of the left eye.

Magnetic resonance imaging (MRI) of the orbit revealed a 18 × 6.8 mm well defined, homogenous dense lesion in the superomedial orbit, predominantly intraconal in location extending from posterior to the insertions of the medial and superior recti upto the trochlea medially. The choroid was indented. Postcontrast study showed homogenous moderate contrast enhancement [Figure 1(a)–(c)].

Hematological investigations like total count, differential blood counts and erythrocyte sedimentation rate were normal. Blood urea and serum creatinine were normal. The patient underwent an uneventful medial orbitotomy by the transconjunctival approach. A well-defined yellowish mass lesion was removed. Histopathological examination of the lesion showed multiple lymphoid follicles separated by fibro-collagenous bundles with foci of necrosis [Figure 2(a) and (b)]. The necrotic areas were surrounded by histiocytes, foamy histiocytes, Langhan's type and Touton giant cell. The lymphoid follicles were attempting to form germinal centers. There were scattered plasma cells and few blood vessels. The diagnosis of NBX was made from the typical histopathology findings. Adjunctive tests were ordered to rule out any systemic associations. Ultrasound of neck and

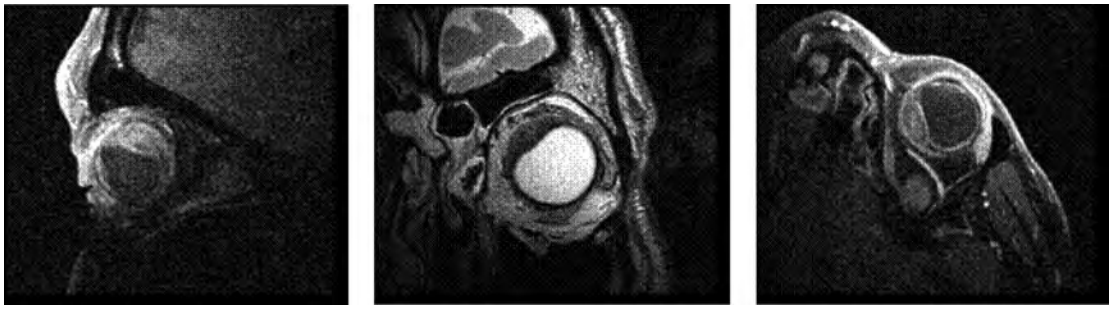

Figure 1 (a) Showing para sagittal, (b) coronal view and (c) axial view of MRI orbit. A well-defined, homogenous predominantly intraconal lesion extending from posterior to the insertions of the medial and superior recti extending upto the trochlea. Post contrast showed homogenous moderate contrast enhancement.

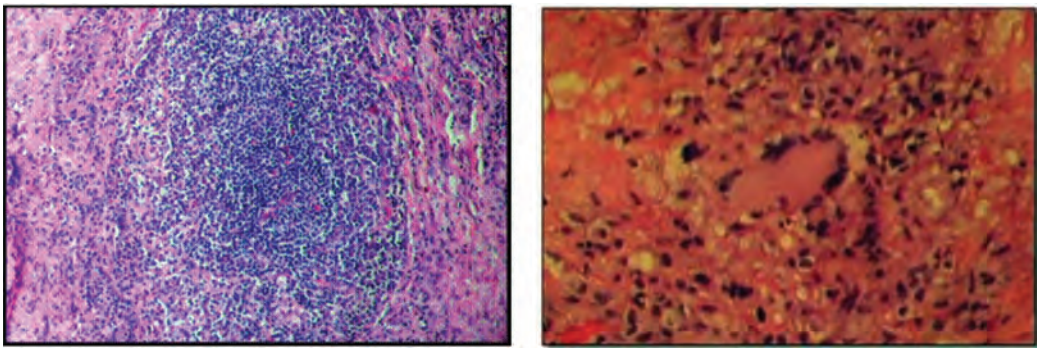

Figure 2 (a) Hematoxylin and eosin mount showing multiple lymphoid follicles separated with fibro collagenous bundles with foci of necrosis (100× magnification). (b) The necrotic areas are surrounded by histiocytes, foamy histiocytes, Langhan's type and Touton giant cell (400× magnification).

abdomen, computerized tomography scan of the chest were normal. Serum protein electrophoresis by slide electrophoresis method revealed normal pattern.

