METABOLIC AND MICROCIRCULATORY DISORDERS IN ISCHEMIC OPTIC NEUROPATHY

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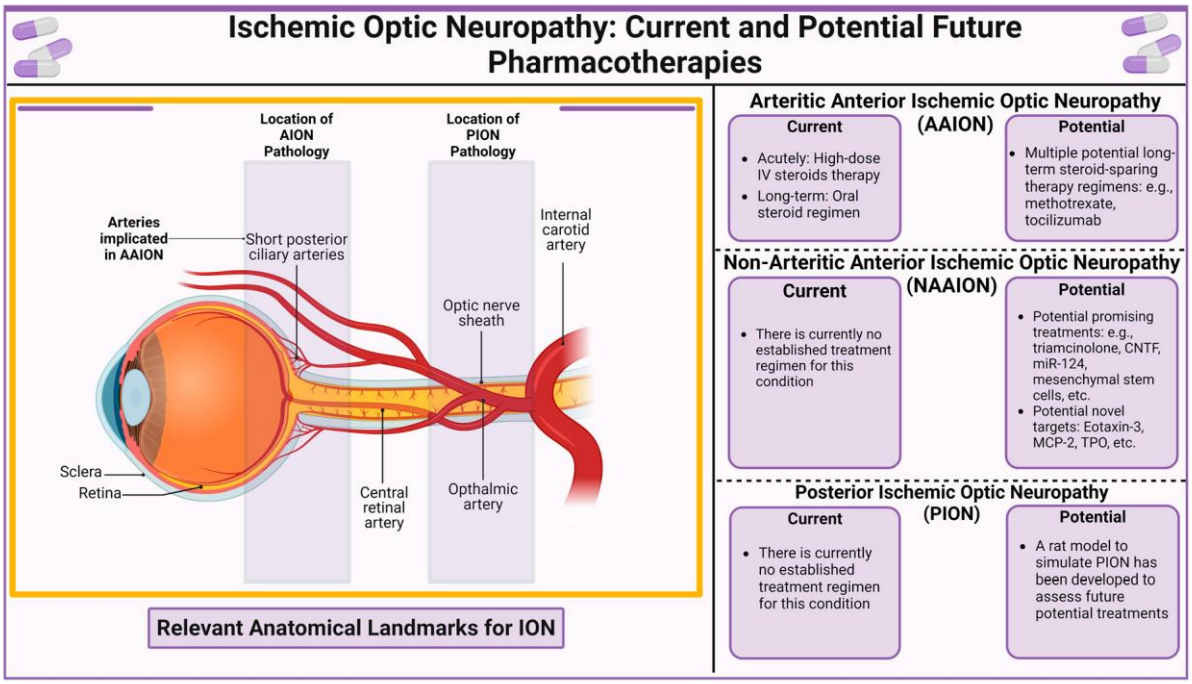
Abstract. *The blockage can occur with inflammation of the arteries (the arteritic form, usually associated with a disease called giant cell arteritis) or without inflammation of the arteries (the non-arteritic form). The only permanent symptom is painless vision loss, which usually comes on suddenly. Doctors make the diagnosis based on a person's symptoms and by examining the patient's eye with an ophthalmoscope. Blood tests and sometimes a biopsy of temporal artery tissue are used to diagnose giant cell arteritis. Treatment for the arteritis type does not restore vision, but it can help protect the healthy eye.*

