GLAUCOMA

ANAGHA.P

Introduction

- Glaucoma refers to a group of diseases characterized by
 - optic neuropathy
 - specific pattern of visual field defect
 - raised intraocular pressure
- Damage to optic nerve is irreversible process
- Normal IOP is 10-21mmHg



Definition

- "Glaucoma is a disease in which optic nerve is damaged, leading to progressive, irreversible loss of vision. It is often, but not always, associated with increased pressure of the fluid in eye." - En.wikipedia.org
- "A disease of the eye in which pressure of fluid inside the eyeball is abnormally high, caused by obstructed outflow of the fluid. The increased pressure can damage the optic nerve and lead to partial or complete loss of vision." www.thefreedictionary.com
- Glaucoma is a group of ocular conditions characterized by optic nerve damage. The optic nerve damage is related to the intra ocular pressure (IOP) caused by congestion of aqueous humor in the eye. - Bruner & Suddarth's

Glaucoma











Development of Glaucoma



Flow of aqueous humour through the drainage canal

Drainage canal blocked: build up of fluid Increased pressure damages blood vessels and optic nerve

Aqueous drainage









Venous circulation of ciliary body, choroid and sclera

HISTORY

- Richard Bannister The association of elevated (IOP) and glaucoma was first described
- **John Collins Warren** -Angle-closure glaucoma was treated with cataract extraction
- Hermann Holtz Invention of ophthalmoscope to identify pathological hallmark of glaucoma, the excavation of the optic nerve head due to retinal ganglion cell loss.
- ► Hjalmar August Scholtz The first reliable instrument to measure intraocular pressure was invented
- **Hans Goldmann** Developed applanation tonometer
- Pilocarpine The first drug to reduce IOP



- ► Glaucoma is the leading cause of irreversible blindness.
- ▶ 6 million individuals are blind in both eyes from this disease.

EPIDEMIOLOGY

- ▶ In the world, glaucoma is the third leading cause of blindness.
- Estimated 13.5 million people may have glaucoma and 5.2 million of those may be blind.

ETIOLOGY

Excess aqueous secretion production

Impaired outflow through the trabecular meshwork work

Obstruction of the out flow from schlemm's canal and aqueous vein created by elevated orbital venous pressure

ETIOLOGY

Ocular hypertension

IOP above 21 mm Hg / above 24 mm Hg –It is not necessarily a pathological condition but it increases risk of glaucoma. One study found a conversion rate of 18% within 5 years, meaning less than 1 in 5 people with an elevated IOP will develop glaucomatous visual field loss. Open-angle glaucoma accounts for 90% of glaucoma cases in US. Closed-angle glaucoma accounts for less than 10% of glaucoma cases in US, but as many as half of glaucoma cases in other nations

Eye injury ,eye surgery ,eye tumors

ETIOLOGY

- Diabetes /Diabetic Retinopathy
- Cataract

Trauma

- Steroid use/steroid induced Glaucoma
- Emotional stress
- Anti-histamine use
- Hypothyroidism
- Sleep apnea
- Leukemia
- ► Sickle cell anemia

RISK FACTORS

- Age over 45 years
- Family history of glaucoma ,Diabetes ,elevated intra-ocular pressure
- Near-sightedness (Myopia)
- Thin cornea
- ► A history of severe anemia or shock
- Cardiovascular disease
- Race
- Decreased Peripheral vision
- Not seeing a rainbow

CLASSIFICATION

Primary glaucoma

Developmental glaucoma

Secondary glaucoma

Absolute glaucoma

CLASSIFICATION

Primary glaucoma

1.Primary open-angle glaucoma/ chronic open-angle glaucoma/ chronic simple glaucoma/ glaucoma simplex

High-tension glaucoma

Low-tension glaucoma /normal tension /normal pressure

2. Primary closed-angle glaucoma /Primary angle closure glaucoma/ narrow-angle glaucoma/pupil-block glaucoma/acute congestive glaucoma

Acute angle closure glaucoma

Chronic angle closure glaucoma

Intermittent angle closure glaucoma

Superimposed on chronic open-angle closure glaucoma ("combined mechanism")



Glaucoma

Primary Open Angle Glaucoma is caused when the normal drainage system of the eye becomes partially blocked, causing pressure to build within the eye. Glaucoma usually affects the perimitery vision first, with sight gradually being lost towards the center of the eye.