The patient was started on a tapering course of oral steroids and advised the need for close follow up. The patient has been recurrence free without development of any systemic associations up to 1 year of follow up.

DISCUSSION

NXG was first recognized as a discrete clinical entity by Kossard and Winkelmann in 1980.[1] It is a rare histiocytic disease characterized by indurated, non-tender, dermal, or subcutaneous yellow nodules and plaques that primarily infiltrate the eyelids, periorbital structures, flexural extremities and trunk. Ophthalmic findings include subcutaneous eyelid nodules and plaques, episcleritis, uveitis, iritis, keratitis, cellulites and proptosis.[2] Systemic findings include hepatosplenomegaly, monoclonal gammopathy in 80% patients, neutropenia, hypocomplementemia, cryoglobulinemia, hyperlipidemia, increased erythrocyte sedimentation rate and leucopenia.[1, 3]

Malignancies such as multiple myeloma and non-Hodgkin's lymphoma are known to be associated in particular with the necrobiotic form.[1–3] Thus, it is important to diagnose this clinical entity as the potentially fatal systemic associations warrant emergent treatment.

The histopathological features of NBX are typical showing sheets of histiocytes, plasma cells, lymphocytes and giant cells separated by fibrocollagenous fibers with variable amounts of necrobiosis. Both Touton and foreign-body giant cells are seen. The Touton giant cells are multinucleate with central eosinophilic cytoplasm with a rim of foamy cytoplasm surrounding the nuclei [Figure 2 (b); center]. Lymphoid nodules with germinal centers are frequently seen.

Therapeutic options in a case of NXG include surgical debulking, radiotherapy, oral and periocular steroids and cytotoxic agents (such as chlorambucil, nitrogen mustard, cyclophosphamide and melphalan). However, the response has been variable.[1–3] The treatment modalities tried in this rare disease have been surgery, radiotherapy, immunosuppressives, steroids and combinations of the above.[4, 5] Our patient underwent surgical excision of the mass lesion followed by systemic steroids which seemed to have been curative.

NBX with systemic associations have been widely reported in the literature. Onset is typically between 50 and 60 years, with an equal incidence in men and women. Our patient was a young male in the third decade of life with isolated NXG of the orbit unassociated with any systemic, biochemical or hematological abnormalities.

Recurrence is known with xanthogranuloma and systemic features may develop in the course of the disease. Hence, careful follow up and regular systemic evaluation is mandatory.

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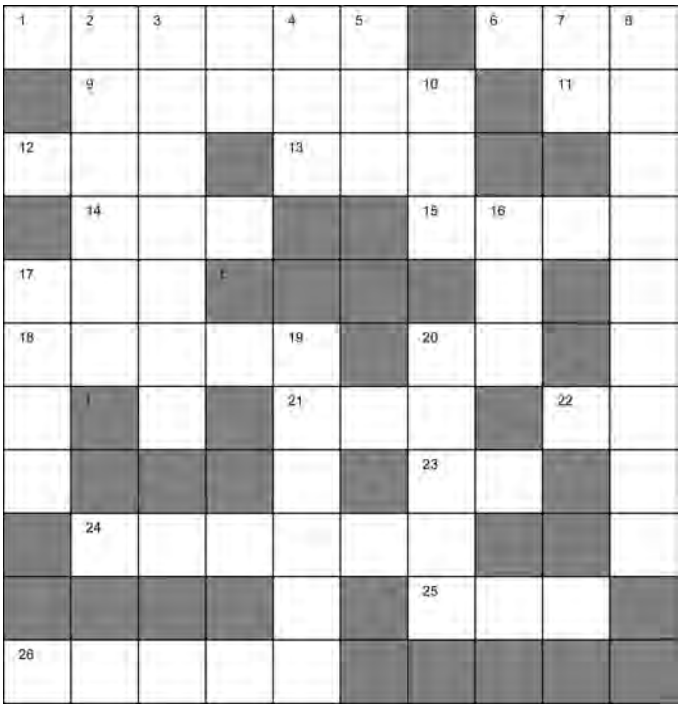
Crossword