Keywords: *Causes, Symptoms, Diagnosis, Treatment, Prognosis.*

Introduction: Occlusion of a blood vessel that supplies blood to the optic nerve within the eye can lead to optic nerve dysfunction and vision loss. There are two types of arterial occlusion: arteritic and non-arteritic. Non-arteritic ischemic optic neuropathy is more common and usually affects people 50 years of age and older. Vision loss is usually not as severe as arteritic ischemic optic neuropathy. Risk factors include anatomical swelling of the optic nerve (certain features of the optic nerve that a doctor can see with an ophthalmoscope), high blood pressure, smoking, diabetes, and atherosclerosis. Other risk factors include obstructive sleep apnea, taking certain medications (such as amiodarone and possibly phosphodiesterase inhibitors, such as sildenafil, which is used to treat erectile dysfunction), a tendency to form blood clots, and low blood pressure at night. Arteritic ischemic optic neuropathy usually affects people over the age of 60. Inflammation of the arteries (arteritis), primarily due to giant cell arteritis, blocks blood flow to the optic nerve. Vision loss usually occurs quickly (within minutes, hours, or sometimes days) but is painless. In nonarteritic ischemic optic neuropathy, vision is usually impaired in one eye. In arterial ischemic optic neuropathy, the impairment can affect both eyes. Vision in the affected eye or eyes can range from almost normal to complete blindness. People with giant cell arteritis (usually adults) may have significant vision loss. They may have pain in the lower jaw when chewing or combing their hair, headaches, muscle pain, and scalp pain.

Research methods and materials: In the diagnosis, the fundus of the eye is examined using a magnifying lens (ophthalmoscope) using a lamp and a visual field test is performed to assess the degree of loss of central or peripheral vision. The optic nerve head in the fundus (the optic disc appears swollen). When determining the cause, it is determined whether the person has diseases called risk factors.

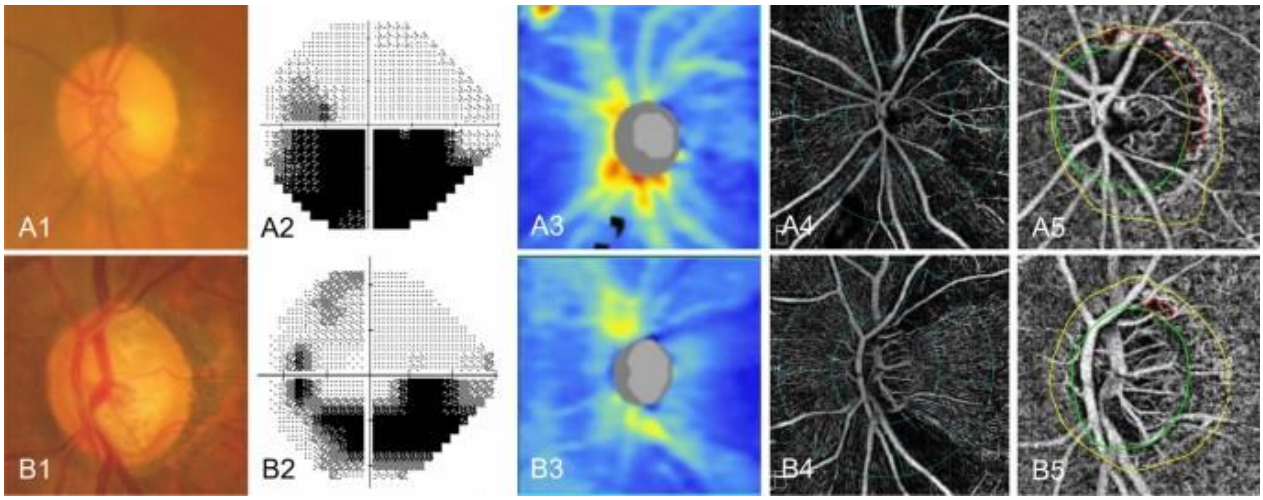


If giant cell arteritis is suspected, blood tests are ordered and treatment with corticosteroids is started immediately to prevent further vision loss. A sample of tissue (biopsy) from the temporal artery may be taken to confirm the diagnosis and examined under a microscope. Blood tests measure the erythrocyte sedimentation rate (ESR), C-reactive protein levels, and levels of certain types of blood cells (complete blood count). The results of these tests may indicate the presence of inflammation, which is a characteristic feature of giant cell arteritis. If there are no signs of giant cell arteritis, a magnetic resonance imaging (MRI) or computed tomography (CT) scan of the brain may be done to make sure that the optic nerve is not being compressed by a tumor.

