Intracranial Hypertension

Emergency Department

Nationwide Children's — When your child needs a hospital, everything matters.

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Exclusion Criteria
- Trauma
- Altered mental status

[Definition and Diagnosis]

[Signs & Symptoms of IH]

Patient with concern for optic nerve edema

[Known IH Algorithm]

Acute neurologic deficits on exam?

Yes → **Off Pathway**
See ED Sudden Neurologic Deterioration Pathway

No

Obtain visual acuity
Consult Ophthalmology

Confirmed optic nerve edema?

No → **Off Pathway**
Consider alternative diagnoses

Yes

Obtain [Neuroimaging]
(MR preferred)

Critical neuroimaging findings*?

Yes → **Treat condition according to standard of care.**
Consult appropriate subspecialists (Neurosurgery, Neurology, Oncology, etc.)

**Critical neuroimaging findings*:*
- Hydrocephalus
- Stroke
- Intracranial mass
- Vascular lesion
- Chiari malformation
- Abnormal enhancement

No

[Risk Assessment] (per Ophthalmology):

Fulminant | **High Risk** | **Low Risk**

[Lumbar Puncture]**
- Use Non-peds tray with 550 mm manometer
- CSF studies
- Obtain blood glucose 30 min before or after LP

Proper LP positioning for opening pressure measurement is left lateral decubitus. **Legs need to be fully extended at time of measurement.

If LP is unsuccessful, patient should be admitted.
For fulminant and high risk patients, LP is needed emergently. Call IR (722-2420) and place order for IR consult.

Admit with Neurology and Ophthalmology Consults

- Consult Neurology
- Discuss need for LP
- Initiate [medical management]
  - Acetazolamide
  - Furosemide

[Discharge] when meeting criteria

Known Intracranial Hypertension

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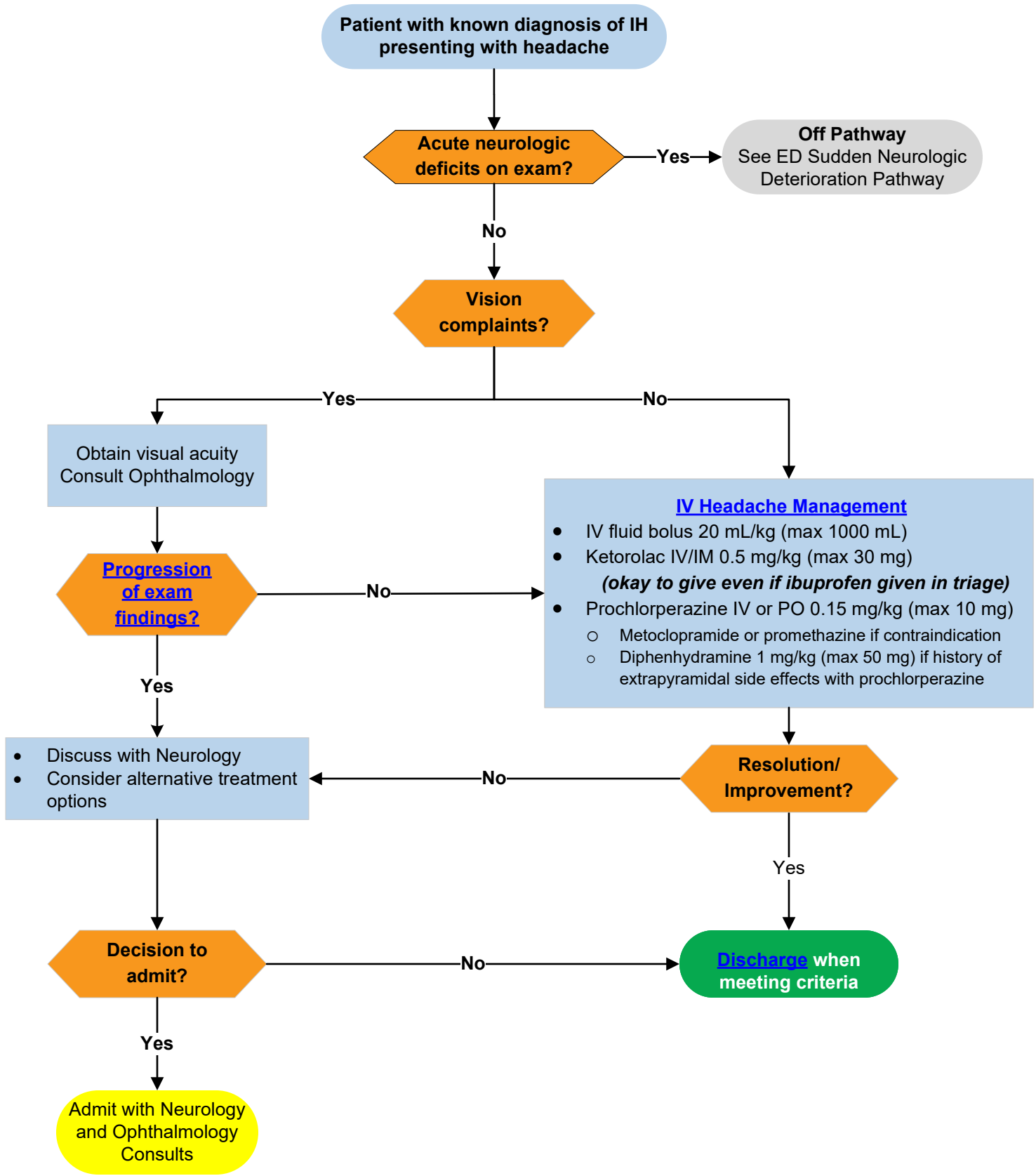

