

The optic chiasm is the junction between the two optic nerves (from the back of the eyes).

Arachnoiditis in this area can arise due to infection (cystercicosis, TB etc.) especially basal meningitis.

Viral infection (poliomyelitis, encephalitis) has also been linked with this type of the condition.

Some authors suggest that paranasal sinus infection is a causative factor.

In some cases, there may be a traumatic origin and there have also been cases arising after eye surgery (retained muslin).

Ramina et al. ([\[i\]](#)) described a case of severe optochiasmatic arachnoiditis after rupture of a cerebral aneurysm; the man developed progressive visual loss after a subarachnoid bleed; OA was confirmed during later surgery.

Fujimura et al. ([\[ii\]](#)) reported a case of visual disturbance due to optochiasmatic arachnoiditis and foreign body granuloma 9 months after surgery for aneurysm in which cotton wrapping was used.

The authors recommended the avoidance of use of the cotton sheet close to the optic nerve.

There have been suggestions of three phases of optochiasmatic arachnoiditis:

- 1) inflammatory
- 2) fibrous
- 3) hyperplastic (including proliferation of arachnoidal cells)

Aldrete notes that 4 types of OA have been considered:

- 1) Parenchymatous
- 2) Diffuse
- 3) Adhesive
- 4) Cystic

Prado and Oribe ([\[iii\]](#)) proposed the term *optochiasmatic leptomeningitis*, describing lesions as exudative (oedema plus white cell proliferation), haemorrhagic (haemorrhages with fibroblast proliferation) or productive (a thick membrane with islands of arachnoidal proliferation; hyaline degeneration and calcification)

Balado and Franke ([\[iv\]](#)) described deep demyelination in the optic nerve.

The effects of OA may result from direct compression of the arachnoid membrane on the optic chiasm and/or from constriction of the blood supply to the optic nerves.

Clinically, OA manifests initially with fronto-temporal headaches followed by eye problems in one or both eyes: firstly with reduced visual acuity. Both eyes may have loss of visual fields (often central scotoma).

Examination may reveal optic disc pallor or papilloedema.

Neuroendocrine manifestations may also occur: gonadotrophin insufficiency (impotence, amenorrhoea) or secondary hypothyroidism may be detected; diabetes insipidus, somnolence, pyrexia may suggest hypothalamic effects in isolated cases.

Diagnosis may be difficult. Visual-evoked potentials may be altered.

MR scans may demonstrate localised inflammation in the perichiasmal area. Differential diagnosis includes Multiple Sclerosis (optic neuritis) and Devic's disease.

[i] Ramina R, Arruda WO, Prestes AC, Parolim MK. *Arq Neuropsiquiatr* 1989 Jun; 47(2):192-6 severe optochiasmatic arachnoiditis after rupture of an internal carotid artery aneurysm.

[ii] Fujimura M, Nishijima M, Umezawa K, Hayashi T, Mino Y, Sakuraba T, Midorikawa H *J Clin Neurosci* 2003 Mar; 10(2):254-7 Optochiasmal arachnoiditis following cotton wrapping of anterior communicating artery aneurysm treated by surgical removal of granuloma.

[iii] Prado J Oribe M *Arch Histol N Patol* 1945; 2: 477-496 [Contribucion al estudio histopatologico de las arachnoiditis (microleptomeningitis)].

[iv] Balado M Franke E *Arch Arg Neurol* 1937; 15: 199 [Aleraciones quiasmaticas en las arachnoiditis del quiasma]