Vision loss with Glaucoma



Risk-factors

- Ocular risk Factors
 - . IOP
 - . Myopia
 - . Increased cup/disc ratio
 - . Asymmetric cupping
 - . Disc hemorrhage

NORMAL CDR < 0.5

Risk-factors

- Non ocular risk factors
 - . Age
 - . Race
 - . Family history
 - . Diabetes and Systemic hypertension
 - . Migraine and peripheral vasospasm
 - . Alcohol consumption
 - . Cigarette smoking

Primary open-angle glaucoma



Symptoms

- Commonly Asymptomatic
- Detected Incidentally
- Mild headache, ocular pain
- Minimal blurring of vision
- Frequent changes in presbyopic spectacles
- Subjective visual field defect occasionally



Glaucoma

Angle Closure Glaucoma (Sometimes referred to as Narrow Angle Glaucoma) is caused when the normal drainage system of the eye becomes suddenly blocked, causing pressure to build within the eye at a very rapid rate. Complete blindness can occur in as little as 3 to 5 days!



Normal Eye



Sudden blockage causes pressure to build rapidly.



Primary closed-angle glaucoma



Symptoms :

1-eye redness and painful.

2-Blurred vision.

3-Patient may notice haloes (circles of light) around light.



Open and closed





CLASSIFICATION

Developmental glaucoma

- Primary /true congenital glaucoma IOP is raised during intrauterine life
 - Infantile glaucoma Diseases manifests prior to the child
 - Glaucoma associated with hereditary or familial diseases
 - Juvenile glaucoma Children develop pressure rise between 3-16 yrs. of life

Congenital glaucoma

- > It present at birth.
- due to the abnormal development of the anterior chamber angle before birth.

this causes decrease in aqueous outflow

loss of vision..

SYMPTOMS

- Tearing
- Light sensitivity

- Bupthalmus
- Cloudy cornea





PRIMARY CONGENITAL GLAUCOMA

DESCRIPTION

- A rare condition
- manifests without associated anomalies

Pathogenesis

Maldevelopment of the angle structures

Classification

- True congenital glaucoma (40%). IOP becomes elevated intrauterine life and child is born with ocular enlargement.
- Infantile glaucoma (50%). It manifests prior to child's third birthday.
- Juvenile glaucoma (10%). It manifests between 3-16 years.

Developmental glaucoma







Inflammatory glaucoma

* Uveitis of all types-inflammation of middle layer of eye

* Fuchs heterochromic iridocyclitis - c/c unilateral uveitis with triad of heterochromia (multicolored iris), predisposition to cataract and glaucoma, keratic precipitates (cellular deposits) on posterior corneal surface

Phacogenic glaucoma –lens induced glaucoma

* Angle-closure glaucoma with mature cataract

* Phacoanaphylactic glaucoma secondary to rupture of lens capsule

*Phacolytic glaucoma due to phacotoxic meshwork blockage-sudden onset of

open angle glaucoma caused by leaking mature/ hyper mature cataract

* Subluxation of lens –partial dislocation of lens

Glaucoma secondary to intraocular hemorrhage

* Hyphema – pooling / collection of blood inside anterior chamber of eye

* Hemolytic glaucoma / erythroclastic glaucoma – hemoglobin laden macrophages block trabecular outflow channels

Traumatic glaucoma

*Angle recession glaucoma - Traumatic recession on anterior chamber angle *Postsurgical glaucoma Aphakic pupillary block Ciliary block glaucoma

Neovascular glaucoma

abnormal vessels begin developing in the angle of the eye that begin blocking the drainage.

Drug-induced glaucoma

*Corticosteroid induced glaucoma

*Alpha-chymotrypsin glaucoma. Postoperative ocular hypertension from use of alpha chymotrypsin.

Glaucoma of miscellaneous origin

*Associated with intraocular tumors

*Associated with retinal detachments

*Secondary to severe chemical burns of the eye

*Associated with essential iris atrophy

Toxic glaucoma - open-angle glaucoma with an unexplained significant rise of IOP following unknown pathogenesis. IOP can sometimes reach 80 mmHg. It manifests as ciliary body inflammation and massive trabecular edema that sometimes extends to Schlemm's canal.

