"To the ER or Not to the ER..."

An evidence-based approach to ocular emergencies

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I have no financial disclosures

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Before we get started...

- Not an all-inclusive list of "ocular emergencies"
- Focus on the "why" instead of the "what"
- Although these are almost all my patient examples most are not actually my patients' photos

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Course Objectives

By the end of this presentation, participants will be able to:

- Recognize ocular signs and symptoms that indicate a need for emergency room referral.
- Identify vision-threatening and life-threatening ocular conditions.
- Understand the systemic diseases associated with these presentations.
- Understand the evidence-based rationale for emergency room referrals including urgent imaging.
- Implement a systematic approach to evaluating urgent ocular presentations to avoid delayed or inappropriate referrals.

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Why send someone from your chair to the ER?

- Needs stat imaging
- Concern for imminent systemic threat
- Vision threatening without immediate treatment

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Emergency Department Wait Times

Table 3. Wait time at emergency department visits: United States, 2022

Visit characteristic	Number of visits (standard error) in thousands	Percent distribution (standard error)
Time spent waiting to see a physician, APRN, or PA[1]	156,398 (10,251)	100.0 ...
Less than 15 minutes		63,084 (5,607)	40.6 (2.5)
15–59 minutes		46,195 (4,352)	29.7 (1.5)
3 hours, but less than 4 hours	2,538 (534)	1.6 (0.3)
4 hours, but less than 6 hours	1,535 (304)	1.0 (0.2)
	915 (202)	0.6 (0.1)
	5,044 (523)	3.2 (0.3)
Time spent in the emergency department	17,106 (3,189)	11.0 (2.0)
1 hour, but less than 2 hours		28,612 (2,787)	18.4 (1.1)
2 hours, but less than 4 hours		51,510 (3,953)	33.1 (1.1)
4 hours, but less than 6 hours		27,401 (2,449)	17.6 (0.9)
6 hours, but less than 10 hours		17,740 (1,573)	11.4 (0.9)
24 hours or more	2,544 (333)	1.6 (0.2)
Blank	6,421 (1,647)	4.1 (1.1)
Patient arrived in emergency department after business hours[2]		
Yes	87,511 (5,953)	56.3 (0.7)
No	65,774 (4,558)	42.3 (0.7)
Blank	2,113 (477)	1.4 (0.3)

... Category not applicable.
[1]APRN is advanced practice registered nurse. PA is physician assistant. The median wait time to see a physician, APRN, or PA was 18.0 minutes; the mean wait time to see a physician, APRN, or PA was 38.1 minutes.
[2]Business hours are defined as Monday through Friday, 8 a.m. to 5 p.m.
NOTE: Numbers may not add to totals due to rounding.
SOURCE: National Center for Health Statistics, National Hospital Ambulatory Medical Care Survey, 2022.

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- **59yo AAF**
- **CC:** loss of vision in right eye and right sided headache
- **PMHx:** hypertension, hyperlipidemia, prediabetes, h/o smoking, obesity, anxiety disorder
- **Acuity:** 20/800 OD, 20/20 OS
- **Pupils:** Round OU, sluggish OD, +APD OD
- **IOP:** 18/17

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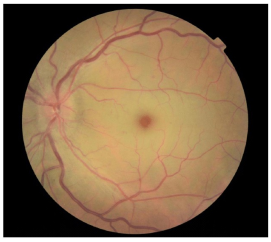
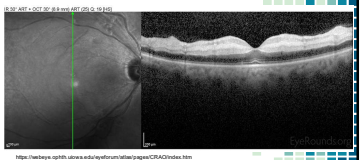


https://www.researchgate.net/figure/Fig-1-Central-retinal-artery-occlusion-Note-pale-retina-narrowed-arterioles-and_fig3_316279154



https://webeye.ophth.uiowa.edu/eyeforum/atlas/pages/CRAO/index.htm

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Once you finish everything you need for your exam, how many people would send this patient to the ER?

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Central Retinal Artery Occlusion

- Incidence 1.9 per 100,000 in the US
  - Increases to 10 per 100,000 over age 80
- Mean age 60-65 years old
- Men > women
- Vasculopaths
  - Risk factors: diabetes, hypertension, hyperlipidemia, h/o smoking

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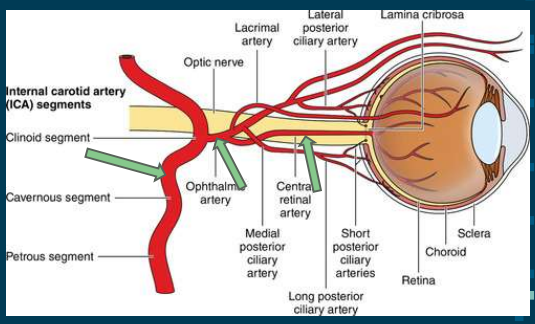

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CRAO Etiology

- Arteritic vs Nonarteritic
- >40yo: carotid artery atherosclerosis
- <40yo: cardiogenic embolism
- Other: hematologic disease, inflammatory disease, rarely infection or secondary to ocular surgery/injection

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CRAO Treatment

- Minimal benefit with in-office treatments
- What about tPA?
  - *Schrag et al.* 50% rate of visual recovery if given **within 4.5 hours** of onset
    - "Visual recovery" defined as 20/100 or better
  - 3 randomized trials being conducted in Europe
  - Risk: intracranial hemorrhage
  - tPA directly into the ophthalmic circulation?

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What's the systemic risk?

- Internal carotid disease
  - 103 cases of CRAO at a single center
    - **37%** had "ipsilateral *critical* carotid disease"
      - (≥70% stenosis, arterial dissection, or intra-arterial thrombus)
  - EAGLE study: 84 patients
    - **40%** had ≥70% stenosis
  - Large US inpatient database:
    - 17,117 inpatient CRAO admissions
    - **22.1%** of patients with carotid artery stenosis
- Stroke

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Test Finding	Non-arteritic CRAO	BRAO*
Patent foramen ovale	(n = 234)	(n=141)
	6 (2%)	3 (2%)
Carotid Doppler/angiography:	(n=199)	(n=128)
Occlusion		
0-15%	109 (55%)	69 (54%)
16%-49%	23 (12%)	21 (16%)
50%-79%	32 (16%)	21 (16%)
80%-99%	13 (7%)	11 (9%)
100%	22 (11%)	6 (5%)
Carotid Doppler/angiography:	(n=187)	(n=123)
Plaque present	133 (71%)	81 (66%)
Echocardiogram	(n=131)	(n=76)
Normal	39 (30%)	32 (42%)
Abnormal, no embolic source	24 (18%)	12 (16%)
Abnormal, with embolic source	68 (52%)	32 (42%)