Parthopratim Dutta Majumder

Correspondence to:
Parthopratim Dutta Majumder,
Associate consultant,
Department of Uvea,
Sankara Nethralaya, Chennai.
Email: drparthopratim@gmail.com



Across :

1. Name of the desert associated with LASIK surgery
6. Innermost layer of the meninges,
9. One of the important cause of CNVM in young patient
11. Genetic inheritance of Stargardt disease (abbreviation)
12. Genome of Chikungunya virus
13. An electrophysiological test
14. Innermost boundary of the retina, composed of terminations of Müller cells (abbreviation)
15. Jacques Daviel, a French ophthalmologist, was known to perform first this type of cataract surgery
17. Silicone oil insertion is often abbreviated as
18. Nuclear layer of retina, where dot haemorrhages occur
20. Goldman is associated with this procedure (abbreviation)
21. This organ is often involved in Vogt–Koyanagi–Harada syndrome
22. World's most widely used system of measurement (abbreviation)
23. A qualification in ophthalmology
24. Thin vertical streaks in the posterior corneal stroma, seen in keratoconus and named after Vogt.
25. Greek word for young, often used in vascular pathophysiology
26. Most sensitive part of the retina.

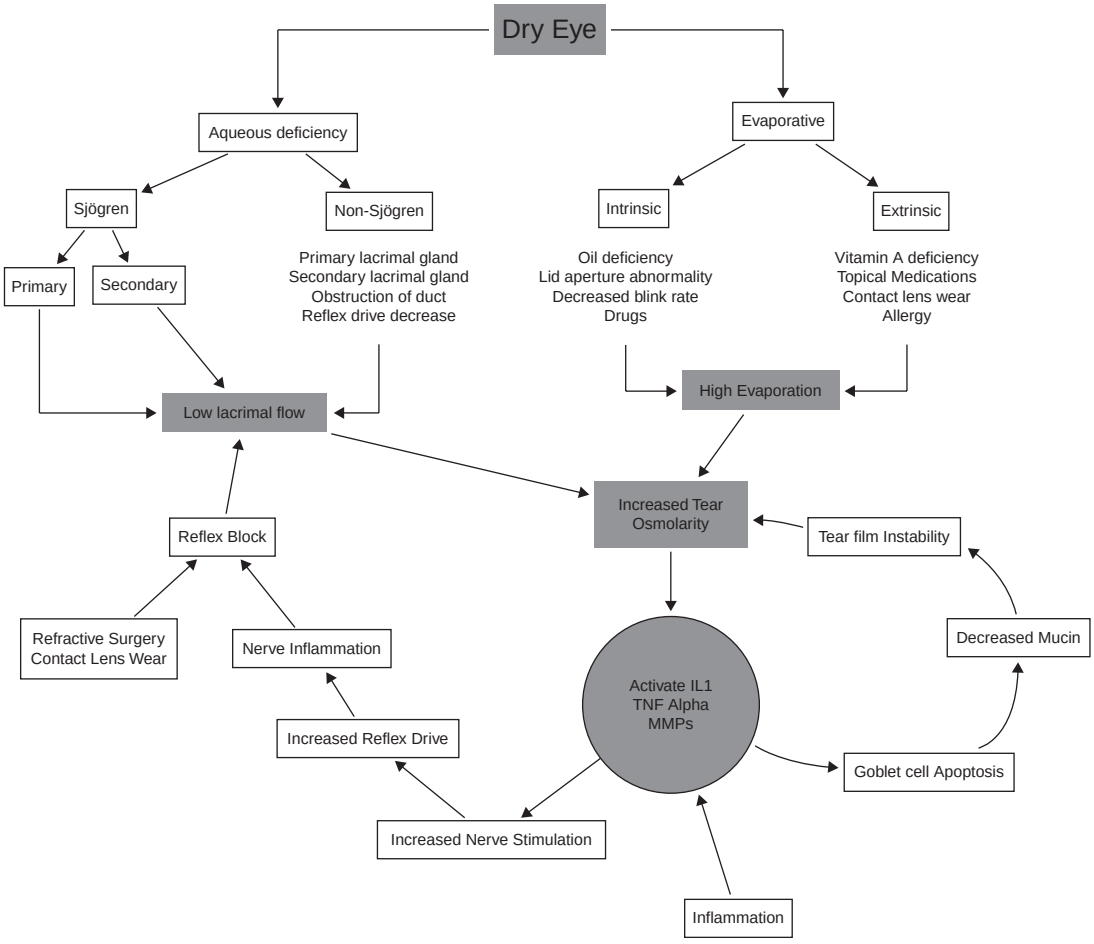
Down:

2. Innermost layer of placenta, used in various reconstructive surgeries in ophthalmology
3. A transparent, glassy substance seen in cartilages and deposited in arteriosclerosis
4. Single-cell thick, pigmented outermost layer of retina (abbreviation)
5. Anwar's big bubble technique uses this to separate descemet's membrane from corneal stroma
8. Flag of this country is seen as a complication in surgery of total cataract
10. Most important risk factor for glaucoma
16. White eye reflex in children and an animal
17. Dreaded form of scleritis seen after surgical procedures (abbreviation)
19. Name of this structure of eye is derived from a Latin word meaning 'net'
20. Ratio used in electrophysiology.

Pathophysiology of Dry Eye

Ashwin Mohan

Correspondence to:
Ashwin Mohan,
Post graduate student,
Sankara Nethralaya,
Chennai,
email: drashwinm@gmail.com



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Crossword: Answer

1 S	2 A	3 H	A	4 R	5 A	■	6 P	7 I	8 A
■	9 M	Y	O	P	I	10 A	■	11 A	R
12 R	N	A	■	13 E	R	G	■	■	G
■	14 I	L	M	■	■	15 E	16 C	C	E
17 S	O	I	L	■	■	■	A	■	N
18 I	N	N	E	19 R	■	20 A	T	■	T
N	I	E	■	21 E	A	R	■	22 S	I
S	■	■	■	T	■	23 D	O	■	N
■	24 S	T	R	I	A	E	■	■	A
■	■	■	■	N	■	25 N	E	O	■
26 F	O	V	E	A	■	■	■	■	■

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Choroidal Tuberculoma

Karpagam Damodaran

Address for Correspondence: Karpagam Damodaran, Senior Resident, Department of uvea & intraocular inflammation, Sankara Nethralaya, Chennai.
Email: karpagamdamodaran@gmail.com

A 30 year old female, pathologist by profession, presented with complaints of diminution of vision in the left eye of a week's duration. Her right eye was asymptomatic.

Her past history was significant for endobronchial tuberculosis, and had been treated for the same with a six month course of antitubercular treatment, four drug regimen, and 5years back.

Her best corrected visual acuity using Snellens Visual acuity charting for distance and near was 6/5 with N6 at 30cm in her right eye and 6/7.5 and N6 with effort in her left eye respectively. Slit lamp examination of the anterior segment was found to be normal in both her eyes. Right eye fundus showed a well circumscribed domed shaped lesion, measuring 1DD located 1DD temporal to the fovea. Left eye fundus showed a similar dome shaped circumscribed lesion measuring 2DD in involving the fovea. Colour vision and the intraocular tension in both her eyes were normal.