Absolute glaucoma

Absolute glaucoma is the end stage of all types of glaucoma. The eye has no vision, absence of pupillary light reflux and pupillary response, and has a stony appearance. Severe pain is present in the eye.
Pathophysiology

2 Theories regarding how increased IOP damages the optic nerve in glaucoma.

The direct mechanical theory

High IOP damages retinal layer as it passes through optic nerve head.

□ <u>The indirect ischemic theory</u>

High IOP compresses the microcirculation in optic nerve head, resulting in cell injury & death

□ Some glaucoma's appear as exclusively mechanical & some are exclusively ischemic types. Typically most cases are a combination of both.



- 1. Initiating Events: Precipitating factors include illness, emotional stress, congenital narrow angles, long term use of corticosteroids & mydriatics. These events lead to second stage.
- 2. <u>Structural alterations in the aqueous outflow system</u>: Tissue & cellular changes caused by factors that affect aqueous humor dynamics lead to structural alterations & to the third stage.
- 3. <u>Functional alterations</u>: Conditions such as increased IOP / impaired blood flow create functional changes that lead to fourth stage.
- 4. <u>Optic nerve damage</u>: Atrophy of optic nerve is characterized by loss of nerve fibers & blood supply & this fourth stage inevitably progresses to the fifth stage.
- ► 5. <u>Vision loss</u>: Progressive loss of vision is characterized by visual field defects.

Clinical manifestations

- Severe eye pain
- Eye redness
- Blurred vision
- Severe headache
- Nausea
- Vomiting
- Dry eyes with itching or burning
- Dark spot at the center of viewing
- Excess tearing /watery eyes
- Difficulty focusing on near or distant object

DIAGNOSTIC MEASURES

- **Ocular & medical history** : investigate history of predisposing factors.
- **Tonometry**: determines pressure in eye by measuring tone / firmness of its surface.
- **Ophthalmoscope**: done to examine optic nerve (seen as optic disc) at back of eye.
- Gonioscopy: To examine filtration angle of anterior chamber. purpose of this test is to examine drainage angle and drainage area of the eye.
- Perimeter: To assess the visual fields. The visual fields to detect any early (or late) signs of glaucomatous damage to the optic nerve. Visual fields are measured by a computerized assessment

DIAGNOSTIC MEASURES

Pachymetry : It is a relatively new test being used for the diagnosis and treatment of glaucoma. Pachymetry determines thickness of cornea. After eye has been numbed with anesthetic eye drops, the pachymeter tip is touched lightly to the front surface of the eye (cornea).Recent studies have shown that corneal thickness can affect measurement of intraocular pressure.

Dilated pupil examination

	What the test examines	Eye drops used	Physical contact with the eye	Procedure
Tonometry	Inner eye pressure	Maybe	Maybe	Eye drops may be used to numb eye. Examiner then uses a tonometer to measure inner pressure of eye through pressure applied by a puff of warm air or a tiny tool.
Ophthalmoscopy (dilated eye examination)	Shape and color of optic nerve	Yes	No	Eye drops are used to dilate pupil. Using a small magnification device with a light on end, examiner can examine magnified optic nerve.
Perimetry (visual field test)	Complete field of vision	No	No	The patient looks straight ahead and is asked to indicate when light passes patient's peripheral field of vision. This allows examiner to map patient's field of vision.
Gonioscopy	Angle in eye where iris meets cornea	Yes	Yes	Eye drops are used to numb eye. A hand-held contact lens with a mirror is placed gently on eye to allow examiner to see angle between cornea and iris.
Pachymetry	Thickness of the cornea	No	Yes	Examiner places a pachymeter gently on front of eye to measure its thickness.
Nerve fiber	Thickness of the nerve	Maybe	Maybe	Using one of several techniques, nerve fibers are

Glaucoma Prevention

We can't prevent glaucoma. But if find early, we can lower risk of eye damage. Steps that help protect vision:

- Have regular eye exams. The sooner doctor spots signs of glaucoma, sooner can start treatment. If above age 40, family history of disease, get a complete eye exam from an eye doctor every 1 to 2 years. If there is diabetes or are at risk of other eye diseases check more often.
- Learn family history. Ask relatives whether any of them have been diagnosed with glaucoma.
- Follow doctor's instructions. If they find that we have high eye pressure, they might give eye drops to prevent glaucoma.
- Exercise. Moderate activity like walking / jogging at least 3 times a week help lower eye
 pressure.
- Protect eyes. Use protective eyewear when playing sports /working on home improvement projects.

