

Cavernous Sinus Thrombosis

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Summary

An analysis of six cases of cavernous sinus thrombosis, admitted in the Department of Ophthalmology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar, Pakistan from March 1986 to July 1986, is presented. All cases survived on aggressive antibiotic therapy combined with anti-inflammatory and anti-coagulant drugs. The common organism isolated was Staphylococcus aureus. All cases were septic in nature. The commonest source was a furuncle on the upper half of the face near or on the eyelids. There was sinusitis in one case. The signs, symptoms, differential diagnosis, sources of spread of infection, investigations and treatment are discussed.

Introduction

Cavernous sinus thrombosis may be defined as thrombosis of the cavernous sinus due to septic or aseptic thrombophlebitis, which may be unilateral or bilateral. Aseptic cases may be caused by neurological procedures or head injuries complicated by fractures of the anterior or middle cranial fossae. In septic cases the suppurative process may be in i) the orbit (spread via the ophthalmic veins); ii) the oral cavity and nasal sinuses (spread via the pterygoid plexi); iii) the upper half of the face (spread via the frontal and angular veins); iv) the middle ear

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(spread via the petrosal sinus) and v) the side of the head (spread via the superior petrosal sinus).¹

The common routes of the spread of infection are i) the anterior route comprising the orbits, frontal sinuses, nasal cavity and upper half of the face; ii) the middle route comprising the sphenoid sinuses, the pharyngeal and pterygoid plexi, the pharynx, the upper jaw and the teeth; iii) the posterior route from the petrosal sinus and occasionally the ear and lateral sinus.² Spread from the lower jaw occurs twice as often as from the upper jaw due to direct spread from the pterygoid plexus.

The common causative lesions, acting as a source of infection, in the septic thrombosis are i) a furuncle, especially on the upper half of the face, which in itself may appear to be of an insignificant nature; ii) acute sinusitis, especially when complicated by osteomyelitis and iii) ear infections. Commonly isolated organisms are gram-positive cocci, i.e. staphylococci, streptococci and pneumococci; occasionally gram-negative organisms may also be isolated. In diabetics and immuno-suppressed individuals, it may be caused by mucormycosis.³ It may also be caused by metastasis in pyaemias, septicaemias and general febrile illnesses.¹

The signs and symptoms of the disease may be ocular as well as systemic. Ocular signs may affect one or both eyes. Common ocular signs are swelling of the lids, conjunctival chemosis, proptosis, decreased visual acuity, oedema of the upper lids, base of the nose, face and mastoid process due to venous engorgement. Oedema of the mastoid region is said to be diagnostic of cavernous sinus thrombosis.^{1,5} Pain, photophobia and lacrimation may be early symptoms. The earliest cranial nerve to be involved is the Abducent (VI) and in unilateral cases, weakness of the opposite lateral rectus may be the first sign of spread to the opposite side.² Involvement of the third, fourth and sixth cranial nerves causes limitation of ocular movements and proptosis. Involvement of the Optic nerve causes decreased vision due to neuritis, while involvement of the fifth nerve especially its ophthalmic division causes pain locally, supra-orbitally and in the region of its distribution.² Corneal anaesthesia followed by corneal clouding and corneal ulcers as well as hyperaesthesia of the forehead occurs. Paralysis of the parasympathetics causes dilated pupils. Ophthalmoscopic findings are engorged retinal veins, which may or may not be present, papillitis, papilloedema and retinal haemorrhages. These signs, if present, can only be seen if the eyelids can be separated. They may, however, be absent until a very late stage of the disease.