Definition & Diagnosis

Intracranial hypertension (IH) occurs when there is elevated intracranial pressure (ICP). Primary (aka idiopathic) IH refers to cases in which there is normal cerebrospinal fluid (CSF) composition and no identifiable cause of IH. Secondary IH is characterized by increased ICP due to identifiable causes.

If not recognized and managed appropriately, IH can lead to severe visual dysfunction, including permanent vision loss.

Classification of intracranial hypertension:

Primary	Secondary
Risk factors may include: Female gender Post-pubertal patients Obesity (especially recent weight gain) Polycystic ovarian syndrome Iron deficiency anemia	Increased pressure resulting from: Sinus venous thrombosis Chiari malformation Meningitis Refeeding syndrome Tetracycline/minocycline Growth hormone Corticosteroids (withdrawal) Vitamin A Accutane

Diagnostic Criteria:

Prepubertal Primary IH Criteria:

1) CSF opening pressure of >18 cmH_2O in children < 8 years or >25 cmH_2O in children ≥ 8 years or < 8 years without optic edema.
2) No focal neurological signs (with the exception of cranial nerve IV or VI palsy).
3) Normal CSF composition.
4) Exclusion of recognized secondary causes.
5) Bilateral optic disc edema.
6) Symptoms suggestive of elevated ICP (headache, nausea, vomiting, transient visual obscurations, tinnitus) that improve following CSF drainage.

Definite diagnosis: Patient meets criteria 1-6.
Probable diagnosis: Patient meets criteria 1-4 and either 5 or 6.

Adapted from Aylward SC. Pediatric Idiopathic Intracranial Hypertension: A Need for Clarification. Pediatric Neurology. 2013;49(5): 303-304

Modified Dandy Criteria (for adolescents):

1) Signs and symptoms of increased ICP (headache, nausea, vomiting, transient obscurations of vision, papilledema).
2) Absence of localizing neurologic signs (unilateral or bilateral abducens nerve palsies excluded).
3) CSF opening pressure ≥ 25 cmH_2O with normal composition.
4) Normal to small symmetrical ventricles as demonstrated by neuroimaging.