Medical management

- Aim = prevention of optic nerve damage through medical therapy.
- ▶ Lifelong therapy is almost necessary because glaucoma cannot be cured.
- Goal = maintain an IOP within a range unlikely to cause further damage.
- Patient is monitored for stability of optic nerve.
- Medical management relies on systemic & topical ocular medications that lower IOP.
- The patient is usually started on lowest dose of topical medication & then advanced to increased conc. until desired IOP level is reached & maintained.



A .Eye drops.

Lower creation of fluid in eye / increase its flow out, lowering eye pressure. Side effects include allergies, redness, stinging, blurred vision, and irritated eyes. Some glaucoma drugs may affect your heart and <u>l</u>ungs.

B.Oral medication

<u>1.Prostaglandin analogs</u> - latanoprost, bimatoprost, travoprost increase uveoscleral outflow of aqueous humor. Bimatoprost also increases trabecular outflow.

<u>2.Topical beta-adrenergic receptor antagonists</u> - timolol, levobunolol, betaxolol decrease aqueous humor production by epithelium of ciliary body.

3.Alpha2-adrenergic agonists - brimonidine , apraclonidine work by a dual mechanism, decreasing aqueous humor production and increasing uveoscleral outflow.

Treatment

<u>4.Less-selective alpha agonists</u> - epinephrine decrease aqueous humor production through vasoconstriction of ciliary body blood vessels, useful only in open-angle glaucoma. Epinephrine's mydriatic effect renders it unsuitable for closed-angle glaucoma due to further narrowing of uveoscleral outflow (i.e. further closure of trabecular meshwork, which is responsible for absorption of aqueous humor).

5.Miotic agents (parasympathomimetics) - pilocarpine work by contraction of ciliary muscle, opening trabecular meshwork and allowing increased outflow of the aqueous humor. Echothiophate, an acetylcholinesterase inhibitor, is used in chronic glaucoma.

<u>6.Carbonic anhydrase inhibitors</u> - dorzolamide, brinzolamide, acetazolamide lower secretion of aqueous humor by inhibiting carbonic anhydrase in ciliary body

\triangleright	Several types of	focular medications	are used to treat glaucoma.
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S.	NAMEOF DOSE		ACTION	SIDE EFFECT	NSG.					
N.	DRUG				RESPONSIBILIT Y					
1.	Cholinergic	1%, 2%	- It increases aqueous	- Per orbital	- Caution pts					
	(Miotics):	or 4%/	fluid outflow by	pain, blurry	about					
	Pilocarpine,	3-4	contracting the ciliary	vision, difficulty	diminished					
	Carbachol	times/	muscles & causing	seeing in the	vision in dimly					
		day	meiosis (constriction of	dark.	lit areas.					
			the pupil) & opening of							
			the trabecular							
		0.504	meshwork.							
2.	Adrenergic agonists :	0.5%, 1% or	Peduces preduction of	- Eye redness &	- Teach pt punctual					
	Dipivefrin,	2%/1-	 Reduces production of aqueous humor & 	burning, anxiety palpitation,	occlusion to					
	Epinephrine	2 times	increases outflow.	elevated B.P.,	limit systemic					
	epinepinite	/day	increases outriow.	headache &	effects.					
3.	Beta	0.25 or		- Bradycardia,	Contraindicated					
	blockers :	0.5%	- Decreases aqueous	hypotension	in pts with					
	Betaxolol,	/2	humor production.		asthma, COPD					
	Timolol	times			or cardiac					
		/day			failure.					
4.	Alpha	0.5%/		- Eye redness,	- Teach pt					
	adrenergic	2-3	- Decreases aqueous	dry mouth &	punctual					
	agonists :	times/	humor production.	nasalpassage	occlusion to					
	Apraclonidin	day			limit systemic					
	Brimonidine				effects					
5.	Carbonic	250 mg		- Electrolyte loss,	- Monitor					
	anhydrase	/tds or	- Decreases aqueous	depression,	electrolyte					
	inhibitors :	qid	humor production.	lethargy, GI	levels & Do not					
	Acetazolami			upset, weight	administer to					
	de, methazolam			loss & topical	pts with sulfa					
	ide			allergy	allergies					
	ide									

I



C .Laser surgery

This procedure can slightly raise flow of fluid from eye in open-angle glaucoma. It can stop fluid blockage in angle-closure glaucoma. Procedures include:

Trabeculoplasty - This opens the drainage area.