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Study	# Patients	Timeframe	CVA Incidence	Other
Lee et al. (2014)	33 RAO patients	7 days following visual symptoms	8/33 **(24.2%)** - 5 CRAO, 3 BRAO	Neurologic signs/symptoms in 62% of the CVA group vs. 0% of the non-CVA group
Chang et al. (2012)	3248 total patients	3 years following RAO	371 CVA patients - 91/464 **(19.6%)** RAO patients - 280/2784 (10.1%) control patients	Risk of developing stroke highest in the first month CRAO patients higher CVA incidence vs. BRAO
Park et al. (2015)	1655 CRAO patients	Unclear	165/1655 **(10%)** with CVA/MI - IRR 14.68 1-30 days after CRAO - IRR 7.14 1-30 days prior - IRR 2.99 31-90 days prior	
Mayo Clinic (2019)	300 CRAO patients	15 days before/15 days after CRAO	- 16/300 **(5.3%)** with symptomatic ischemic CVA - 10/94 with asymptomatic diffusion restriction	7 CVA within 15 days prior to CRAO, 4 simultaneously, 5 after CRAO

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More on Stroke Risk

- Mir et al. 2019 AJO
- 17,117 patients admitted for CRAO
- Higher risk for stroke:
  - Female
  - Hypertension
  - Carotid artery stenosis
  - Aortic valve disease
  - H/o smoking
  - H/o alcohol dependence

	N (%) (N = 17 117 Patients)
Stroke	2202 (12.9)
Ischemic stroke	2080 (12.2)
Hemorrhagic stroke	142 (0.8)
TIA	428 (2.5)
Carotid endarterectomy and carotid artery stent	1157 (6.8)
Acute MI	639 (3.7)
Cardiac intervention[a]	430 (2.5)
Systemic fibrinolytic therapy	494 (2.9)
Died during hospitalization	222 (1.3)
Combined risk of stroke, TIA, MI, or death	3248 (19.0)

MI = myocardial infarction; TIA = transient ischemic attack.

[a]Percutaneous coronary intervention; coronary artery bypass grafting.

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Stroke Risk Controversy

- Cleveland Clinic "*Retinal artery occlusion does not portend an increased risk of stroke*"
  - 221 patients with RAO diagnosed by FA
    - 5 patients (**2.3%**) developed CVA
    - 8 patients (3.6%) had MI
    - 22 patients (**14.2%**) had >50% internal carotid stenosis
- Hayreh *"Do Patients with Retinal Artery Occlusion Need Urgent Neurological Evaluation?"*
  - 196 RAO patients
    - **7%** incidence of symptomatic TIA/CVA in CRAO
    - **3%** incidence of symptomatic TIA/CVA in BRAO

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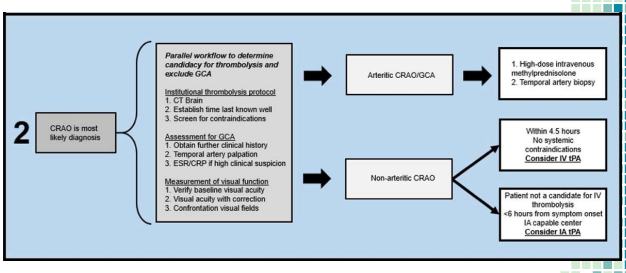
Referral



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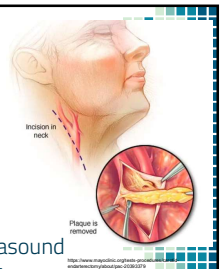
Hollenhorst Plaques

- Why work these patients up?
- Bakri et al.
  - 237 patients with Hollenhorst plaque
    - 60 (25.3%) symptomatic
    - 177 (74.7) asymptomatic
  - 83.5% of patients underwent carotid ultrasound
    - **97.4% had some degree of atherosclerosis**
      - Of patients who had >69% stenosis, 25% were symptomatic and 9.2% were asymptomatic



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Hollenhorst Plaques

- What about stroke risk?
- Egan & Lutsep
  - 65 patients with ischemic CVA diagnosis
    - 10/65 patients with visible cholesterol emboli on ocular examination

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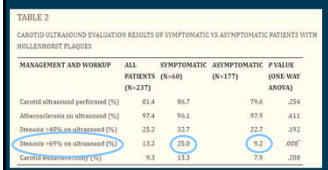

TABLE 2

CAROTID ULTRASOUND EVALUATION RESULTS OF SYMPTOMATIC VS ASYMPTOMATIC PATIENTS WITH HOLLENHORST PLAQUES

MANAGEMENT AND WORKUP	ALL PATIENTS (N=237)	SYMPTOMATIC (N=40)	ASYMPTOMATIC (N=177)	P VALUE (ONE-WAY ANOVA)
Carotid ultrasound performed (%)	81.4	86.7	79.6	.254
Atherosclerosis on ultrasound (%)	97.4	96.1	97.9	.611
Stenosis >40% on ultrasound (%)	25.2	32.7	22.7	.192
Stenosis >69% on ultrasound (%)	13.2	25.0	9.2	.008*
Carotid endarterectomy (%)	9.3	13.3	7.9	.208

Bakri SJ et al. Is Carotid Ultrasound Necessary in the Clinical Evaluation of the Asymptomatic Hollenhorst Plaque? (An American Ophthalmological Society Thesis)

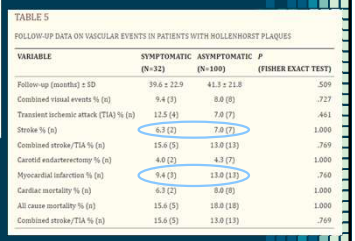

TABLE 5

FOLLOW-UP DATA ON VASCULAR EVENTS IN PATIENTS WITH HOLLENHORST PLAQUES

VARIABLE	SYMPTOMATIC (N=32)	ASYMPTOMATIC (N=100)	P (FISHER EXACT TEST)
Follow-up (months) ± SD	39.6 ± 22.9	41.3 ± 21.8	.509
Combined visual events % (n)	9.4 (3)	8.0 (8)	.727
Transient ischemic attack (TIA) % (n)	12.5 (4)	7.0 (7)	.461
Stroke % (n)	6.3 (2)	7.0 (7)	1.000
Combined stroke/TIA % (n)	15.6 (5)	13.0 (13)	.769
Carotid endarterectomy % (n)	6.0 (2)	4.3 (7)	1.000
Myocardial infarction % (n)	9.4 (3)	13.0 (13)	.760
Cardiac mortality % (n)	6.3 (2)	8.0 (8)	1.000
All cause mortality % (n)	15.6 (5)	18.0 (18)	1.000
Combined stroke/TIA % (n)	15.6 (5)	13.0 (13)	.769

