Cefalea in P.S.: red flags e percorsi diagnostici.

Enrico Grassi

Responsabile Ambulatorio Cefalee

S.O.C. Neurologia - Ospedale di Prato

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Villa Capugi, Pistoia

Associazione Neurologia Emergenza Urgenza

J Headache Pain (2005) 6:287–289
DOI 10.1007/s10194-005-0210-1

Rosanna Cerbo
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Primary headache in Emergency Department: prevalence, clinical features and therapeutical approach

Headache is one of the most common reported complaints in the general adult population and it accounts for **between 1% and 3% of admissions** to an Emergency Department (**ED**).

It has been found to be the fifth most common reason for visiting the ED in the USA.

Italian data suggest that the **prevalence of headache visits in Italian EDs** is anywhere **from 0.6 to 1.2 %**.

Other Italian data (Cortelli et .al, Headache 2004; 44:587-595) suggest a prevalence **from 1.7 to 4.5%.**

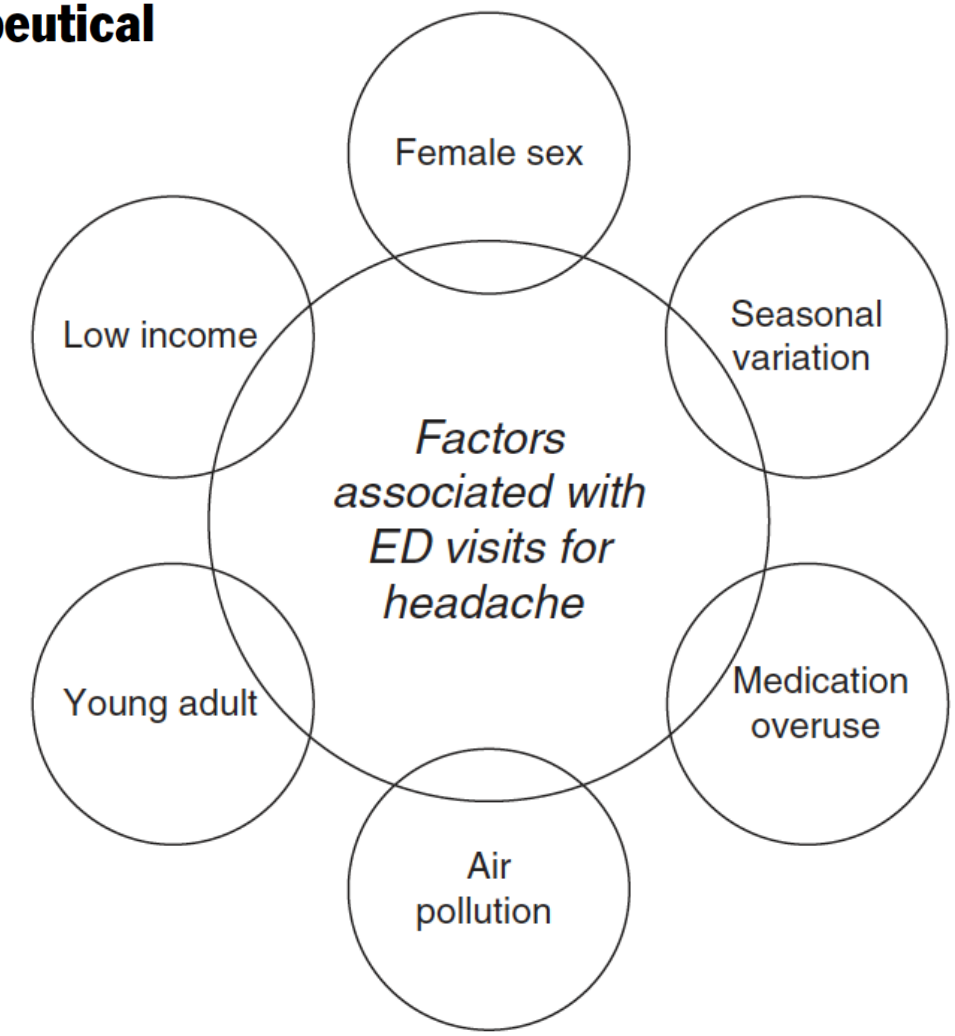

Figure 2.1 Factors associated with ED visits for headache.

Neurol Sci (2008) 29:67–75

The neurologist in the emergency department. An Italian nationwide epidemiological survey

Fabrizio Antonio de Falco · Roberto Sterzi · Vito Toso
Domenico Consoli · Donata Guidetti
Leandro Provinciali · Maurizio A. Leone · Ettore Beghi

In this study carried out in Italy, **headache was the second most common reason for neurological consultation in the ED**, representing **22 % of all ED consultations to neurology**.

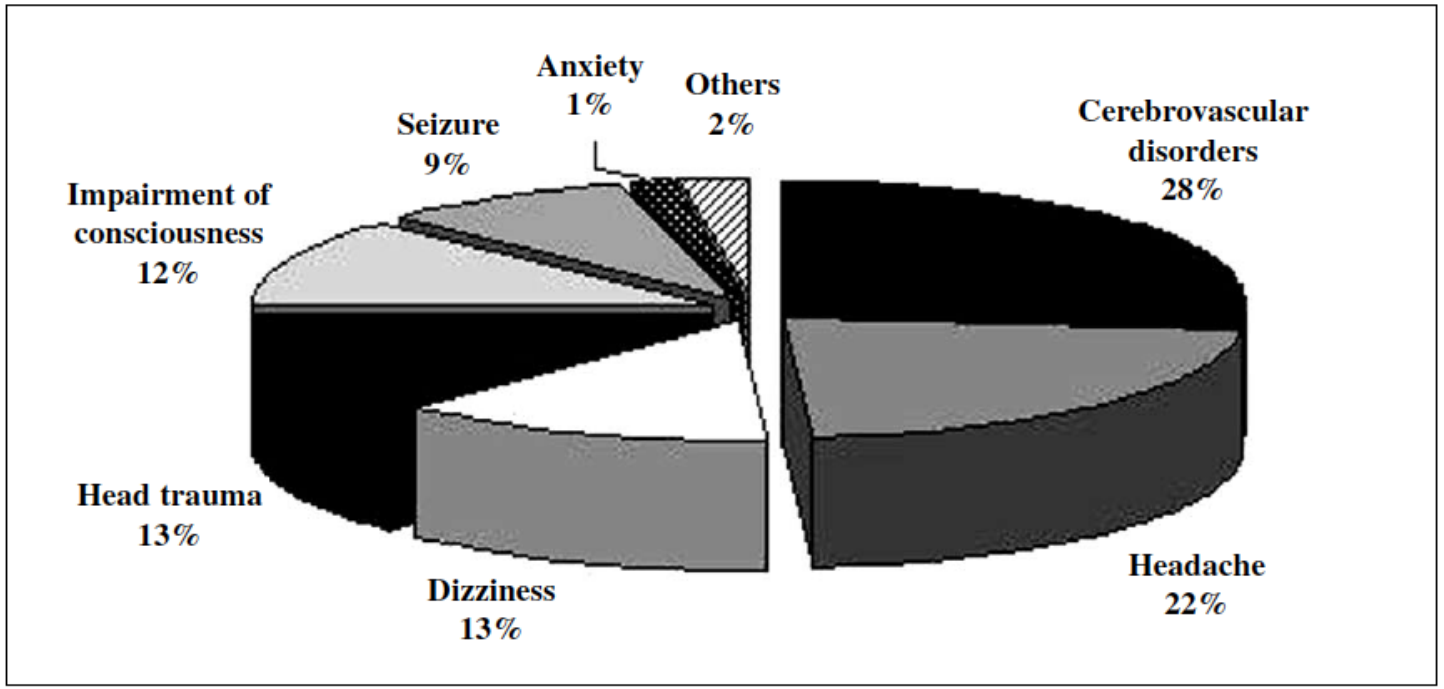

Fig. 2 Commonest clinical conditions requiring ER neurological consultation in 159 Italian neurological units