- Iridotomy This makes a tiny hole in your iris to let fluid flow more freely for papillary block glaucoma ,contraindicated in pts with corneal edema. complication is burns to cornea, lens or retina, transient elevated IOP.
- Cyclophotocoagulation This treats areas of middle layer of your eye to lower fluid production.



D .Laser-assisted nonpenetrating deep sclerectomy

Most common surgical approach currently used for treatment of glaucoma is trabeculectomy, in which the sclera is punctured to alleviate intraocular pressure.

- (NPDS) surgery is similar, but modified, procedure, in which instead of puncturing scleral bed and trabecular meshwork under a scleral flap, a second deep scleral flap is created, excised, with further procedures of deroofing Schlemm's canal, upon which, percolation of liquid from the inner eye is achieved and thus alleviating IOP, without penetrating eye. NPDS have few side effects. NPDS is performed manually and requires higher level of skills that may be assisted with instruments. In order to prevent wound adhesion after deep scleral excision and to maintain good filtering results, NPDS is performed with a variety of biocompatible spacers / devices, such as Aqua flow collagen wick, ologen Collagen Matrix or Xenoplast glaucoma implant
- Laser-assisted NPDS is performed with use of a CO₂ laser system. Laser-based system is self-terminating once required scleral thickness and adequate drainage of intraocular fluid have been achieved. This self-regulation effect is achieved as CO₂ laser essentially stops ablating as soon as it comes in contact with intraocular percolated liquid, which occurs as soon as the laser reaches the optimal residual intact layer thickness.

Treatment

- **E. Microsurgery.** In trabeculectomy doctor creates a new channel to drain fluid and ease eye pressure. This form of surgery may need to be done more than once. Doctor might implant a tube to help drain fluid. This surgery can cause temporary or permanent vision loss, as well as bleeding or infection.
- **F.Canaloplasty** .A nonpenetrating procedure using micro catheter technology. To perform a canaloplasty, an incision is made into eye to gain access to Schlemm's canal in a similar fashion to a viscocanalostomy. A micro catheter will circumnavigate the canal around iris, enlarging main drainage channel and its smaller collector channels through injection of a sterile, gel-like material called viscoelastic. The catheter is then removed and a suture is placed within the canal and tightened.
- By opening canal, the pressure inside eye may be relieved, although reason is unclear, since canal of Schlemm does not have any significant fluid resistance in glaucoma or healthy eyes

Treatment

- G.Drainageimplants or shunts:- These are an open tubes implanted in anterior chamber to shunt aqueous humor to an attached place in conjunctiva space. A fibrous capsule develops around episcleral plate & filters aqueous humor, thereby regulating the outflow & controlling IOP.
- * first-generation Molteno and other nonvalved implants sometimes require ligation of tube until bleb formed is mildly fibrosed and water-tight. This is done to reduce postoperative hypotony—sudden drops in postoperative IOP.
- *Valved implants-Ahmed glaucoma valve, attempt to control postoperative hypotony by using mechanical valve.
- *Ab interno implants- Xen Gel Stent, are trans scleral implants by an ab interno procedure to channel aqueous humor into non-dissected Tenon's space, creating a sub conjunctival drainage area similar to a bleb. The implants are trans scleral and different from other ab interno implants that do not create a trans scleral drainage, such as iStent, CyPass, or Hydrus.
- H.<u>Filtering procedures</u> :- (for chronic glaucoma) used to create an opening or fistula in trabecular meshwork to drain aqueous humor from the anterior chamber to sub conjunctival space, thereby bypassing the usual drainage structures. This allows aqueous humor to flow & exit by different routes.

NURSING ASSESSMENT

Child may need examination under anesthesia

- History on lacrimation, photophobia, and blepharospasm
- Assess visual acuity and perform refraction to find out loss of vision
- Examine cornea for edema and opacity
- Measure IOP with hand held perkin's applanation tonometer
- Measure corneal diameter by calipers.
- Perform a dilated fundus examination to evaluate the optic disc and retina

NURSING DIAGNOSIS

- Altered visual perception secondary to increased intraocular pressure and manifested as Profound lacrimation, photophobia, corneal haze, and buphthalmos
- Loss of vision

EXPECTED OUTCOME

- Intraocular pressure will be controlled and bring down to normal.
- Lacrimation will be controlled.
- Photophobia and corneal haze will be eliminated
- Prevent eye from loss of vision.