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- **78yo WF**
- **CC:** sudden onset loss of vision in the left eye with left sided headache
- **PMHx:** hypertension, hyperlipidemia, hypothyroidism
- **VA:** 20/30 OD, HM OS
- **Pupils:** Round OU, brisk OD, sluggish OS, +APD OS
- **Color vision:** 14/14 OD, 0/14 OS
- **IOP:** 15/15

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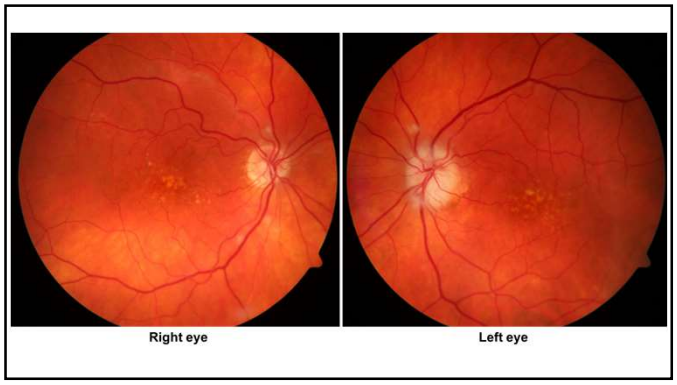

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Would you send this patient to the ER?

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Next steps...

Obtain	Order	Start
Obtain ESR/CRP/CBC	Order temporal artery biopsy	Start high dose systemic steroids as soon as possible

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Giant Cell Arteritis

- Inflammatory disease affecting medium to large blood vessels
- 2.3 per 100,000 cases per year in the sixth decade of life
  - 44.7 per 100,000 cases per year in patients in their ninth decade
- Older patients (generally >70yo), Caucasians, Females
- Most common ophthalmic manifestations: AAION, CRAO

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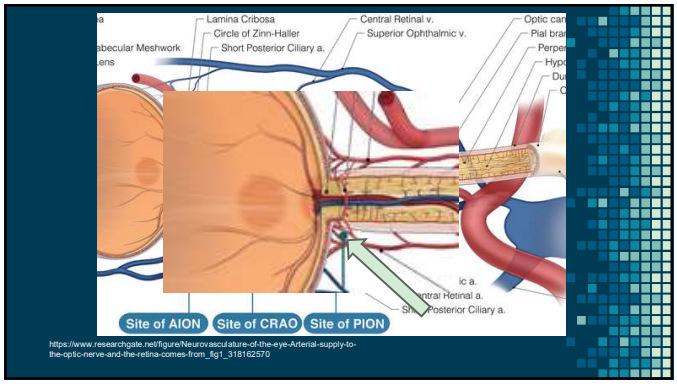

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Odds of Positive Biopsy

- Jaw claudication: 9x greater
- Neck pain: 3.4x greater
- CRP > 2.45mg/dl: 3.2x greater
- ESR >47mm/hour: 2.0x greater
- Age >75yo: 2.0x greater

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Evidence for Urgent Intervention

Study	# Patients	Vision Loss	Time to Vision Loss
Aiello et al. (1993)	245 patients	34 **(14%)** with permanent VL	Unknown
Gonzalez-Gay et al. (2004)	239 patients	34 **(14.2%)** with permanent VL - 11 **(4%)** with bilateral permanent VL	5 days (range 3-14 days)
Hayreh et al. (2003)	144 patients	91 **(63%)** with VL *permanence not specified - 9/91 (9.8%) with further VL after therapy	Within 5 days of starting therapy

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What Does this Data Suggest?

- If normal vision at diagnosis and appropriate treatment initiated immediately, visual loss is highly unlikely
- When GCA is suspected, steroid therapy should begin ASAP
- Guarded prognosis during the first week of steroid therapy

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GCA Treatment

- Corticosteroids
- No statistically significant difference in initial treatment of pulse IV doses vs oral steroids
- Tocilizumab

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What's tocilizumab?

- Monoclonal antibody targeting the IL-6-receptor
- Initiated as a second-line treatment for systemic GCA
- What is the frequency of vision loss in patients with known GCA already treated with steroids + tocilizumab?
  - 186 patients between 2010 – 2018
  - 11% had vision loss at baseline
  - 2 patients developed vision loss while on TCZ

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- **29yo WF**
- **CC:** "dimming of vision OD>OS that comes and goes, reduced hearing right side"; denies headaches
- **PMHx:** BMI 36.6
- **VA:** 20/25+ OD, 20/20 OS
- **Color vision:** 14/14 OD, 14/14 OS
- **IOP:** 14/14
- **Pupils:** PERRL -APD

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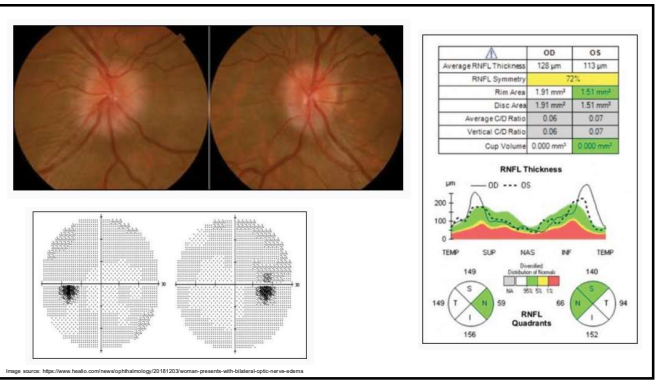

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At this point, would you send this patient to the ER?

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Idiopathic Intracranial Hypertension

- Incidence: 1-2 per 100,000
  - Increases to 4-21 per 100,000 if you filter for overweight women of childbearing age
- Can be triggered by: weight gain, pregnancy, iron-deficiency anemia, tetracycline antibiotic use, or use of systemic and topical vitamin-A derivatives

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Symptoms of IIH

Symptom	Percentage of patients
Headache	84%
Transient visual obscurations	68%
Back pain	53%
Pulsatile tinnitus	52%
Photopsia	48%
Retrobulbar pain	44%
Neck pain	41%
Sustained visual loss	26%
Diplopia	18%

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IIH Workup

MRI brain and orbits with and without contrast

↓

MRV

↓

LP with CSF studies

Diagnostic criteria:

- ✓ Papilledema or sixth nerve palsy
- ✓ Otherwise normal neurologic exam
- ✓ Neuroimaging (MRI with and without gadolinium and MRV is preferred) shows normal brain without evidence of hydrocephalus, mass, structural lesion, or meningeal enhancement
- ✓ Normal CSF composition
- ✓ Elevated LP opening pressure

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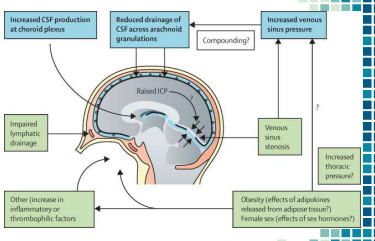
Other causes of intracranial hypertension

- Intracranial mass
- Obstruction of venous outflow (i.e. venous sinus thrombosis)
- Obstructive hydrocephalus
- Decreased cerebrospinal fluid (CSF) absorption
- Increased CSF production



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Patient presents with disc edema, what is the likelihood it's IIH?