Systemic signs and symptoms include pyrexia with rigors, prostration, malaise, nausea, vomiting, leucocytosis, tachycardia and cerebral symptoms like

convulsions.¹ Convulsions may occur early in the course of the disease but the sensorium remains clear until late when coma may supervene and eventually lead to death. The cerebrospinal fluid remains normal unless there is an associated subdural abscess or meningitis³, or in some cases may be haemorrhagic due to infarction of the brain.⁴

Material and Methods

Six patients were admitted for cavernous sinus thrombosis in the Department of Ophthalmology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar, from March 1986 till July 1986. Diagnosis was made primarily on the basis of history and clinical findings. Laboratory investigations were carried out for supportive evidence: these include swabs for culture and sensitivity from the suspected source, total and differential leucocytic counts, platelet counts, ESR. Bleeding, clotting and prothrombin time was also done prior to starting anti-coagulants and thereafter regularly as long as the patient was using these drugs. X-rays of the skull, both A.P. and lateral views, as well as X-rays of the paranasal sinuses were taken. In one patient a brain scan was also done, as he was having convulsions. We also obtained the help of a consultant physician in the diagnosis and treatment. Fundoscopy and a full ophthalmic examination were carried out where and when possible. In cases where abscess formation had occurred, incision and drainage was carried out.

Results

These six patients can be divided into two basic groups:—

Group One consists of five patients who developed cavernous sinus thrombosis following the development of a seemingly insignificant furuncle :-

- on the upper lid (two cases): Photos No. 1 and 2;
- on the lower lid (one case): Photo No. 3 and
- on the upper half of the face near the lower lid (two cases): Photos No. 4 and 5.

About 3–4 days later they suddenly developed swelling of the eyelids, forehead, face, neck and over the mastoid process of the same side as the furuncle; the swelling became worse spreading to the opposite side of the face. One of these patients also had pitting oedema over the opposite mastoid process. All these patients presented about 6–7 days after the development of the furuncle and about 3 days after developing swelling of the face. All patients complained of severe headache. They had all used a variety of broad spectrum antibiotics in small quantities. Three of these patients had developed lid abscesses which were



Photos: Top 1 & 2:
Cavernous Sinus
Thrombosis following
furuncle on upper lid

Centre :
Cavernous Sinus
Thrombosis following
furuncle on lower lid



Bottom 4 & 5 :
Cavernous Sinus
Thrombosis following
furuncle on upper
half of face near
lower lid



subsequently drained. One patient had multiple small pustules which resolved with treatment. The eye in most cases could not be initially examined due to oedema; however, in one patient, the opposite lateral rectus showed weakness suggesting spread to the other side as well. Once the oedema had subsided sufficiently to allow examination, two patients showed limitation of ocular movements and proptosis, and all patients showed sluggish pupils and some degree of venous congestion on the affected side. One patient showed early papilloedema. Moderate visual loss occurred in one case. In four of these cases *Staphylococcus aureus* was cultured from the source, while in one case no growth was obtained. All cases responded rapidly to treatment.

Second Group: Our sixth case needs to be separately grouped: he presented with a history of severe frontal headache and nasal obstruction, followed 2 days later by a swelling of the same side of the eyelids, forehead, face, neck and mastoid process. Later the same day he had two bouts of epistaxia. The next day the other side of the face was also swollen, when he presented to us. X-rays of the skull and paranasal sinuses showed haziness of the frontal sinus of the same side. On examination there was also oedema of the lateral wall of the nose and septum on the side involved. The area over the frontal sinus was very tender. Blood dyscrasias were ruled out. No growth was obtained from the swab sent for culture and sensitivity. The patient responded slowly to treatment and his condition improved gradually after more than 2 weeks of intensive therapy. When the oedema subsided there appeared a depressed area in the skull vault over the inflamed sinus. The source appeared to be acute sinusitis with osteomyelitis leading to cavernous sinus thrombosis. He later developed convulsions, which settled completely with treatment.

The ages of these patients ranged from 14–30 years. The average age being 20 years. Four patients were female while two were male. All patients on admission were very toxic with high fever ranging from 103 to 104 °F with sweating, rigors, weakness and malaise.