Headache
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Research Submission

Less Is Not More: Underutilization of Headache Medications in a University Hospital Emergency Department

Madhavi X. Gupta, MD; Stephen D. Silberstein, MD; William B. Young, MD;
Mary Hopkins, RN; Bernard L. Lopez, MD; Gregory P. Samsa, PhD

There is evidence that patients presenting to the ED with headache may be **undertreated**.

This US study found that just over **one-third** of patients visiting the ED for headache received neither an intravenous line nor **any medication**, and that **only 21.8 %** of the patients were **headache-free at discharge**.

Many patients leave without a discharge diagnosis, outpatient medications, or instructions.

ED physicians could help **identify the migraineurs** and channel them toward **appropriate outpatient treatment**.

Headache. 2002;42:747-753

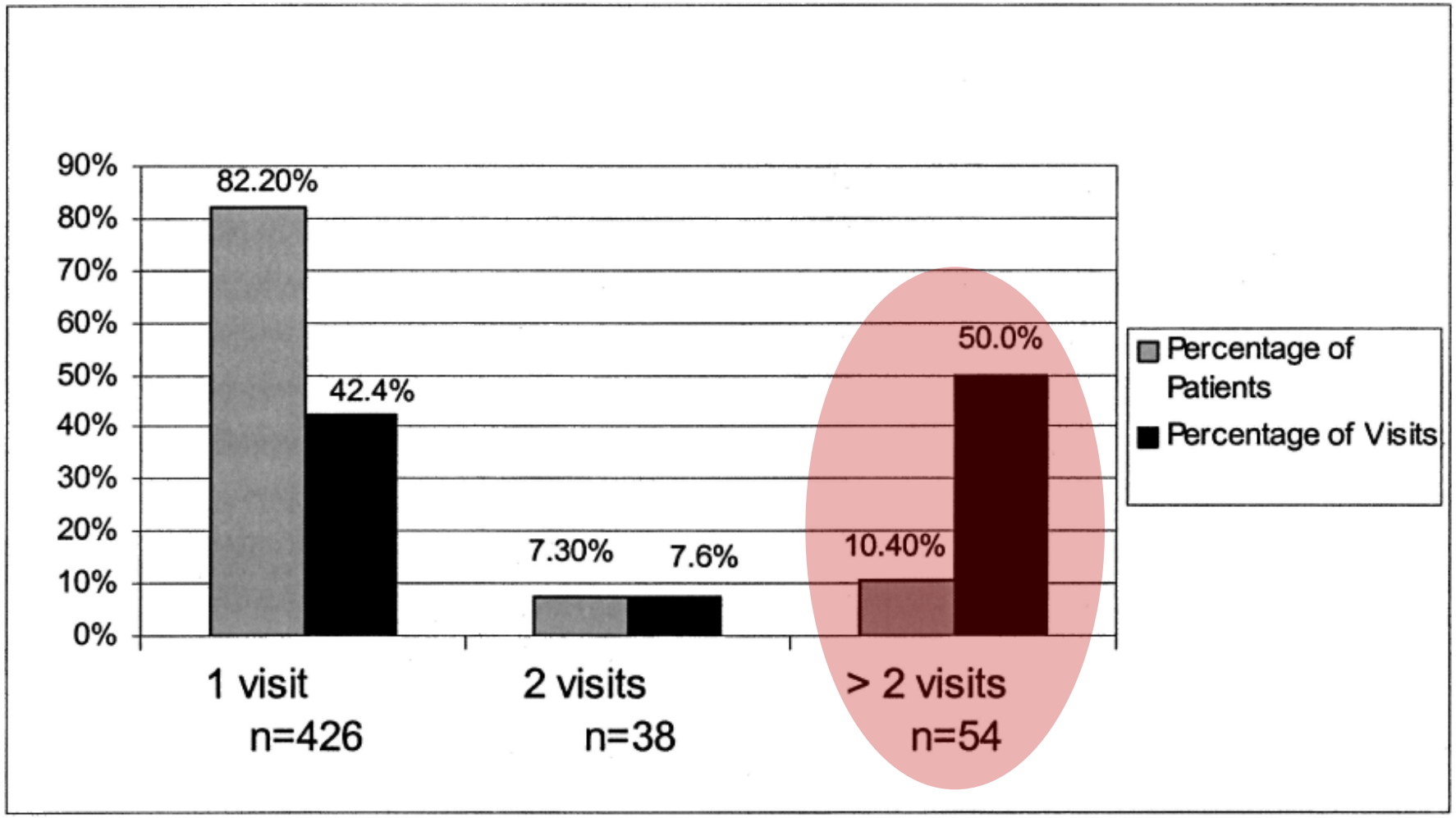
Health Resource Utilization of the Emergency Department
Headache "Repeater"