- IIH: 58 patients (87%)
  - Higher median BMI and headache prevalence
  - Increases to 95% when filtering for patient demographics
- Alternate etiology: 9 patients (13%)
  - Intracranial tumor, cerebral venous sinus thrombosis, granulomatous meningitis
  - 2/9 had other neurologic signs

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Table 1. Etiologies of Papilledema

Characteristic	Patients presenting with papilledema, No. (%) (N = 86)							
	Previously diagnosed				Undiagnosed at time of papilledema			
	Intracranial tumor (n = 10)	Intracranial hemorrhage (n = 6)	Venous sinus thrombosis (n = 2)	Neurosarcoidosis (n = 1)	Idiopathic intracranial hypertension (n = 58)	Intracranial tumor (n = 4)	Venous sinus thrombosis (n = 4)	Granulomatous meningitis (n = 1)
Women	5 (50)	1 (17)	2 (100)	1 (100)	53 (91)	2 (50)	3 (75)	1 (100)
Age, median (range), y	38.2 (7.9-58.0)	45.7 (15.2-64.2)	26.0 (24.1-27.9)	41.6 (NA)	27.3 (11.8-48.7)	15.0 (7.0-41.9)	28.4 (6.2-48.5)	26.6 (NA)
BMI, median (range)	23.3 (14.2-38.8)	27.6 (16.7-46.9)	40.3 (39.6-41.0)	25.6 (NA)	37.5 (20.4-55.7)[a]	25.4 (16.7-40.1)	29.8 (16.6-35.1)	33.0 (NA)
Headaches	6 (60)	4 (67)	2 (100)	1 (100)	54 (93)	3 (75)	2 (50)	1 (100)
Focal neurologic deficits	4 (40)	0	1 (50)	0	0	2 (50)	0	0

Abbreviation: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); NA, not applicable.

[a] Data were not available for 11 patients.

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Same question, but in a neuro-ophthalmologist's chair

- Jhaveri et al. (2025) studied the leading causes of optic disc edema in one neuro-ophthalmology clinic in Toronto (654 patients)

TABLE 1. Leading causes of optic disc edema in age group

Age Group, yrs	Top 3 Causes of Optic Disc Edema (n, %)
Younger than 18	1. IIH papilledema (n = 17, 85%) 2. Optic neuritis (n = 1, 5%) 3. Accutane use (n = 1, 5%) 4. Non-IIH papilledema (n = 1, 5%)
18–30	1. IIH papilledema (n = 167, 79%) 2. Non-IIH papilledema (n = 20, 9.4%) 3. ON (n = 14, 6.6%)
31–40	1. IIH (n = 97, 67.8%) 2. ON (n = 15, 9%) 3. Miscellaneous causes of ODE (n = 14, 9.7%)
41–50	1. IIH (n = 45, 47.4%) 2. Non-IIH papilledema (n = 17, 18%) 3. NAION (n = 13, 13.4%)
51-60	1. NAION (n = 27, 35%) 2. IIH (n = 19, 24.7%) 3. Non-IIH papilledema (n = 12, 15.6%)
61–70	1. NAION (n = 30, 61.2%) 2. Other (n = 8, 16.3%) 3. Non-IIH papilledema (n = 6; 12.2%)
71–80	1. NAION (n = 24, 63.2%) 2. Other (n = 7, 18.4%) 3. Non-IIH papilledema (n = 3, 7.9%)
81–90	1. NAION (n = 13, 65%) 2. AAION (n = 5, 25%)

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Cause	# of patients
IIH	351 (53.7%)
NAION	116 (17.4%)
Non-IIH Papilledema	71 (10.9%)
Optic neuritis	46 (7.0%)
Uveitis	17 (2.6%)

Cause	# of patients
Dural/cerebral venous sinus thrombosis	15 (21.2%)
Brain mass	12 (16.9%)
Medication induced	7 (9.9%)
Infectious	7 (9.9%)
Other	30 (42.3%)

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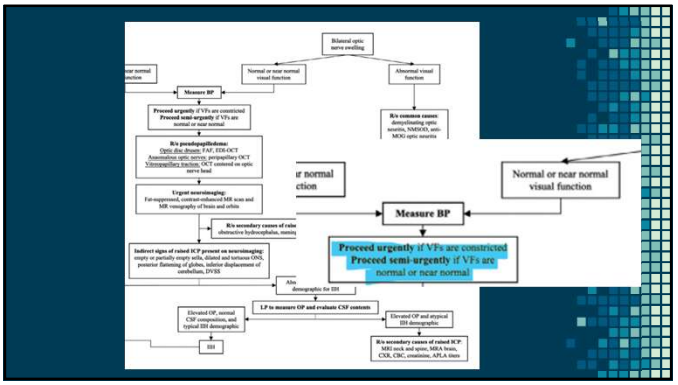

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IIH Treatment

- IIHTT: acetazolamide + low sodium diet vs. placebo + low sodium diet
  - Both groups experienced improvement at month 6
  - Treatment failure: worsening of mean deviation by 2 or 3dB on HVF
    - Only occurred in 7 eyes
      - 1 in the acetazolamide group vs 6 in the placebo group
    - Risk factors for treatment failure:
      - Grade 3-5 edema on Frisen scale
      - Increased frequency of TVOs
      - Headaches

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Fulminant IIH

- Severe form of IIH
- <4 weeks between initial onset of symptoms and severe vision loss
- Emory University Study
  - 14/483 patients with diagnosed IIH had fulminant IIH **(2.9%)**
  - Severe loss of acuity, severe papilledema, severe visual field constriction noted in all cases
  - Mean **16.1 days** from symptom onset → worst visual loss
  - Surgery almost always recommended in these cases
  - Visual function improved in all patients following surgery though 8 remained legally blind

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So…when should I send suspected IIH patients to the ER?