Differential Diagnosis : The following conditions were kept in mind :—

1. Orbital cellulitis: most important and commonest.
2. Orbital tumours.
3. Meningioma.
4. Tumours in the region of the sphenoid.
5. Trichinosis.
6. Malignant exophthalmos.
7. Arteriovenous aneurysms.
8. Romana's sign (in Chagas' disease).

Treatment consisted of aggressive antibiotic, anti-coagulant and anti-inflammatory therapy. The antibiotics used were Inj. Cefamandole 1 Gm. I/V-6 hourly, Inj. Nebcin 80 mg. I/V, t.d.s., or Inj. Benzyl Penicillin 24,000,000 units I/V a.t.d.* in 24 hours in a continuous drip or 2 hourly 2,000,000 units I/V a.t.d.* with tab. Septran DS b.d. When the condition appeared to settle down, then Cefamandole and B. Penicillin were substituted by Cap. Ceporex or Ampiclox. The anti-coagulants used were tab. Persantin Plus, tab. Aspirin and Inj. Heparin 5000 units 6 hourly or 30,000 units in a continuous drip. We used Ibuprofen 400 mg. t.d.s. as anti-inflammatory and oral analgesics for pain in all patients. Tab. Chymoral was also used in some patients. Locally Genticin skin cream, Polyfax eye ointment and Genticin eye drops were given t.d.s. when required.

Patients were followed up for 1-4 months. All six patients recovered completely. One patient developed mild cicatricial ectropion and retraction of the upper lid; while two of them developed gangrene of the lids: debridement was done followed by Betnesol-N eye ointment t.d.s. Subsequent plastic repair for cicatricial ectropion was required in these cases. Prednisolone tab. 5 mg. 2 tab. q.i.d. was used in two patients in a tailing off dose.

Discussion

In all cases the diagnosis was clinched by the presence of mastoid oedema^{1,5} along with gross facial oedema in an extremely toxic patient with signs of infection in a) the upper half of the face and b) the frontal sinus. Bilateral thrombosis occurred in one patient indicated by bilateral mastoid oedema and involvement of the lateral rectus of the second eye. On admission the eye on the side first involved could not be examined due to gross oedema. In two cases, on resolution of the oedema (2-4 days later), restricted ocular movements were found (in one case in both eyes). All patients showed sluggish pupils and some degree of venous congestion on examination of the fundus of the side primarily affected. When vision was finally recorded, 5 cases had 6/6 vision in both eyes, while one patient was able to count fingers at 4 meters in the eye on the affected side but this later on improved to 6/6 with treatment.

Although most patients presented 3-4 days after the appearance of symptoms, the prior use of some antibiotic may have helped to retard the course of the disease. In four of our first five patients, we used various anti-coagulants; however, subsequent clotting time was not delayed significantly in a single case, bringing to mind the value of these drugs. Heparin, when stored out in the open, could affect its potency resulting in the use of a degraded end-product having no

* After test dose.

anti-coagulant properties. Another possibility is that we used too small a dosage as the standard dose according to the British National Formulary is 10,000 units q.i.d or 40,000 units in a continuous drip.⁷ As two of our patients recovered satisfactorily, we do not advise the use of Heparin in patients with cavernous sinus thrombosis, especially as its value has not been proved³ and it may produce haematuria;⁸ as a matter of fact some workers consider it to be contraindicated as cavernous sinus thrombosis produces haemorrhagic brain tissue.²

In our last patient, we only used Benzyl Penicillin 24,000,000 units I.V. daily in a continuous drip and Ibuprofen 400 mg. t.d.s. with local Genticyn skin cream and she responded to this treatment even more rapidly than the other patients, showing a marked improvement within 24 hours, while the other four patients required 3–4 days to show improvement. The patient whose source of infection was sinusitis took 10 days to show improvement. The use of steroids has also been advocated;⁶ we used Prednisolene 40 mg/day in divided doses in two of our patients but did not note any significant change in the recovery of the patients.

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