Results: Depending on the suspected cause, other tests and investigations may be needed. For example, if a person has symptoms of obstructive sleep apnea (such as daytime sleepiness or snoring), a polysomnogram may be performed. If a person has blood clots, blood tests may be done to check for bleeding disorders. In non-arteritic ischemic optic neuropathy - controlling risk factors for atherosclerosis

For arteritic ischemic optic neuropathy due to giant cell arteritis - corticosteroids and tocilizumab

Treatment is not effective in restoring vision in people with nonarteritic ischemic optic neuropathy. Treatment includes reducing risk factors for atherosclerosis, including controlling blood pressure and diabetes. Treatment of other conditions that cause nonarteritic ischemic optic neuropathy, such as bleeding disorders and obstructive sleep apnea, may also be required.



To prevent vision loss in the other eye, people with arteritic ischemic optic neuropathy due to giant cell arteritis are given high doses of oral and/or intravenous corticosteroids as soon as possible. Adding tocilizumab (a drug that reduces inflammation) to corticosteroids has recently been shown to help people with giant cell arteritis.

People with vision loss may benefit from magnifying glasses, magnifying glasses, and talking watches (low vision aids). There is no effective treatment for non-arteritic ischemic optic neuropathy. However, about 40% of people with non-arteritic ischemic optic neuropathy experience spontaneous, modest improvement in vision. Recurrent episodes of the disease in the same eye are rare in this condition, but in 15% of people, the other eye is affected.

Discussion: Visual loss in the arteritic variety of the disease caused by giant cell arteritis is usually more severe than in nonarteritic ischemic optic neuropathy. Prompt treatment with corticosteroids does not restore vision lost in the affected eye but does protect the unaffected eye. Inadequate treatment increases the risk of vision loss in the other eye. Nutritional and toxic optic neuropathies are damage to the optic nerve caused by poor nutrition (nutritional optic neuropathy) or exposure to substances that are harmful to the optic nerve (toxic optic neuropathy), such as lead, methanol (wood alcohol or methyl alcohol), ethylene glycol (antifreeze), or certain drugs. Optic neuropathy caused by nutritional deficiencies (especially vitamins B1 and B12 or folic acid [folic acid; see General Information on Vitamins]) is called nutritional optic neuropathy. People who have had weight loss surgery (bariatric surgery) and people with alcohol use disorder are especially

at risk of developing nutritional optic neuropathy. The real cause of people with alcohol use disorder may be poor nutrition rather than the toxic effects of alcohol.

Rarely, optic neuropathy is caused by drugs (such as chloramphenicol, isoniazid, ethambutol, and digoxin) or toxins such as lead, ethylene glycol (antifreeze), or methanol (wood or methyl alcohol). When the disease is caused by a substance, drug, or toxin, it is called toxic optic neuropathy. People with nutritional or toxic optic neuropathy gradually lose their vision over days or weeks. A blind spot may appear and gradually enlarge, usually affecting the center of vision. It may not be noticeable at first. Loss of color vision can be more serious than loss of visual acuity. Both eyes are usually affected.

Poisoning with ethylene glycol and especially methanol can cause rapid and complete loss of vision. Both substances can cause other serious symptoms such as coma, shortness of breath, vomiting, and abdominal pain.

Drinking antifreeze (ethylene glycol) or methanol (wood alcohol or methyl alcohol) can cause sudden and complete loss of vision.

Doctors diagnose nutritional or toxic neuropathy based on a person's history of poor nutrition or exposure to toxins or chemicals, as well as eye symptoms and vision tests. Sometimes tests are done to look for toxins or vitamin deficiencies.