- Unable to easily obtain outpatient imaging
- Severe or worsening acuity, field loss, or disc edema
- Severe headache or other neurologic symptoms
- Any associated diplopia/cranial nerve palsy
- Suspicion of alternate underlying etiology

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- **82yo AAF**
- **CC:** "droopy eyelid" left eye x 2-3 weeks
- **MHx:** Type II DM, arthritis, hyperlipidemia, hypertension, chronic kidney disease
- **VAcc:** 20/40 OD, 20/50 OS
- **IOP:** 11/10
- **Ocular health exam:** mixed cataracts OU, otherwise unremarkable

*when you lift the "droopy" left eyelid, the patient then complains of double vision

Pupils: PERRL –APD, NO anisocoria

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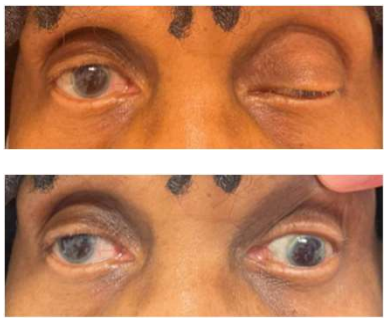

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How many people would send this patient to the ER?

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Let's change it up…

- What if her pupils were actually:
- OD: 5mm dim, 3mm bright
- OS: 5mm dim, 4.5mm bright

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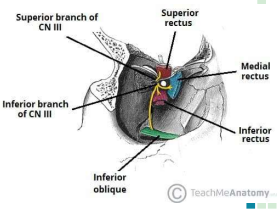
Acquired Cranial Nerve 3 Palsy

- Complete: complete ptosis, total inability to adduct, infraduct, or supraduct, dilated pupil with sluggish reaction
- Partial: variable duction limitations, variable degrees of ptosis or pupil involvement
- Isolated vs nonisolated



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What's the most urgent etiology of CN3 palsy?

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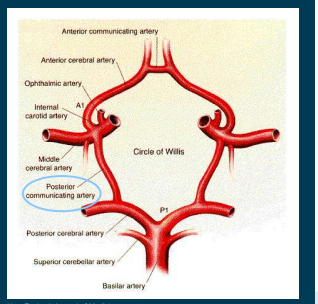

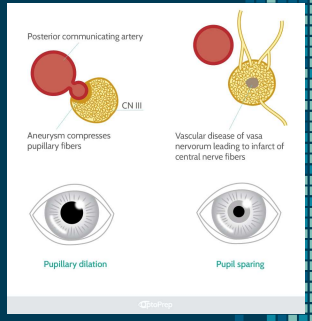

New England Journal of Medicine

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Table. Characteristics of Acquired Third Nerve Palsy*

Cause	Cases, No.	No. (%) Pupil Involvement	Ptosis	Complete External Third Nerve Dysfunction	Recovery	Neurologically Isolated (Not Including Eye Pain or Headache)	Eye Pain and/or Headache	Aberrant Regeneration
Microvascular	61	10 (16)	56 (92)	20 (33)	58 (95)	58 (95)	37 (61)	0
Stroke	6	4 (67)	4 (67)	1 (17)	4 (67)	1 (17)	3 (50)	0
Compression	25	16 (64)	20 (80)	6 (24)	6 (24)	9 (36)	15 (60)	4 (16)
Aneurysm	9	3 (33)	8 (89)	2 (22)	3 (33)	6 (67)	7 (78)	1 (11)
Meningioma	5	4 (80)	3 (60)	2 (40)	2 (40)	0	1 (20)	0
Metastasis	5	4 (80)	4 (80)	1 (20)	0	1 (20)	4 (80)	0
Pituitary adenoma	2	2 (100)	2 (100)	1 (50)	0	1 (50)	0	0
Other	4	3 (75)	3 (75)	0	1 (25)	1 (25)	3 (75)	3 (75)
Trauma	18	14 (78)	16 (89)	3 (17)	4 (22)	4 (22)	17 (95)	4 (22)
MVC	12	12 (100)	10 (83)	3 (25)	2 (17)	0	12 (100)	4 (33)
Other	6	2 (33)	6 (100)	0	2 (33)	4 (67)	5 (83)	0
Postneurosurgery	14	10 (71)	12 (86)	4 (29)	5 (36)	3 (14)	11 (79)	2 (14)
Clipping of aneurysm	6	4 (67)	4 (67)	0	2 (33)	0	6 (100)	0
Meningioma	2	1 (50)	2 (100)	1 (50)	0	1 (50)	0	0
Other	6	5 (83)	6 (100)	3 (50)	3 (50)	1 (17)	5 (83)	2 (33)
Undetermined	6	1 (17)	4 (67)	1 (17)	3 (50)	4 (67)	3 (50)	1 (17)
Pituitary apoplexy	3	2 (67)	3 (100)	2 (67)	2 (67)	0	3 (100)	0
Tolosa–Hunt syndrome	3	0	3 (67)	0	3 (100)	0	2 (67)	0
Giant cell arteritis	2	0	2 (100)	0	2 (100)	1 (50)	2 (100)	0

Abbreviation: MVC, motor vehicle crash.
* Percentages do not equal 100% owing to rounding. There was a single case of carotid-cavernous sinus fistula, hemorrhage, zoster, carcinomatous meningitis, ophthalmoplegic migraine, postviral palsy, and cavernous sinus thrombosis causing third nerve palsy.

Fang et al, Incidence and Etiologies of Acquired Third Nerve Palsy Using a Population-Based Method

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Table 1. Causes of neurologically isolated third cranial nerve dysfunction

Cause	Total	Age <49 years	Age ≥50 years
Vascular	22	4	18
Aneurysm	10	0	10
Head trauma	10	8	2
Neoplasm	5	3	2
Congenital	1	1	0
Others	15	8	7
Total	63	24	39

Akagi et al, Cause and Prognosis of Neurologically Isolated Third, Fourth, or Sixth Cranial Nerve Dysfunction in Cases of Oculomotor Palsy

Table 2. Presence of ptosis and pupillary involvement in third cranial nerve dysfunction

Cause	Ptosis (+)	Ptosis (−)	Anisocoria	Isocoria
Vascular	18	4	7*	15
Aneurysm	9	1	9	1
Head trauma	9	1	9	1
Neoplasm	4	1	3	2
Others	15	1	7	9
Total	55	8	35	28

*In four cases, the difference in diameter was <0.5 mm, and in three it was <1.0 mm.

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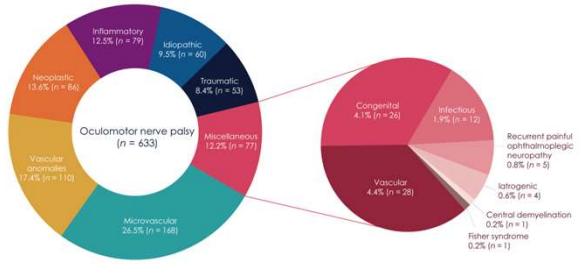

FIGURE 3 Etiological distribution of isolated oculomotor nerve palsy. The most common etiology was microvascular followed by vascular anomalies, neoplastic, inflammatory, idiopathic and traumatic. These six etiologies overall explained 87.8% of isolated oculomotor nerve palsy.