Adapted from Smith JL. Whence Pseudotumor Cerebri? J Clin Neuroophthalmol. 1985;5:55-56

Return to Algorithm

Signs & Symptoms of IH

- Optic nerve edema (unilateral or bilateral)
- Cranial nerve palsy (Abducens [CN VI] is the most common)
- Headache (worsening frequency or continuous)
  - Headache worse when laying down
  - Headache can have migrainous features including nausea, vomiting
- Visual symptoms
  - Transient visual obscurations (instant blacking out)
  - Diplopia
  - Vision loss
- Pulsatile tinnitus (whooshing, hearing heartbeat)

Return to Algorithm

Differential Diagnoses

- Stroke (ischemic or hemorrhagic)
- Intracranial mass lesions (e.g. tumor, abscess)
- Obstructive hydrocephalus
- Vascular malformation
- Venous sinus thrombosis
- Chiari Malformation
- Migraine with or without aura
- Tension headache
- Meningitis/encephalitis
- Trauma/concussion
- Optic neuritis
- Pseudopapilledema

Return to Algorithm

Neuroimaging

Imaging Modality:

- MR Head Fast Imaging (Non-SND) **AND** MRV (MR Head Angiography without contrast) if able and patient can tolerate
- If unable to obtain MR, obtain CT Head Venogram without and with contrast

Findings concerning for IH:

- Empty sella turcica
- Distension of the optic nerve sheaths
- Increased tortuosity of optic nerve
- Posterior globe/scleral flattening
- Optic nerve head protrusion
- Transverse cerebral venous sinus stenosis

Critical Neuroimaging Findings/ Secondary Causes of IH:

- Hydrocephalus
- Stroke
- Intracranial mass
- Vascular lesion
- Chiari malformation
- Abnormal enhancement

Return to Algorithm

Lumbar Puncture

Use Non-Peds LP Tray with 550 mm manometer

Proper LP positioning for opening pressure measurement is left lateral decubitus.
Legs need to be fully extended at time of measurement

Lower pressure no more than 10 cmH2O and do not remove more than 10 mL unless directed by neurology. Removing too much CSF may cause post-LP headache.

Laboratory Testing

- Use ED IP Lumber Puncture Order Set
- CSF studies: cell counts, glucose, and protein
- Blood glucose 30 min before or after LP

If LP is unsuccessful, patient should be admitted.

For fulminant and high risk patients, LP is needed emergently. Call IR (722-2420) and place order for IR consult.

NCH Lumbar Puncture Policy (Lumbar_Puncture_110-30.pdf)

Return to Algorithm

Risk Assessment

Risk Assessment (to be determined by Ophthalmology)

	Low Risk	High Risk	Fulminant
Papilledema grade	Grade 1-2 optic nerve edema	Grade 3 optic nerve edema	Grade 4-5 optic nerve edema
Vision	Normal visual acuity and visual fields	Early signs of visual field or acuity abnormalities	Definite visual field or acuity loss

Return to Algorithm

Medications

Headache Medications

Drug	Dosing	Mechanism of Action	Clinical Pearls
Prochlorperazine	PO or IV 0.15 mg/kg (max 10 mg) over 2-10 min	Dopamine receptor antagonists reduce pain and nausea. Additionally have antihistamine and anticholinergic effects.	• Typical agent of choice • May prolong QT interval, consider baseline ECG prior to administration
Diphenhydramine	PO or IV 1 mg/kg (max 50 mg) over 3-5 min	Antihistamine used to treat phenothiazine-related extra pyramidal side effects	• Used to treat phenothiazine-related extrapyramidal side effects, such as dystonia and akathisia
Ketorolac	IM or IV 0.5 mg/kg (max 30 mg) over 15-30 sec	Nonsteroidal anti-inflammatory drugs (NSAID) inhibit cyclooxygenase to reduce prostaglandins and thromboxanes synthesis.	• Pretreatment with IV fluids may protect kidneys in setting of dehydration • Onset of analgesic effect is delayed up to 30 minutes, peak effect is at 1 - 2 hours • Do not mix in the same syringe with prochlorperazine
Metoclopramide	IV 0.3 mg/kg (max 20 mg) over 2-20 min	Dopamine receptor antagonists reduce pain and nausea. Additionally has anticholinergic effects.	• May prolong QT interval, consider baseline ECG prior to administration
Promethazine	PO or PR or IV 0.25 mg/kg (max 25 mg) **Central line required for IV**	Dopamine receptor antagonists reduce pain and nausea. Additionally have antihistamine and anticholinergic effects.	• Reserved for those with reactions to metoclopramide or prochlorperazine • Rapid infusion may result in a drop in blood pressure • Serious tissue injury may result from extravasation – central line required