If you drink alcohol or have a poor diet, take vitamin supplements and avoid alcohol;

treatment of drug-induced and toxic injuries (e.g., chelating agents to treat lead poisoning or hemodialysis and fomepizole to treat ethylene glycol or methanol exposure);

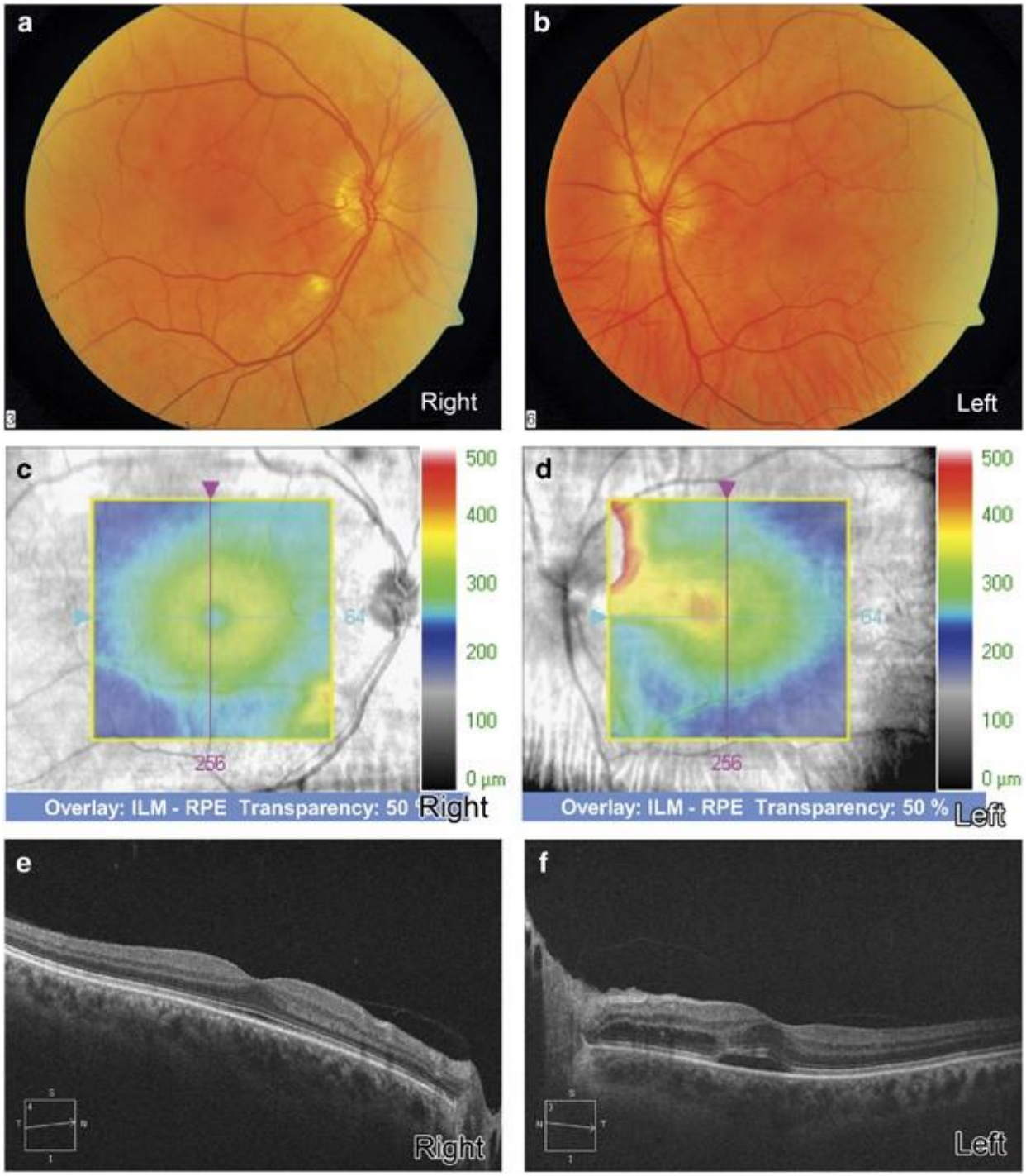
If optic neuropathy is caused by alcohol consumption or a poor diet, the person should stop drinking, eat a balanced diet, and take vitamin supplements containing folic acid and B vitamins. However, if the cause is a vitamin B12 deficiency, treatment with supplements alone is not enough. B12 deficiency is usually treated with vitamin B12 injections.