Kim et al, Etiological distribution of isolated oculomotor nerve palsy: analysis of 633 patients and literature review

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TABLE 2 Previous studies on the etiology of isolated oculomotor nerve palsy.

	Country	Publication	Study duration	Age	No. of cases	Microvascular no. (%)	Idiopathic no. (%)	Aneurysm no. (%)	Trauma no. (%)	Neoplasm no. (%)	Miscellaneous no. (%)
Rucker [5]	USA	1958	1945–1956		335	63 (18.8)	95 (28.4)	64 (19.1)	51 (15.2)	35 (10.4)	27 (8.1)
Goldstein and Cogan [6]	USA	1960			61	28 (45.9)	7 (11.5)	11 (18.0)	5 (8.2)	6 (9.8)	4 (6.6)
Green et al. [7]	USA	1964		0–70	130	25 (19.2)	31 (23.8)	38 (29.2)	14 (10.8)	5 (3.8)	17 (13.1)
Rucker [8]	USA	1966	1958–1964		274	47 (17.2)	55 (20.1)	50 (18.2)	34 (12.4)	50 (18.2)	38 (13.9)
Rush and Younge [9]	USA	1981	1966–1978	0–91	290	60 (20.7)	67 (23.1)	40 (13.8)	47 (16.2)	34 (11.7)	42 (14.5)
Berlit [10]	Germany	1990	1966–1978	14–84	172	84 (48.8)	17 (9.9)	16 (9.3)	10 (5.8)	12 (7.0)	33 (19.2)
Ing et al. [11]	Canada	1992	1970–1991	0–17	54	1 (1.9)	2 (3.7)	0 (0.0)	31 (57.4)	2 (3.7)	18 (33.3)
Richards et al. [12]	USA	1992	1978–1988	0–93	231	55 (23.8)	53 (22.9)	25 (10.8)	34 (14.7)	22 (9.5)	42 (18.2)
Akagi et al. [13]	Japan	2008	1993–2001		63	22 (34.9)	0 (0.0)	10 (15.9)	10 (15.9)	5 (7.9)	16 (25.4)
Keane [14]	USA	2010	1971–2007	3–89	500	151 (30.2)	30 (6.0)	101 (20.2)	107 (21.4)	11 (2.2)	100 (20.0)
Kim et al. [15]	Korea	2017	2004–2015		63	40 (63.5)	17 (27.0)	1 (1.6)	3 (4.8)	2 (3.2)	0 (0.0)
Fang et al. [3]	USA	2017	1978–2014	All	145	61 (42.1)	6 (4.1)	9 (6.2)	18 (12.4)	16 (11.0)	35 (24.1)
Choi et al. [16]	Korea	2019	2015	9–88	81	44 (54.3)	9 (11.1)	2 (2.5)	3 (3.7)	1 (1.2)	22 (27.2)
Jung et al. [4]	Korea	2020	2002–2015	All	387	204 (52.7)	100 (25.8)	21 (5.4)	20 (5.2)	30 (7.8)	12 (3.1)
Current study	Korea	2023	2003–2020	0–85	638	168 (26.3)	60 (9.4)	89 (13.9)	53 (8.3)	86 (13.5)	177 (27.7)

Kim et al, Etiological distribution of isolated oculomotor nerve palsy: analysis of 633 patients and literature review

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Cerebral Aneurysm Risk of Morbidity/Mortality

UNRUPTURED INTRACRANIAL ANEURYSMS — RISK OF RUPTURE AND RISKS OF SURGICAL INTERVENTION

The International Study of Unruptured Intracranial Aneurysms Investigators*

- 1449 patients with known aneurysms (all locations)
  - 32 with confirmed rupture (2.2%)
    - **66% fatality rate** in this cohort
- Posterior communicating artery aneurysms are known to have a higher risk of rupture
- Risk of rupture is higher in larger aneurysms

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Cerebral Aneurysm Risk of Morbidity/Mortality

The Natural Course of Unruptured Cerebral Aneurysms in a Japanese Cohort

The UCAS Japan Investigators*

- 5720 patients with 6697 aneurysms
  - 111 aneurysms ruptured (1.66%)
    - In 39 aneurysms, rupture resulted in death **(35%)**
    - In 32 aneurysms, rupture resulted in moderate – severe disability (29%)
- Female gender and hypertension associated with higher risk of rupture
- Aneurysms in anterior communicating and posterior communicating arteries most likely to rupture

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	# patients/ demographics	# ruptured aneurysms	Mortality rate	Morbidity rate	Other findings
Unruptured Intracranial Aneurysms — Risks of Rupture and Risks of Surgical Intervention (NEJM, 1998)	1449 (93% white, 73% female)	32 with confirmed rupture **(2.2%)**	**66%** fatality rate in the ruptured aneurysm cohort	Not documented	• Posterior communicating artery aneurysms are known to have a higher risk of rupture • Risk of rupture is higher in larger aneurysms
The Natural Course of Unruptured Cerebral Aneurysms in a Japanese Cohort (NEJM, 2012)	5720 patients with 6697 aneurysms (all Japanese, 67% female)	111 aneurysms ruptured **(1.66%)**	**35%** fatality rate in the ruptured aneurysm cohort	32 ruptured aneurysms with moderate-severe disability (28%)	• Female gender and hypertension associated with higher risk of rupture • Aneurysms in anterior communicating and posterior communicating arteries most likely to rupture

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Aneurysm surgical intervention

- Surgical clipping
- Endovascular embolization
- Flow diversion

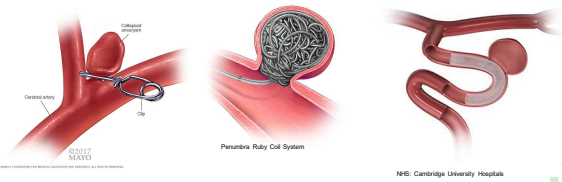

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TABLE 4. Most common complications of initial treatment period after PCoA aneurysm rupture