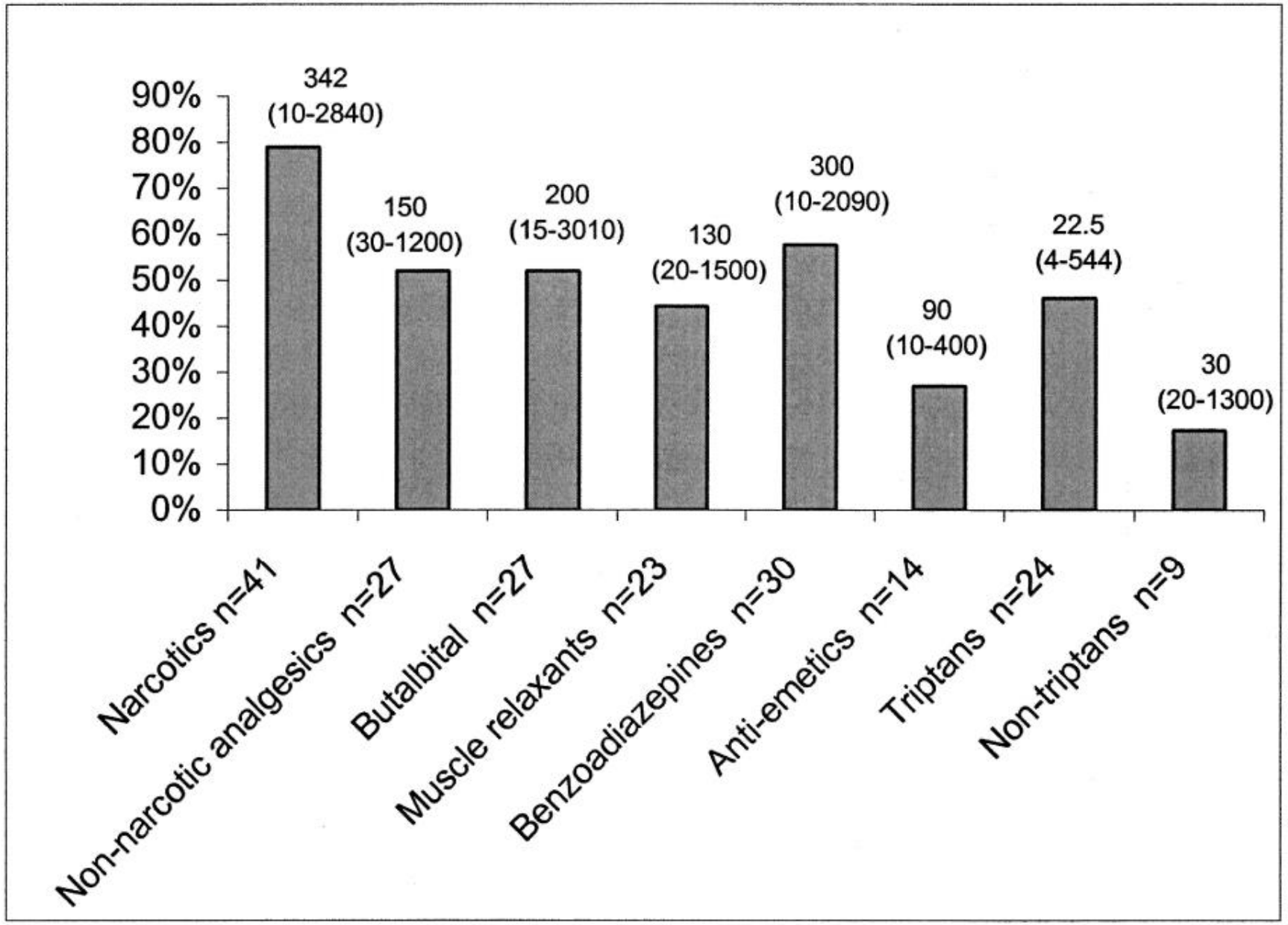
Morris Maizels, MD

There is a very expensive **cluster of patients**, accessing the **ED more than three times in a period of six months**.

In the US experience, these are **10% of the total population**, accounting for about **50% of headache-related visits to the ED**.

Using the same parameters, the percentage of **repeaters in Italian population**, was **11.8%**.





Prescription utilization during the previous 12 months for 52 "repeaters" of total number of doses for each medication category.

EPIDEMIOLOGY OF HEADACHE IN EMERGENCY DEPARTMENT

Whereas **primary headaches** are the most common cause of headache in the **adult ED**, secondary, non-lifethreatening causes, such as headaches associated with **viral illnesses**, underlie most headaches in the **pediatric ED**.

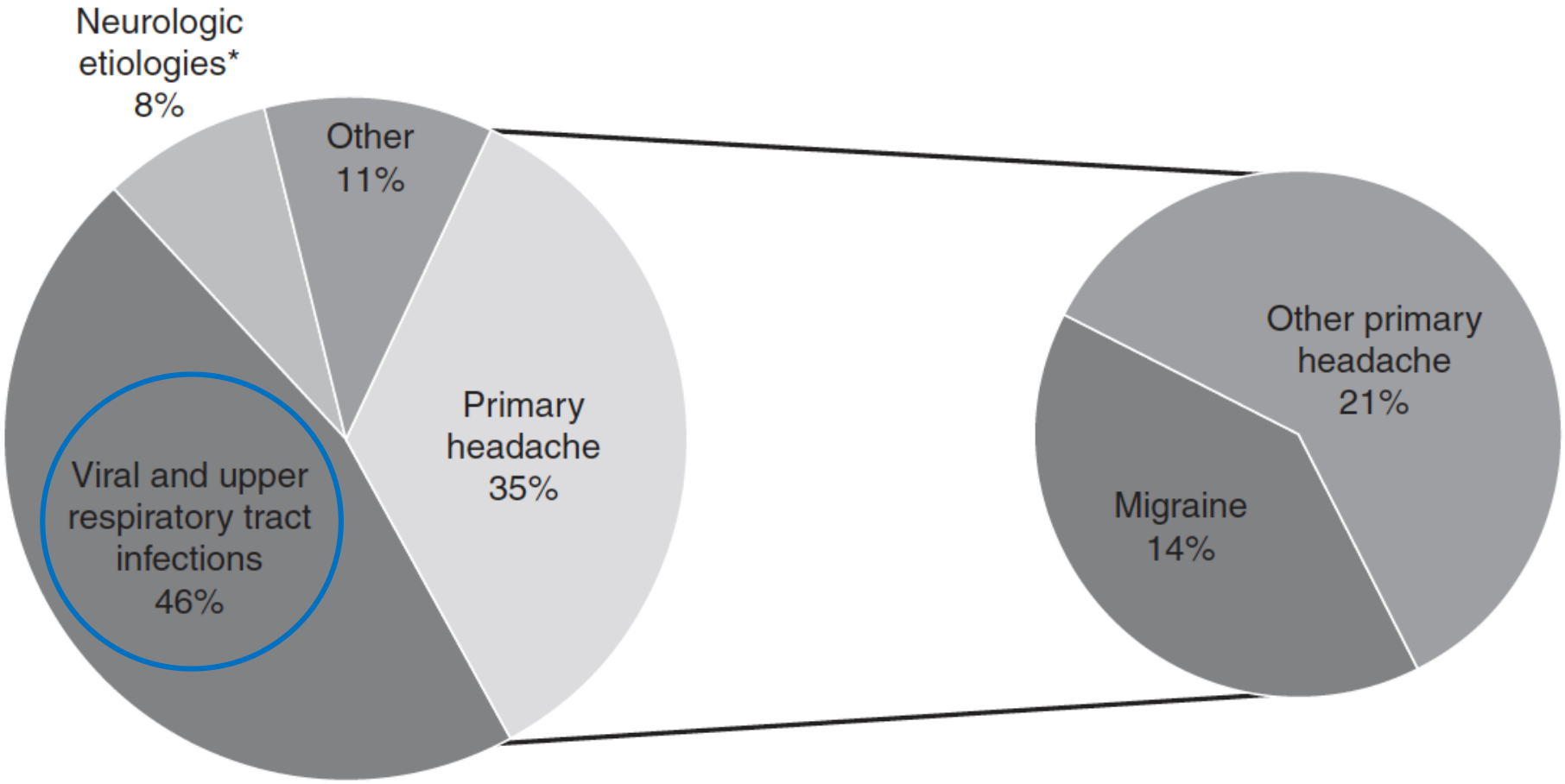


Figure 2.2 Etiology of headache in the pediatric ED.

* Neurologic etiologies include meningitis, brain tumor, post-traumatic headache, seizure, cerebrovascular diseases and ventriculoperitoneal shunt-related headaches.