People with toxic optic neuropathy should avoid alcohol and other chemicals, drugs, or medications that may have toxic effects. If lead is the cause of toxic optic neuropathy, chelating drugs (such as succinate or dimercaprol) can help remove it from the body. If the cause is ethylene glycol or methanol poisoning, rapid hemodialysis to remove these toxins and the antidote fomepizole may help. Ethyl alcohol (sometimes called ethanol, grain alcohol, or simply alcohol) may also help because it partially stops the toxin (ethylene glycol or methanol) from being converted into a harmful byproduct.

People with vision loss can use magnifying glasses, font magnifiers, and talking watches (low vision aids).

With timely treatment, most people with nutritional or toxic optic neuropathy recover their vision.

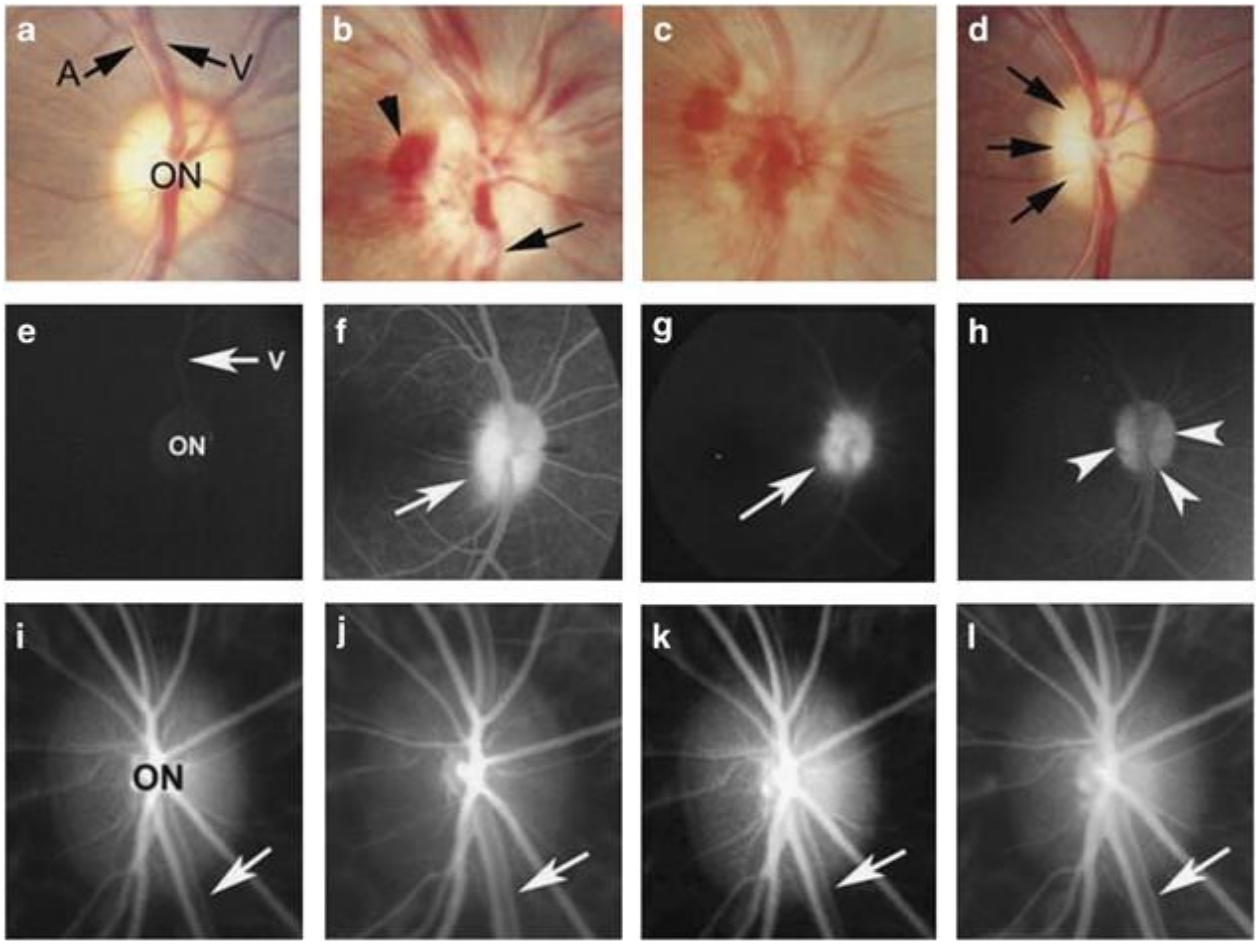
Optic neuritis is the most common optic nerve disease in people under the age of 50. The most common cause of optic neuritis is multiple sclerosis. Some people with optic neuritis have already been diagnosed with multiple sclerosis, while other people with optic neuritis are diagnosed with multiple sclerosis later. Causes of optic neuritis can also include:



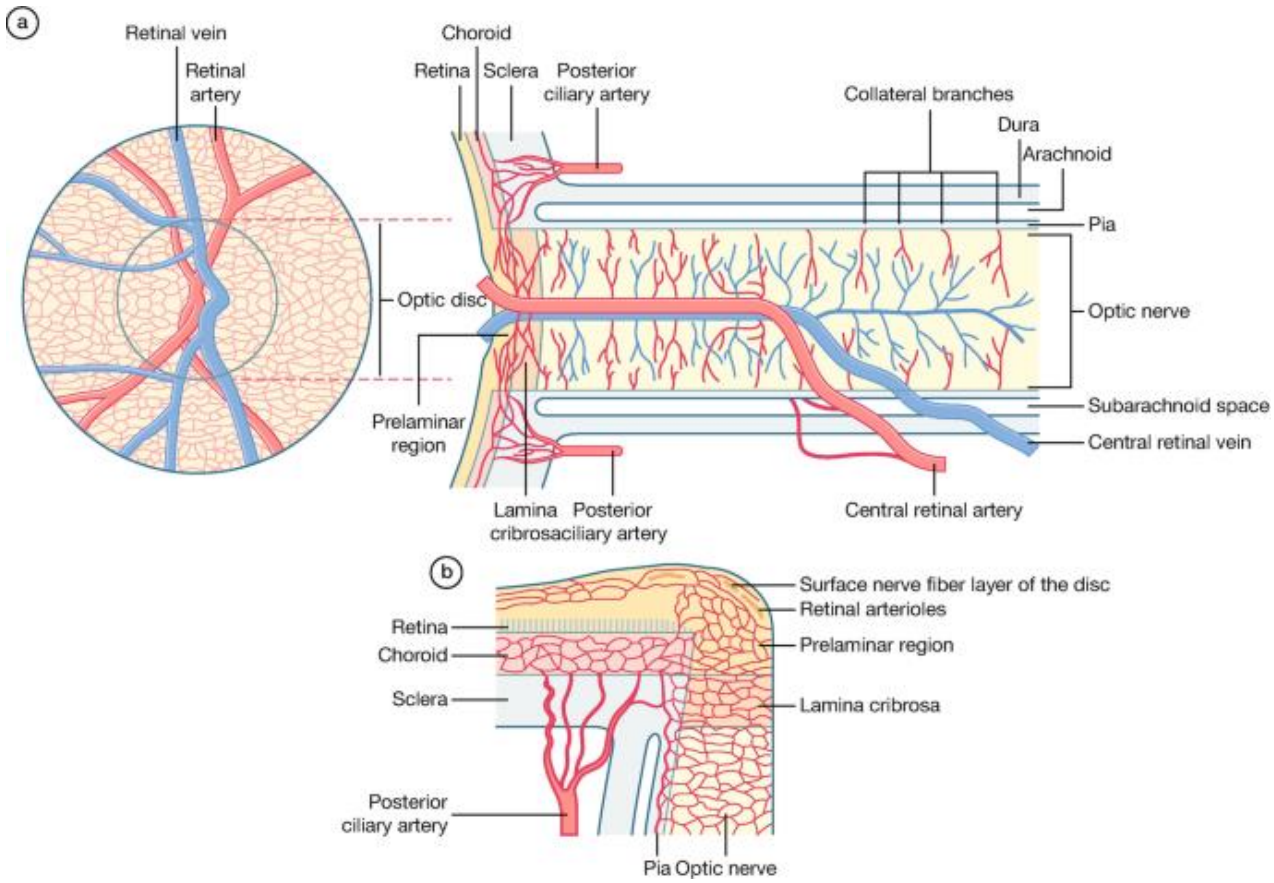
Optic neuritis causes vision loss that may be noticeable in one or both eyes. Vision loss can develop over a period of days. Vision in the affected eye or eyes can range from almost normal to complete blindness. Color vision can be particularly affected, but the person may not be aware of it. Most people experience moderate eye pain, which is usually worse with eye movement.