IH Medications

Drug	Dosing	Mechanism of Action	Clinical Pearls
Acetazolamide	PO 10mg/kg/day divided BID; increase to 20mg/kg/day divided BID in 1 week. (Max. dose 2000 mg/day.) **Adolescents**: start 250mg BID x1week, then 500mg BID x1week, then 1000mg BID.	Reversibly inhibits carbonic anhydrase to reduce hydrogen ion secretion at renal tubule and increased renal excretion of sodium, potassium, bicarbonate, and water. Decreases CSF production by lowering sodium transport across choroidal epithelium.	• Should be taken with food to decrease GI upset. • Use with caution in patients with history of hypersensitivity to sulfonamides.
Furosemide	PO 0.5mg/kg/day divided BID; increase to 1 mg/kg/day divided BID in 1 week. (Max. dose 40 mg/day) **Adolescents**: start 10mg BID x1week, then 20mg BID.	Inhibits renal reabsorption of sodium and chloride. Increases diuresis and reduce sodium transport into brain.	• Use with caution in patients with depleted intravascular volume and/or kidney disease. Loop diuretics may lead to acute kidney injury and diuresis, resulting in hypovolemia.
Topiramate	PO 1-2mg/kg/day divided BID; increase to 3-4mg/kg/day divided BID in 1 week. (Max. dose 200 mg/day). **Adolescents:** start 50mg BID x1week, then 100mg BID.	Decreases CSF production by lowering sodium transport across choroidal epithelium.	• Doses >200 mg/day can cause treatment failure of oral contraceptives. • Also used for migraine and associated with weight loss.

Return to Algorithm

Discharge Criteria

- Improved symptoms (pain, vision)
- No adverse effects from LP or sedation
- MRI/MRV in 48 hours (if not done with initial neuroimaging)
- Follow-up planning in place
- For patient with known IH, family to call neurologist in 1-2 days

Return to Algorithm

Helping Hand

Intracranial Hypertension Helping Hand

Return to Algorithm

References

1. Aylward SC. Pediatric idiopathic intracranial hypertension: a need for clarification. *Pediatr Neurol*. 2013;49(5):303-304. doi:10.1016/j.pediatrneurol.2013.05.019
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4. Mollan SP, Davies B, Silver NC, et al. Idiopathic intracranial hypertension: consensus guidelines on management. *J Neurol Neurosurg Psychiatry*. 2018;89(10):1088-1100. doi:10.1136/jnnp-2017-317440
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6. Ploof J, Aylward SC, Jordan CO, Drapeau AI. Case Series of Rapid Surgical Interventions in Fulminant Intracranial Hypertension. *J Child Neurol*. 2021;36(11):1047-1053. doi:10.1177/08830738211026798

Return to Algorithm

Metrics

Pathway Goal

- Standardize the evaluation and management of patients with suspected intracranial hypertension.
- Timely recognition and work-up of high risk and fulminant cases.

Quality Measures

Outcome Metrics

- ED length of stay
- Admission rate

Process Metrics

- Pathway Visualization

Balancing Metrics

- Return visit within 24 hours

Return to Algorithm

Pathway Team & Process

Pathway Development Team

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Advisory Committee Date: *March, 2026*

Origination Date: *March, 2026*

Next Revision Date: *March, 2026*

Clinical Pathway Development

This clinical pathway was developed using the process described in the NCH Clinical Pathway Development Manual Version 6, 2022. Clinical Pathways at Nationwide Children's Hospital (NCH) are standards which provide general guidance to clinicians. Patient choice, clinician judgment, and other relevant factors in diagnosing and treating patients remain central to the selection of diagnostic tests and therapy. The ordering provider assumes all risks associates with care decisions. NCH assumes no responsibility for any adverse consequences, errors, or omissions that may arise from the use or reliance on these guidelines. NCH's clinical pathways are reviewed periodically for consistency with new evidence; however, new developments may not be represented, and NCH makes no guarantees, representations, or warranties with respect to the information provided in this clinical pathway.

For more information about our pathways and program please contact:
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Return to Algorithm