** The percentages displayed were generated by pooling together results from the referenced studies [16–21].

Primary headaches are the most common cause of headache in the **adult ED patient.**

There is a tendency to label a large proportion of primary headache patients with a diagnosis of **"headache not otherwise specified."** Although it can be **challenging**, ED clinicians should always strive to **make a specific headache diagnosis**, as identifying the specific headache disorder is more likely to lead to more specific and optimal management and outcome.

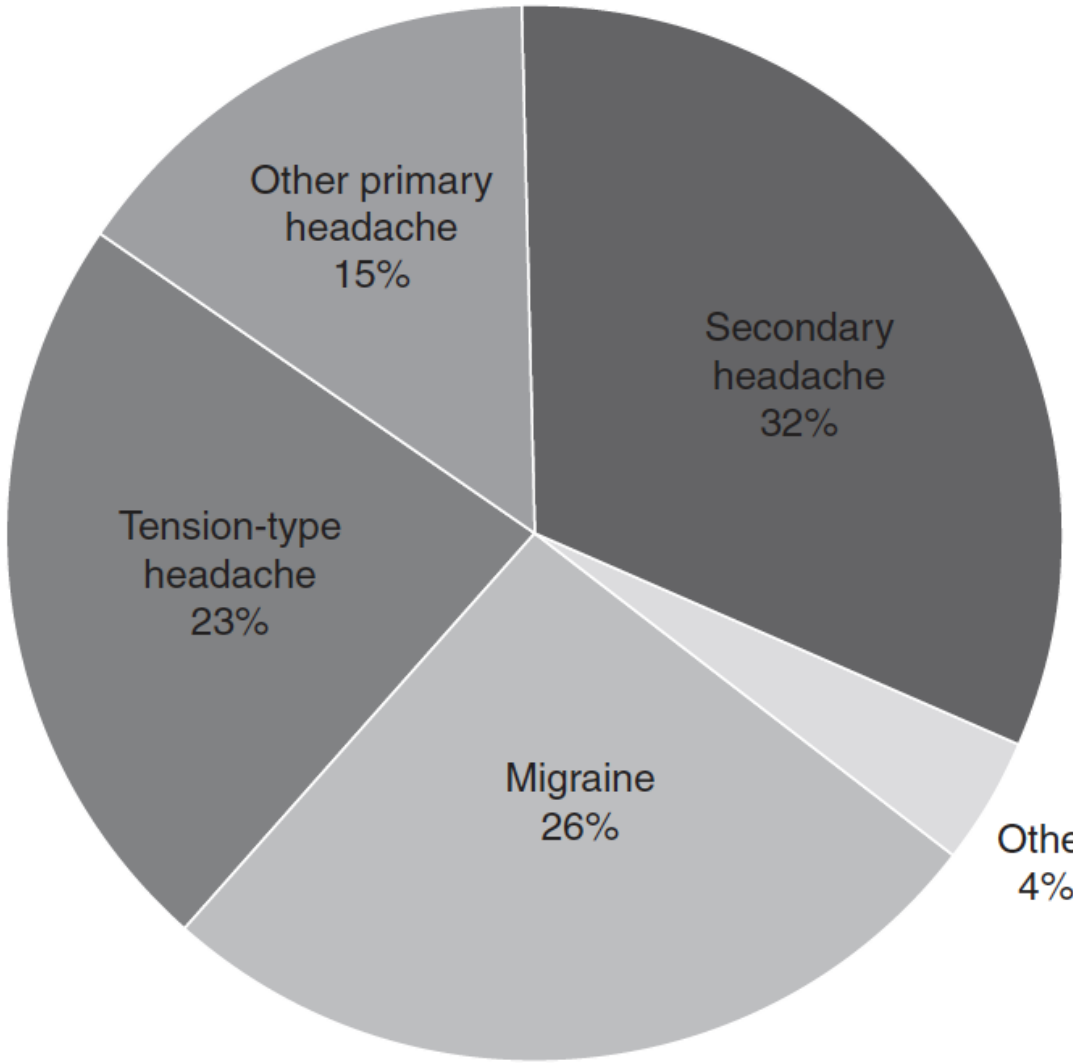


Figure 2.3 Etiology of headache in the adult ED.
* The percentages displayed were generated by pooling together results from the referenced studies [4,5,8,22–24].

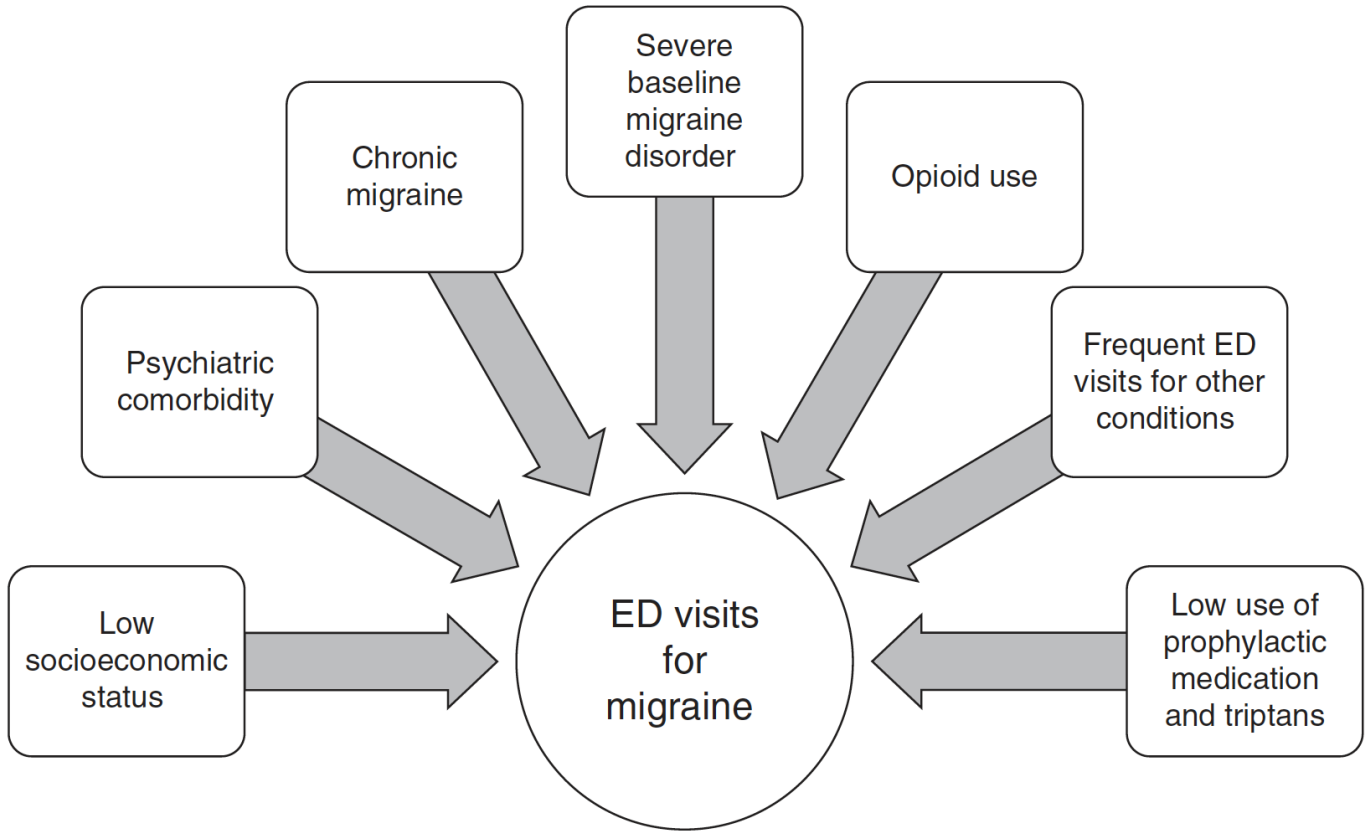


Figure 7.1 Factors associated with frequent ED visits for migraine.