Depending on the cause, vision usually returns within 2-3 months, but not always completely. In some people, optic neuritis may recur.

Diagnosis involves checking the pupillary reactions and examining the fundus using a magnifying lens (ophthalmoscope) with a lamp. The optic nerve head (optic disc) in the fundus may be swollen. When testing the visual field, it is common to find that part of the visual field is lost.



Magnetic resonance imaging (MRI) of the brain may show signs of multiple sclerosis; or myelin oligodendrocyte glycoprotein antibody disorder (also called MOGAD), a neurological immune-mediated disorder that involves inflammation of the optic nerve; or neuromyelitis optica (also called NMO), a rare immune disorder that affects the spinal cord and optic nerve. MRI of the brain and orbit usually reveals optic nerve abnormalities. Diagnostic imaging of the spinal cord may be performed in people with neurological symptoms.



Summary: In some cases, corticosteroids are given intravenously to treat optic neuritis. Oral corticosteroids may be prescribed after a few days. These medications can speed recovery. Plasmapheresis may sometimes be used if vision is severely reduced and does not improve with corticosteroids. If optic neuritis is associated with multiple sclerosis, NMN, MOGAD, or infection, the underlying disease should also be treated.

People with vision loss can use magnifying glasses, font magnifiers, and talking watches (low vision aids).

At first, optic nerve swelling may not affect vision. The short-term visual disturbances characteristic of optic nerve swelling—blurred vision, double vision, flickering, or complete loss of vision—usually last a few seconds. Other symptoms may occur as pressure in the brain increases. A pulsating, whistling noise in the ears, headache, nausea, vomiting, or a combination of these symptoms may occur. This condition does not cause eye pain.

To diagnose papilloma, a doctor uses an ophthalmoscope (a light with a magnifying lens used to examine the back of the eye). Often, an ophthalmologist (a doctor who specializes in evaluating and treating eye diseases) will need to confirm the diagnosis and may need help determining the cause.

Magnetic resonance imaging (MRI) or computed tomography (CT) scans of the brain and eye sockets may be performed to determine the cause and monitor the effectiveness of treatment. MRI or CT venography of the head may be performed to rule out cerebral venous sinus thrombosis.

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