Parameter	Treatment (no. of patients [% total])				
	Microsurgical	Endovascular	Combined	Indirect Method	Total No. (% total)
No. of patients	517	49	10	6	582
Rebleeding before treatment	113 (22)	9 (18)	3 (30)	1 (17)	126 (22)
Artery occlusion on postop angiography	54 (10)	4 (8)	0 (0)	2 (33)	60 (10)
Occlusive treatment–related ischemia	80 (15)	4 (8)	2 (20)	2 (33)	88 (15)
Postop infection					
Meningitis	31 (6)	3 (6)	1 (10)	0 (0)	35 (6)
Pneumonia	133 (26)	17 (35)	5 (50)	1 (17)	156 (27)
Septicemia	21 (4)	2 (4)	0 (0)	0 (0)	23 (4)
UTI	139 (27)	15 (31)	1 (10)	1 (17)	156 (27)
Wound infection	12 (2)	0 (0)	0 (0)	0 (0)	12 (2)
Pulmonary embolism	7 (1)	0 (0)	0 (0)	0 (0)	7 (1)
Deep venous thrombosis	8 (2)	0 (0)	0 (0)	0 (0)	8 (1)
Postop myocardial infarction	6 (1)	1 (2)	2 (20)	0 (0)	9 (2)
Postop hematoma	14 (3)	1 (2)	1 (10)	0 (0)	16 (3)
EVD-related hematoma	10 (2)	2 (4)	0 (0)	0 (0)	12 (2)
Hydrocephalus requiring shunt	77 (15)	9 (18)	3 (30)	0 (0)	89 (15)
Delayed vasospasm	103 (20)	6 (12)	0 (0)	2 (33)	111 (19)
Death w/in 1 mo	49 (9)	7 (14)	2 (20)	3 (50)	61 (10)

UTI = urinary tract infection.

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- **42yo WM**
- **CC:** "my eyelid feels droopy"
- **Associated symptoms:** headache and left side orbital pain; denies h/o trauma
- **VAcc:** 20/20 OD, OS
- **EOM:** FROM OD, OS
- **CVF:** FTFC OD, OS
- **IOP:** 17/18
- **Slit lamp exam and DFE:** unremarkable

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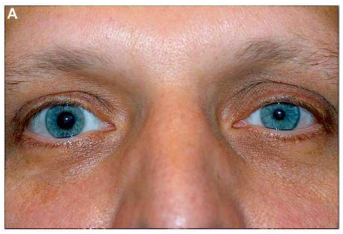

Kanagalingam & Mille[redacted]nical perspectives

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Using only this information, would you send this patient to the ER?

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Horner Syndrome

- Ptosis, miosis, anhidrosis
- Disruption in sympathetic nerve supply
  - Usually acquired
- Many systemic associations
  - Neck procedures, carotid dissection, cluster headache, CVA, tumor, and many others
  - In multiple studies, up to 78% of patients had a "dangerous cause"
- Incidence: ~4.24/100,000/year

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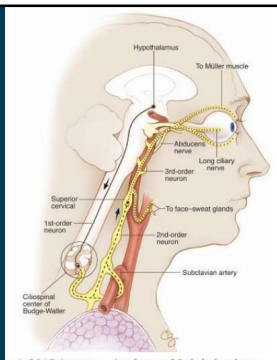

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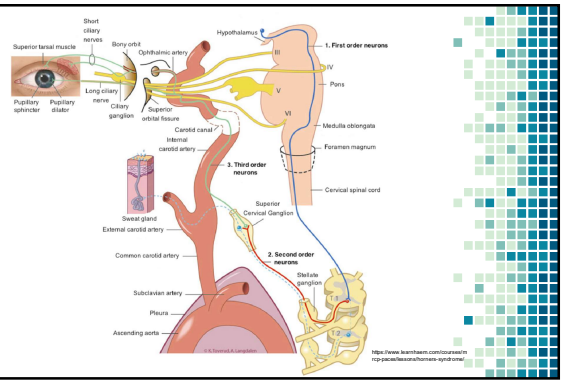

https://www.learnhaem.com/courses/mrcp-paces/lessons/horners-syndrome/

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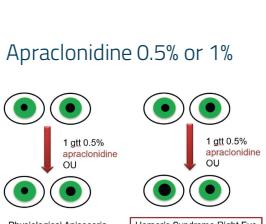
Testing for Horner Syndrome

- Apraclonidine 0.5% or 1%



Physiological Anisocoria (NEGATIVE TEST)

Horner's Syndrome Right Eye (POSITIVE TEST)

https://www.reviewofoptometry.com/article/follow-this-practical-workup-for-acquired-ptosis

https://webeye.ophth.uiowa.edu/eyeforum/atlas/pages/apraclonidine-test-for-horners/index.html#gsc.tab=0

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Table 2 Possible etiologies of with pediatric and adult Horner syndrome

Primary cause of Horner syndrome	Pediatric subjects (N = 139)			Adult subjects (N = 1331)		
	0–4 (N = 99)	5–9 (N = 24)	10–14 (N = 16)	15–39 (N = 327)	40–59 (N = 676)	60– (N = 328)
Idiopathic or undetermined	80 (81%)	19 (79%)	10 (63%)	151 (46%)	258 (38%)	127 (39%)
Diseases or trauma						
Neuroblastic tumors	7	3	2	0	0	0
Peripheral nerve tumors	0	0	0	32 (9.8%)	30 (4.4%)	5
Other tumors						
Head	0	0	0	1	3	1
Cervical	0	0	0	21 (6.4%)	51 (7.8%)	23 (3.3%)
Thoracic/mediastinal	0	0	0	1	13 (1.9%)	6
Carotid artery diseases	0	0	0	5	22	20
Cluster headache	0	0	0	4	6	1
Stroke	1	0	0	16	49	33
Multiple sclerosis	0	0	0	1	1	3
Meningitis	1	0	0	0	0	0
Trauma	2	0	0	2	2	0
Post procedure						
Surgery						
Head	0	0	0	0	3	0
Cervical	0	0	0	59 (18.0%)	130 (19.2%)	35 (10.7%)
Thoracic	3	0	1	9	14 (2.1%)	11 (3.4%)
Mediastinal	0	0	3	14 (4.3%)	29 (4.3%)	14 (4.6%)
Paraspinal	0	0	0	3	3	0
Sympathectomy	0	0	0	2	19 (2.8%)	13 (4.0%)
Carotid artery stent insertion	0	0	0	0	0	1
Chest tube insertion	3	0	0	7	8	3
Anesthesia	0	0	0	3	12 (1.8%)	9
Central venous catheter	2	2	0	5	23 (3.4%)	23 (7.0%)

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Table 3.