Headache 2003;43:171-178

Migraine Preventive Medication Reduces Resource Utilization

Stephen D. Silberstein, MD; Paul K. Winner, DO; Joseph J. Chmiel, PhD

The **use of migraine prophylactic medications and triptans is inversely related to the frequency of ED visits for migraine**.

In this study, the addition of migraine prophylactic medications to a regimen of sumatriptan for acute migraine relief resulted in an **81.8 % decrease** in ED migraine visits.

Table 3.—Other Resource Utilization in Acute Plus Preventive Cohort*

Resource	Initial Preventive Medication		
	During 180 Days Before	0-179 Days After	180-359 Days After
Office and other outpatient visits	188	223	92
Emergency department visits	22	12	4
CT scans	20	12	5
MRIs	17	11	2
Other migraine medication dispensed, units	588	584	505

Emerg Med J (2011). doi:10.1136/emermed-2011-200088

Atraumatic headache in US emergency departments: recent trends in CT/MRI utilisation and factors associated with severe intracranial pathology

John W Gilbert,[1] Kevin M Johnson,[2] Gregory L Larkin,[3] Christopher L Moore[3]

This study, carried out in the United States, reported that the proportion of ED headache patients undergoing **neuroimaging increased from 12.5 % to 31% over a ten-year (1998-2008)** period, whereas the proportion of neuroimaging studies **uncovering pathology dropped from 10.1 % to 3.5 %** over the same period suggesting a role for clinical decision support to guide **more judicious use of imaging**.

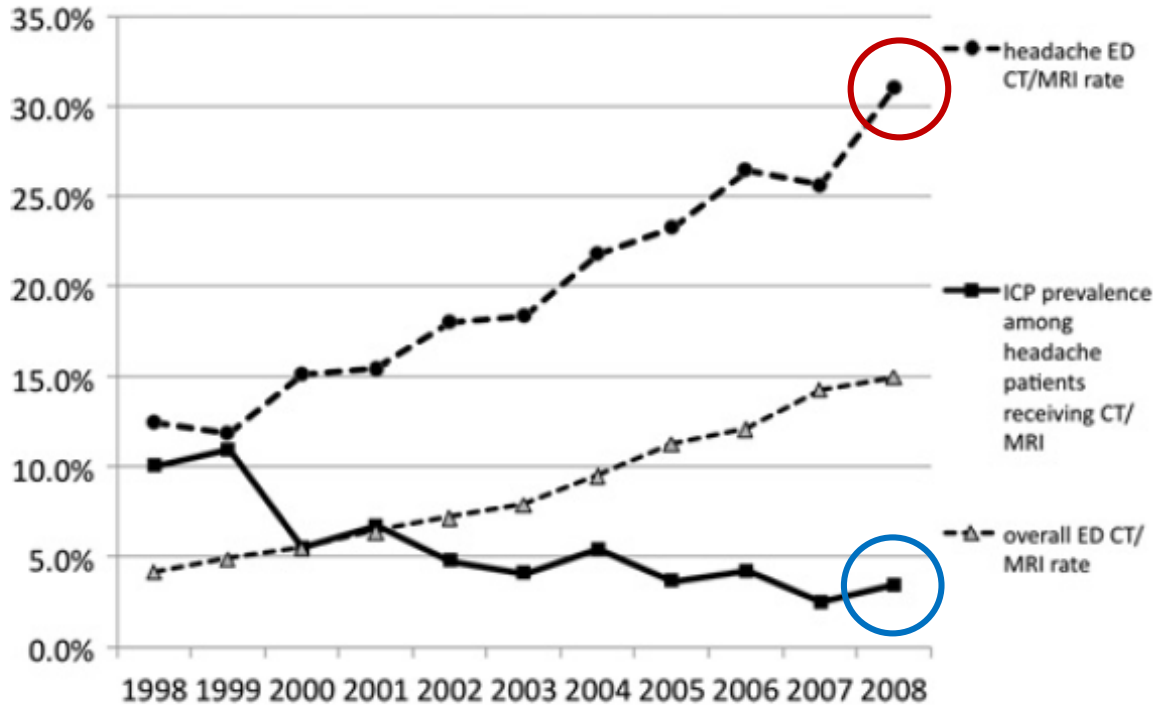


Figure 1 US trends in CT/MRI utilisation and yield of CT/MRI to diagnose severe intracranial pathology (ICP) in the emergency department (ED) evaluation of atraumatic headache, 1998–2008.

JOURNAL OF THE NATIONAL MEDICAL ASSOCIATION VOL. 101, NO. 4, APRIL 2009

Computed Tomography Imaging in the Management of Headache in the Emergency Department: Cost Efficacy and Policy Implications

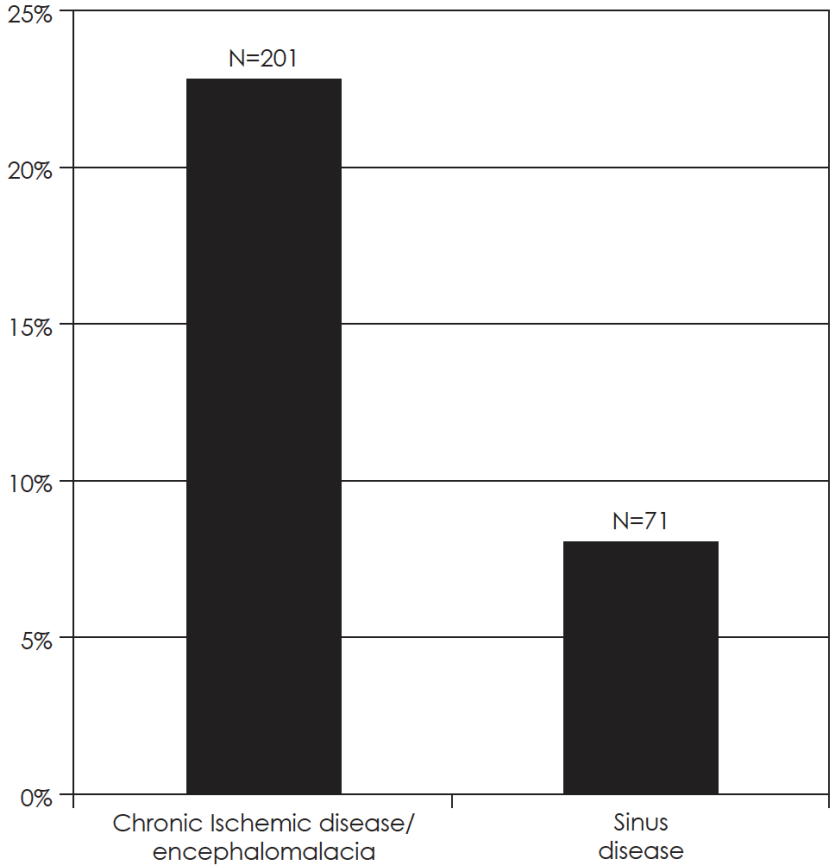
Yusef J. Jordan; Johnson B. Lightfoote, MD, MBA; John E. Jordan, MD, MPP

This US study examined the use of CT among **adult** patients presenting to the ED **with non-focal, atraumatic headaches without neurological symptoms**, immunocompromised status, nor prior history of neurosurgery.