Right eye showing a well circumscribed domed shaped lesion, measuring 1DD located 1DD temporal to the fovea

Left eye showing dome shaped circumscribed lesion measuring 2DD involving the fovea

Fundus fluorescein angiography (FFA) of right eye showing hyperfluorescence in late phase

FFA of left eye showing central hyperflourescene with surrounding pooling

Indocyanine angiography Both eye fundus showing hypofluorescence of the lesions in the venous phase

Optical coherence tomography (OCT) of right eye showing normal foveal dip with elevated RPE located temporally, subretinal fluid (SRF) noted above it with few hyperreflective dot echoes

OCT of left eye-foveal contour seen with SRF located subfoveally

Based on her history and present clinical findings we diagnosed it as a case of Presumed Ocular Tuberculosis presenting with right eye parafoveal choroidal tuberculoma, left eye choroidal tuberculoma involving the fovea with overlying serous detachment.

The patient was then referred to the chest physician for antitubercular treatment to be started along with systemic corticosteroids.

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STEPHEN J RYAN: a visionary, a clinician & an academician

(20 March 1940–29 April 2013)

Abhishek Varshney & Kumar Saurabh

Address for correspondence: Kumar Saurabh, Associate consultant, Department of Vitreoretina, Sankara Nethralaya, Chennai, email: vrfellow@gmail.com

Stephen J Ryan was born in 1940 to an ENT specialist in U.S. Naval hospital at Pearl Harbor just before the World War II in Honolulu, Hawaii. His parents were stationed at the U.S. Naval Hospital at that time. He did his graduation from Providence College in 1961and was awarded M.D. from the John Hopkins medical school in 1965. His initial plan while entering the medical school was to become a cardiac surgeon. It was in his summer lab position at Wilmer that he met Dr Ed Maumenee and found his mentor and the role model for the rest of his life. After his medical school he was selected by Dr Maumenee as a Wilmer resident. He was called for the interview for the chairman of ophthalmology at Cornell. He also did his fellowship at the AFIP with Lorenz Zimmerman and returned to Wilmer to join Dr Maumenee.

Dr Ryan left the Wilmer faculty in 1974 and went to USC to be the first chairman of the USC Department of Ophthalmology. He was also the only full-time faculty member and functioned like a chief resident. He spent majority of his career at the USC Department of Ophthalmology. He struggled a lot with the L.A. county general hospital to get basic equipments at the USC Campus.

He along with Father William G. Ward, Hugh Edmondson, Sid Webb and Ed Landry were the pioneers in building ophthalmology at USC and Doheny eye institute was thus developed. He became dean of the medical school in 1991 at USC and served at this position for 25 years. He also served as the senior vice President from 1993 to 2004.

Dr. Ryan was the Home Secretary of the Institute of Medicine (IOM) of The National Academy of Sciences since 2005. He was a Member of the NIH National Advisory Eye Council (NAEC) from 1982 for 3 years and was also the chairperson of the Retina Panel for the NAEC. He was also the Member of the Visual Sciences "A" Study Section in the Division of Research Grants at the National Institutes of Health from 1975 to 1979.

Dr Stephen J. Ryan was awarded with multiple awards in his exemplary career including The American Academy of Ophthalmology Laureate, The Pan-American Association of Ophthalmology Benjamin Boyd Humanitarian Award and Doheny Professional Association Lifetime Achieve Award to name the few. He was also honoured with Johns Hopkins University Society of Scholars Award and the Distinguished Alumnus Award in recognition to his expertise in the field of retinal diseases and ocular trauma.

Dr. Ryan has provided congressional testimony on numerous occasions over the past 25 years in support of the NIH and the National Eye Institute. He also provided his service on the Boards of Allergan, Inc., The Arnold and Mabel Beckman Foundation, The Johns Hopkins Health System, and the W. M. Keck Foundation.

Dr. Ryan was a member of several ophthalmologic organizations and has served as President of the Association of University Professors of Ophthalmology and the Macula Society. He was the founder President of the Alliance for Eye and Vision Research and the National Alliance for Eye and Vision Research. He was the author or editor of nine books, including RETINA. He was the author of over 285 articles in the scientific peer-reviewed literature.

On 29th April 2013, at the age of 72, he lost his battle against cancer and retired from this world one day before his scheduled retirement from the Allergan Board of Directors which he joined in 2002.

(Photograph courtesy: http://www.hopkinsmedicine.org)

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