Intervention

- Counsel the child's parents for urgent need of surgery
- Prepare them psychologically
- Check the ophthalmologic order of management plan
 - Topical beta-blocker (timolol 0.25% to 0.50% b.i.d.)
 - Goniotomy is the first choice of surgery (Clear cornea)
 - Trabeculotomy
 - corneal clouding prevents visualization of the angle
 - Failed repeated goniotomy
- Monitor IOP, optic disc, and corneal diameter on regular follow up

EVALUATION

Outcome criteria

Cornea should be transparent; and IOP should be maintained with in normal range

- 1. **Nursing diagnosis**:-Acute pain related to increased intra-ocular pressure.
- Assessment :- Evaluate patient for severe pain.
- Nursing goal :- Provide medication, thereafter Client will have Reducing pain.
- Nursing Intervention :- Administered Opioids & other medications as directed.
- *Explain patient that goal of treatment is to reduce IOP as quickly as possible.
- *Reassure patient that, with reduction in IOP, pain & other sign & symptoms should subside.
- *Opioids reduce pain.
- * To reduce anxiety, reassurance is essential to reduce fear & anxiety of patient.
- * Fear & anxiety increases perception of pain.
- **Evaluation** :- Patient will relieve from pain, after giving opioids.

2.Nursing diagnosis: - Fear related to pain & potential loss of vision

- Assessment: Assess patient's level of anxiety & knowledge.
- Nursing goal: Provide emotional support, thereafter Client will have Reducing fear
- Nursing intervention: Provide reassurance & calm presence to reduce anxiety & fear *Provide emotional support.
- * Reassurance is essential to reduce fear & anxiety of the pt.
- * Emotional support is essential to reduce fear & anxiety of the patient.
- * Fear & anxiety increases the perception of pain.
- **Evaluation**:-Patient's fear & anxiety will reduce.

- 3. **Nursing diagnosis:** Nausea & vomiting related to opioids & other medications.
- Assessment: Evaluate patient for nausea & vomiting
- Nursing goal: Provide antiemetic drugs, thereafter Client will have Relieving from Nausea & vomiting.
- Nursing intervention: Patient may be medicated with antiemetic.
- *Explain patient & provide support.
- *Antiemetic reduced nausea & vomiting.
- * Explanation & support reduce the fear & anxiety.
- **Evaluation**:- Patient will relieve from Nausea & vomiting, after taking antiemetic Drugs

- 4. **Nursing diagnosis:** Knowledge deficit related to disease.
- Assessment: Assess level of knowledge of patient regarding disease
- Nursing goal: Provide knowledge regarding glaucoma.
- Nursing intervention: Provide knowledge regarding glaucoma, their sign & symptoms & Management.
- *Provide knowledge regarding medication & their side effects.
- * Patient is able to understand regarding disease.
- *Patient is able to understand regarding medication.
- **Evaluation**: Patient is able to understand regarding glaucoma.

Bibliography

- BLACK JOYCE M., HOWKS JANEHOKANSON (2009), MEDICAL SURGICAL NURSING, 8TH EDITION, VOLUME-2, NEW DELHI : ELSEVIER
- 2) SMELTZER SUZANNEC., BARE BRENDA (2004), BRUNNER& SUDDARTH'S TEXTBOOK OF MEDICAL SURGICAL NURSING, 10TH EDITION, LONDON: LIPPINCOTTWILLIAMS & WILKINS.
- 3) WAUGH ANNE, ALLISONGRANT (2007), ROSS AND WILSONANATOMYAND PHYSIOLOGYINHEALTH & ILLNESS, 10TH EDITION, LONDON: ELSEVIER
 - 4) www.wrongdiagnosis.com

Bibliography

- www.ehealthmd.com
- www.glaucomafoundation.org
- www.webmd.com
- www.emedicinehealth.com
- en.wikipedia.org
- www.answer.com
- www.enotes.com
- Medicaldictionary.thefreedictionary.com
- www.patient.co.uk
- www.healthline.com



THANK YOU