In this group of patients, although **31.9 % had abnormal CT findings**, the vast majority were **incidental and did not alter management** (old infarcts, chronic ischemic changes, encephalomalacia, and sinusitis), with **only 1.02 %** resulting in **management changes**.

The **mean cost** of each **ED headache visit** was **$764** when accounting for the cost of CT.

The authors compared this with the cost of an outpatient visit for headache of similar presentation, and found that the **ED cost was triple the cost of an outpatient visit**.



The Commonality of Headache Recurrence

146 *J Emerg Crit Care Med. Vol. 19, No. 4, 2008*

Unplanned Emergency Department Revisits within 24 Hours in a Referral Hospital

Chiu-Lung Wu, Fa-Tsai Wang, Yao-Chiu Chiang, Yuan-Fa Chiu,
Teong-Giap Lin, Lian-Fong Fu, Tsung-Lung Tsai

The ***fourth most common cause of all unplanned revisits within 72 hours*** in a retrospective analysis from a secondary teaching referral hospital in Taiwan was headache, at 2.1 % (following abdominal pain [12.9 %], fever [12.6 %], and vertigo [4.5 %]).

Table 4 Most common chief complaints or diagnoses of 909 revisit patients

Chief complaints or diagnoses	No.	%
Abdominal pain	162	17.8
Fever	159	17.5
Vertigo	27	2.9
Headache	21	2.3
URI	21	2.3
Uremia	16	1.8
Short of breath	16	1.8
Angina	13	1.4
Flank pain	13	1.4
Allergy	10	1.1
Pneumonia	10	1.1
Asthma	8	0.9

PAIN MANAGEMENT/ORIGINAL RESEARCH

Recurrence of Primary Headache Disorders After Emergency Department Discharge: Frequency and Predictors of Poor Pain and Functional Outcomes

Benjamin W. Friedman, MD, MS From the Departments of Emergency Medicine and Neurology, Albert Einstein College of Medicine, Bronx, NY.

Ann Emerg Med. 2008;52:696-704.

Table 2. Outcomes of primary headache disorder patients after ED discharge, by headache type.*

Outcome	Migraine, n=186	Tension-type, n=34	Unclassifiable, n=77
24-h Outcomes			
Moderate or severe headache within 24 h of ED discharge, %†	31 (25-38)	19 (9-36)	27 (18-38)
Functional disability within 24 h of ED discharge, %‡	50 (43-57)	23 (12-41)	43 (32-54)
3-mo Outcomes			
3-mo MIDAS disability score >5 for 3 mo after ED discharge, %§	37 (30-44)	38 (23-54)	26 (17-37)
Median number of days with headache during 3 mo after ED discharge‖	4 (0-15)¶	1 (0-5)¶	2 (0-10)¶

In an ED-based prospective observational study of patients with primary headache disorders, ***headache recurrence*** was found to be common:
almost ***one-third of migraine*** patients,
one-fifth of tension-type headache patients, and
over ***one-quarter of other headache*** patients
had moderate or severe head pain within 24 hours of discharge from the ED.

Neurology® 2007;69:2038-2044

Randomized trial of IV dexamethasone for acute migraine in the emergency department

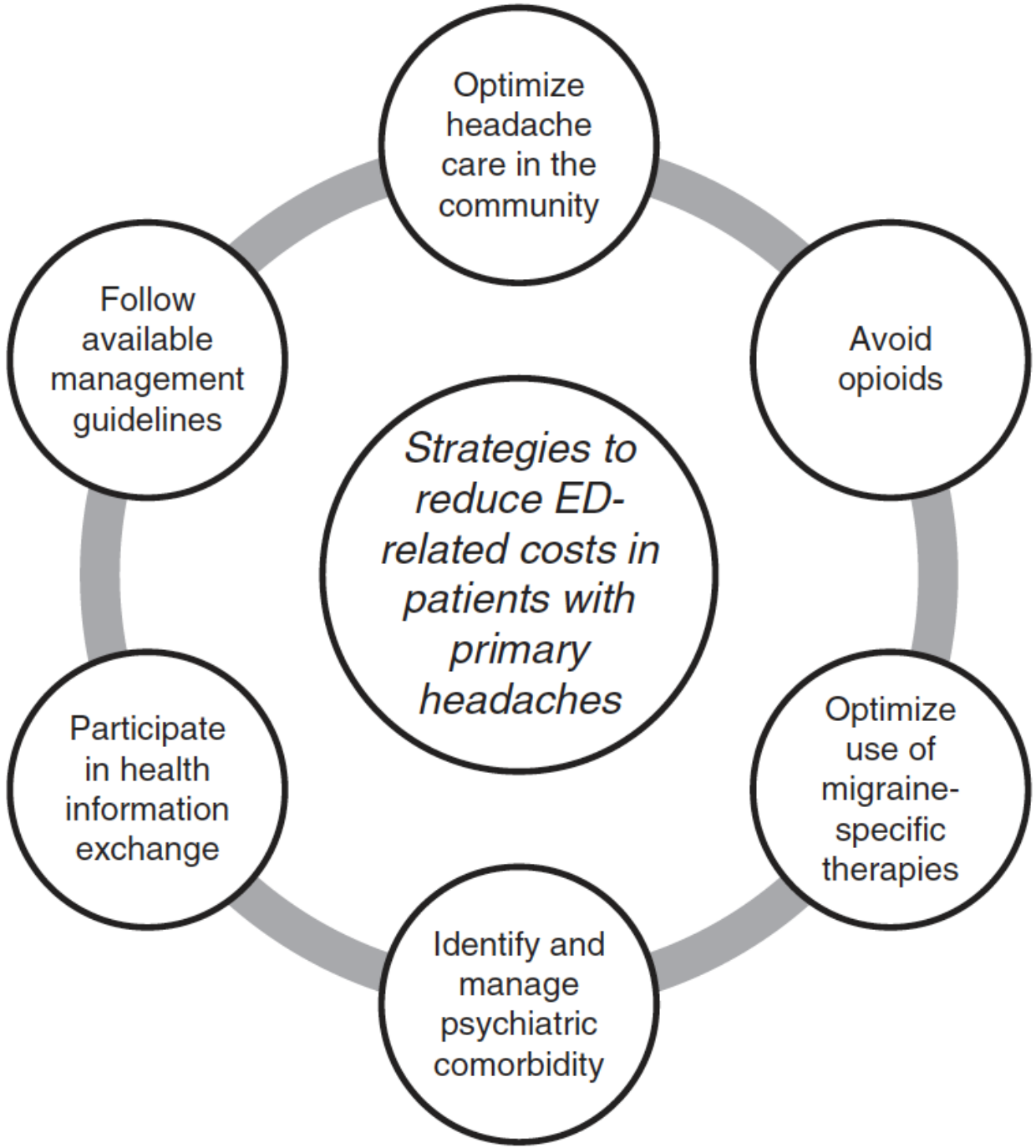
B.W. Friedman, MD, MS
P. Greenwald, MD
T.C. Bania, MD, MS

This trials assessing the efficacy of ***10 mg i.v. dexamethasone*** for **persistent pain freedom at 24 hours** found that the subgroup of patients presenting to the ED with prolonged migraine, defined as ***migraine lasting 72 hours or greater***, were more likely to achieve this outcome than those with shorter durations of migraine at presentation.