Causes of Horner Syndrome in Major Published Studies

Cause of Horner Syndrome	Our Study (Pharmacologically Confirmed) (n = 159)	Our Study (Pharmacologically Unconfirmed) (n = 159)	Giles et al[2] (1958) (n = 216)	Keane[3] (1979) (n = 100)	Maloney et al[4] (1980) (n = 450)	Almog et al[5] (2010) (n = 52)	Sadaka et al[6] (2017) (n = 132)
Undetermined	39%	9%	24%	8%	40%	17%	78%
Post Procedure	21%	36%	19%	5%	10%	24%	3%
Carotid Dissection	9%	11%	0%	0%	0%	7%	2%
Trauma	8%	8%	6%	6%	4%	8%	1%
Cluster Headache	8%	5%	0%	2%	12%	0%	8%
Tumor	7%	18%	36%	23%	13%	16%	5%
Stroke	4%	9%	5%	48%	3%	16%	1%
Miscellaneous	3%	0%	4%	7%	15%	8%	2%
Congenital	1%	4%	6%	0%	3%	2%	0%

Sabbagh MA, De Lott LB, Trobe JD. Causes of Horner Syndrome: A Study of 318 Patients

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Table 3 Distribution of underlying systemic diseases in subjects with Horner syndrome in entire South Korean population

Variables	Horner syndrome with related diseases (N = 362)		
	Before HS (N = 220)	Same as HS (N = 74)	After HS (N = 68)
Neuroblastic tumors	8	0	4
Peripheral nerve tumors	55 (25.0%)	7	5
Other tumors	91 (41.4%)	14 (18.9%)	15 (21.5%)
Carotid artery diseases	21	15 (20.3%)	11 (16.9%)
Cluster headache	3	4	4
Cerebrovascular accidents	40 (18.2%)	34 (45.9%)	25 (38.5%)
Multiple sclerosis	1	0	4
Miscellaneous	1	0	0

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Internal Carotid Artery Dissection (ICAD)

- Intimal tear of the vasa vasorum
- Traumatic or spontaneous cause
- MRI/MRA or CTA is most often utilized for diagnosis
- Major cause of stroke in patients <50yo
- Incidence :2.5-3/100,000

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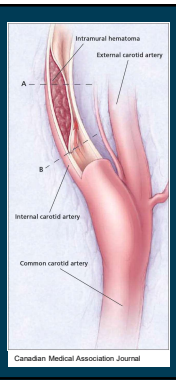
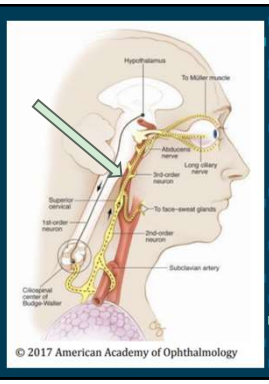


Canadian Medical Association Journal



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ICAD and Pain

- Pain is very common, reported range of 60-95%
- Generally, ipsilateral head or face
  - Headache: 91% ipsilateral
  - Ear pain: 48% of cases
  - Orbital pain: 61% of cases
- Neck pain: 26% of cases
- Painful Horner Syndrome: 58% of cases
  - In 10% of patients, this is the only clinical manifestation
- Gradual onset

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Risks of Carotid Artery Dissection

- High risk of associated **stroke** within first two weeks
- ICAD accounts for approximately 20% of strokes in patients under the age of 50
  - >50-70% of patients over multiple studies
  - Usually embolic
- Overall prognosis is good

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Stroke Risk at Diagnosis

- Rochester Epidemiology Project: **40% of patients** with ICAD had stroke
  - 68% if including stroke + TIA
- Dijon Stroke Registry: **63.6% of patients** with ICAD had a stroke
  - 100% if including stroke + TIA
  - Higher severity in those with ICAD
- Good news!
  - Dijon Stroke registry**: 90% good outcome**, 0 died
  - Rochester Epidemiology Project: **91% good outcome**, 9% poor outcome or death

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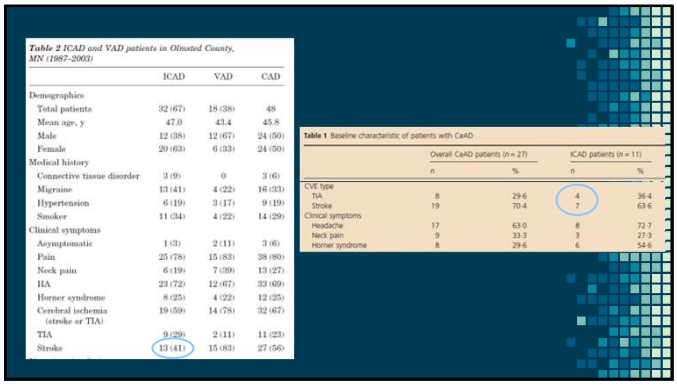

Table 2 ICAD and VAD patients in Olmsted County, MN (1987–2003)

	ICAD	VAD	CAD
Demographics			
Total patients	32 (67)	18 (38)	48
Mean age, y	47.0	43.4	45.8
Male	12 (38)	12 (67)	24 (50)
Female	20 (63)	6 (33)	24 (50)
Medical history			
Connective tissue disorder	3 (9)	0	3 (6)
Migraine	13 (41)	4 (22)	16 (33)
Hypertension	6 (19)	3 (17)	9 (19)
Smoker	11 (34)	4 (22)	14 (29)
Clinical symptoms			
Asymptomatic	1 (3)	2 (11)	3 (6)
Pain	25 (78)	15 (83)	38 (80)
Neck pain	6 (19)	7 (39)	13 (27)
HA	23 (72)	12 (67)	33 (69)
Horner syndrome	8 (25)	4 (22)	12 (25)
Cerebral ischemia (stroke or TIA)	19 (59)	14 (78)	32 (67)
TIA	9 (29)	2 (11)	11 (23)
Stroke	13 (41)	15 (83)	27 (56)

Table 1 Baseline characteristic of patients with CeAD

	Overall CeAD patients (n = 27)		ICAD patients (n = 11)	
	n	%	n	%
CVE type				
TIA	8	29·6	4	36·4
Stroke	19	70·4	7	63·6
Clinical symptoms				
Headache	17	63·0	8	72·7
Neck pain	9	33·3	3	27·3
Horner syndrome	8	29·6	6	54·6

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Stroke Risk Following Diagnosis

- If no ischemia found at diagnosis:
  - Retrospective study of 2791 patients with cervical artery dissection
  - 47 patients (1.68%) developed a stroke within 12 weeks following dissection
    - All events occurred in the first two weeks

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ICAD Treatment

- Generally self-healing
  - Mean healing time: 4 months
- Treatment is aimed at preventing stroke or recurrent stroke
  - Antiplatelet or anticoagulation both acceptable therapies
- Surgical intervention (stenting, reconstruction) is rarely utilized

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So…when should I send a pt with Horner Syndrome to the ER?

- Horner syndrome + pain (inspilateral orbital, headache, neck, or ear pain) = consider ER referral
- Especially if pt reports recent history of trauma or neck injury

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Questions?

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