Table 4 Subgroup analysis: Pain intensity and functional outcomes by duration of headache

Headache duration >72 h	Dexamethasone (n = 22)	Placebo (n = 23)	OR (95% CI)
Persistent pain-free	38	13	4.1 (0.9–18)
No functional impairment after ED discharge	75	55	2.5 (0.7–9.3)
Subject satisfaction with medication	86	76	1.9 (0.4–9.1)
Pain free by the time of discharge	64	39	2.7 (0.8–9.1)
Headache duration ≤72 h	Dexamethasone (n = 83)	Placebo (n = 76)	
Persistent pain-free	22	21	1.0 (0.5–2.2)
No functional impairment after ED discharge	65	60	1.2 (0.6–2.4)
Subject satisfaction with medication	73	79	0.7 (0.3–1.5)
Pain free by the time of discharge	52	49	1.1 (0.6–2.1)

Values are percentages.


Optimize headache care in the community
Avoid opioids
Optimize use of migraine-specific therapies
Identify and manage psychiatric comorbidity
Participate in health information exchange
Follow available management guidelines
Strategies to reduce ED-related costs in patients with primary headaches

Neurologic causes account for **5–15 %** of headache cases, with a higher percentage (27.9%) resulting when post-traumatic headaches are included in this category. Approximately **2.1– 13 % of adult** patients presenting to the ED with headache have a **serious underlying secondary etiology**, such as meningitis, intracranial hemorrhage, or elevated intracranial pressure.

Table 2.1 Table of studies assessing headache etiology in the adult ED

Parameter	Dermitzakis et al., 2010 [4]	Bigal et al., 2000 [8]	Fodden et al., 1989 [5]	Leicht, 1980 [22]	Locker, 2004 [23]	Luda et al., 1995 [24]
Location of ED	Greece	Brazil	England	United States	England	Italy
Number of patients	851	561	130	485	353	215
All primary headaches (%)	77.9	55.6	32	54.5	79.3	56
Migraine (%)	15.4	45.1	19	22.3	30	28.8
Tension-type headache (%)	33.6	7.3	13	32.2	14.7	24.6
Cluster headache (%)	1.2	–	–	–	1.7	2.8
Primary headache not specified (%)	27.7	–	26	–	32.9	–
All secondary headaches (%)	22.1	44.3	31.5	38.1	18.4	43.7
Secondary systemic causes (%)	–	39.4	26	32.6	13	15.8
Secondary neurologic causes (%)	–	5	15	5.5	5.4	27.9*
Severe underlying secondary causes (%)**	–	2.1	13	4.5	5.4	8.4
Other (%)	–	–	–	7.4	2.3	–

* Includes post-traumatic headaches.
** Comprised a variety of different disease processes in each study, including but not limited to: intracerebral hemorrhage, subarachnoid hemorrhage, brain tumors, brain abscesses, and meningitis.

Approach to History Taking and the Physical Examination

Headache is a common reason for patients to present to the ED.
Most will have a non-life-threatening cause for their headache and require therapeutic intervention to reduce their pain, and words of assurance to relieve their concerns.

A **careful history** and **focused physical examination** is essential to identify those few patients who may have more serious and potentially fatal underlying causes for headache.

Time spent up-front obtaining a meticulous headache history will ultimately save time and money by allowing the physician to avoid unnecessary testing.

Why the patient has presented to the ED ?

- Is it because he/ she can **no longer tolerate their usual headaches** or is this a **new headache ?**
- If it is a new headache, is it a **first severe headache** or is it **sufficiently different from the usual headache ?**

1. the age of the patient;
2. location of the pain;
3. quality of the pain (throbbing, pressure, severity);
4. onset of the pain (gradual vs. sudden);
5. associated symptoms (nausea, vomiting, photophobia, phonophobia, neck pain, fever);
6. associated neurological symptoms (visual change, numbness, tingling, focal paresis, vertigo, speech change, gait change, personality change, cognitive change, fluctuating level of consciousness or loss of consciousness);
7. current treatment;
8. previous treatment;
9. trigger (exercise, cough, strain, sex);
10. other present and past medical conditions;
11. family history;
12. social history including habits such as caffeine intake, alcohol, illicit drugs, and smoking;
13. review of systems (weight loss, night sweats, chills, myalgias, cough, shortness of breath, abdominal pain, change in bowel movements, urinary symptoms).

Questions

The first two questions to ask the patient are the following:

1. **How quickly did this headache peak in intensity?** (Thunderclap – maximal intensity within 60 seconds).

2. **Have you ever had this same type of headache before, and if so, when did they begin to occur?**
   (An unusual headache or an attack different from their known headache pattern.)

- If the patient is able to say that he/she has already suffered from **several similar headaches** for months or years, a **primary headache** disorder is the most likely cause.

- If the patient may deny a previous headache history and report having **headaches for the first time** in his/her life for some hours, days, weeks, or months. In such cases, **a secondary headache has to be excluded**.

- If the patient reports a **history** of definite **primary headaches but states that his/her acute headache is different** from his/her usual headaches attacks. In such cases, **a secondary headache has to be suspected** and investigations are noteworthy.
  This is especially true if the headache began and peaked **suddenly** (less than 60 seconds – i.e., thunderclap headache) .

VIEWS & REVIEWS

Red and orange flags for secondary headaches in clinical practice

SNNOOP10 list

Thien Phu Do, MD, Angelique Remmers, MD, Henrik Winther Schytz, MD, PhD, DMSc, Christoph Schankin, MD, Sarah E. Nelson, MD, Mark Obermann, MD, Jakob Møller Hansen, MD, PhD, Alexandra J. Sinclair, MD, PhD, Andreas R. Gantenbein, MD, and Guus G. Schoonman, MD, PhD

Neurology® 2019;92:134-144.

Identifying any red flags in the clinical assessment of an ED patient with headache necessitates further diagnostic investigation to guide therapeutic interventions and optimize outcomes.

Table 1 SNNOOP10 list of red and orange flags

	Sign or symptom	Related secondary headaches (most relevant ICHD-3b categories)	Flag color
1	Systemic symptoms including fever	Headache attributed to infection or nonvascular intracranial disorders, carcinoid or pheochromocytoma	Red (orange for isolated fever)
2	Neoplasm in history	Neoplasms of the brain; metastasis	Red
3	Neurologic deficit or dysfunction (including decreased consciousness)	Headaches attributed to vascular, nonvascular intracranial disorders; brain abscess and other infections	Red
4	Onset of headache is sudden or abrupt	Subarachnoid hemorrhage and other headaches attributed to cranial or cervical vascular disorders	Red
5	Older age (after 50 years)	Giant cell arteritis and other headache attributed to cranial or cervical vascular disorders; neoplasms and other nonvascular intracranial disorders	Red
6	Pattern change or recent onset of headache	Neoplasms, headaches attributed to vascular, nonvascular intracranial disorders	Red
7	Positional headache	Intracranial hypertension or hypotension	Red
8	Precipitated by sneezing, coughing, or exercise	Posterior fossa malformations; Chiari malformation	Red
9	Papilledema	Neoplasms and other nonvascular intracranial disorders; intracranial hypertension	Red
10	Progressive headache and atypical presentations	Neoplasms and other nonvascular intracranial disorders	Red
11	Pregnancy or puerperium	Headaches attributed to cranial or cervical vascular disorders; postdural puncture headache; hypertension-related disorders (e.g., preeclampsia); cerebral sinus thrombosis; hypothyroidism; anemia; diabetes	Red
12	Painful eye with autonomic features	Pathology in posterior fossa, pituitary region, or cavernous sinus; Tolosa-Hunt syndrome; ophthalmic causes	Red
13	Posttraumatic onset of headache	Acute and chronic posttraumatic headache; subdural hematoma and other headache attributed to vascular disorders	Red
14	Pathology of the immune system such as HIV	Opportunistic infections	Red
15	Painkiller overuse or new drug at onset of headache	Medication overuse headache; drug incompatibility	Red

Primary Thunderclap Headache

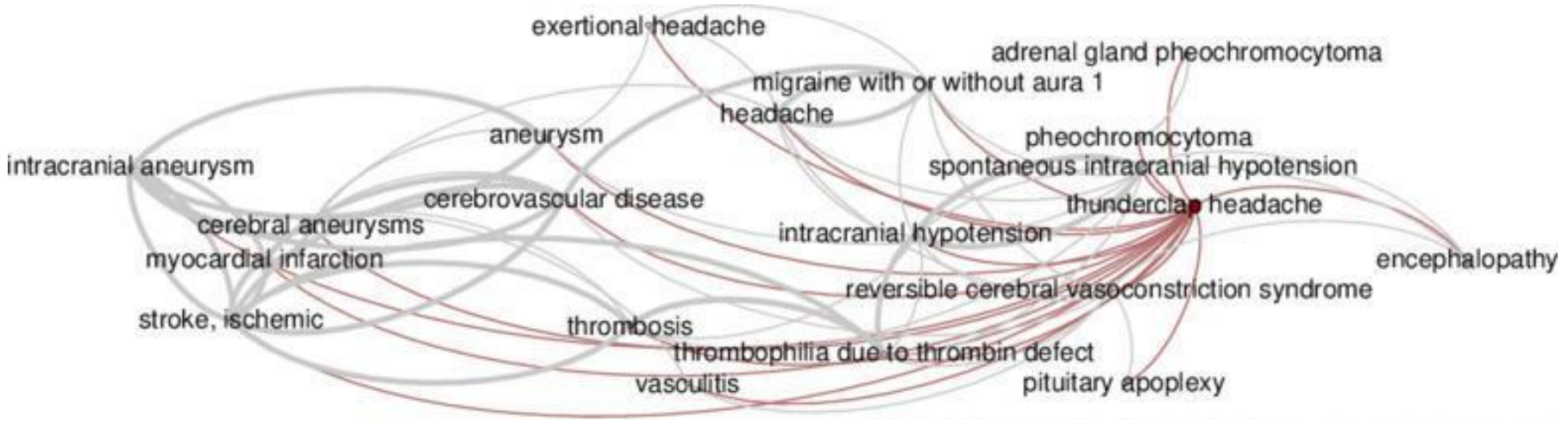
Primary thunderclap headache is a **controversial diagnosis of exclusion**.

Though in the ICHD classification since 2004, most headache specialists **question its existence** and believe that secondary causes were missed due to insensitive, incomplete, or poor timing of investigations.

Vasoconstrictions may not be observed in the early stage of RCVS. For this reason, probable primary thunderclap headache is not a diagnosis that should be made even temporarily.

A better terminology suggested would be **“thunderclap headache of undefined origin.”**

Pain lasts from minutes to hours, and can linger for days to weeks. They can recur over the next two weeks, and less commonly over months to years.



Graphical network of the top 20 diseases related to Thunderclap Headache

Thunderclap Headache in the Emergency Department

A thunderclap headache is normally defined as **severe** in character and reaching **maximum severity within seconds to minutes of onset**. An even more restrictive definition states that it must be of **instantaneous onset**, reaching **maximum** intensity **within one minute**.

A person presenting with a thunderclap headache is at high risk of a **life-threatening condition – 4 – 5 times greater risk** than those who are in the "rule- out **MI**" or "rule-out **pulmonary embolism**" category.

Of those who do have a **high-risk condition, 30 – 50 % will not have a subarachnoid hemorrhage (SAH).**

Many emergency physicians **discharge people with thunderclap headaches after a computed tomography (CT) and a lumbar puncture (LP) have ruled out an SAH, but this strategy needs to change** and encompass consultation and additional imaging if that first series of investigations is negative.

There are many other conditions (different from SAH) that can present with a thunderclap headache.

Panel 3: Causes of thunderclap headache

Usually detected by non-contrast CT

- Subarachnoid haemorrhage (most cases detected by non-contrast CT done within 24 h of symptom onset)
- Intracerebral haematoma
- Intraventricular haemorrhage
- Acute subdural haematoma
- Cerebral infarcts (after 3 h)
- Tumours (eg, third ventricle colloid cyst)
- Acute sinusitis

Usually detected by analysis of CSF after normal CT

- Subarachnoid haemorrhage
- Meningitis

Possibly presenting with normal CT results and normal or near-normal results of analysis of CSF

- Intracranial venous thrombosis
- Dissection of cervical arteries (extra or intracranial, carotid or vertebral)
- Pituitary apoplexy
- Reversible cerebral vasoconstriction syndrome with or without posterior reversible encephalopathy syndrome
- Symptomatic aneurysm without evidence of subarachnoid haemorrhage (painful third nerve paralysis)
- Intracranial hypotension (CSF pressure low)
- Cardiac cephalalgia due to myocardial ischaemia (very rare)

It is important to recognize that thunderclap headaches are neither sensitive nor specific for the diagnosis of SAH – about **50 % of patients with SAH present with a thunderclap headache**, while **SAH is the identified cause in only 11– 21 % of patients who present with thunderclap headache**.

Approximately **25 to 30 % of patients** presenting to an emergency department **with a thunderclap headache** will be suffering from a **lifethreatening condition**, making this one of the most high-risk symptoms emergency physicians encounter.

A significant proportion of these life-threatening conditions will not be an SAH .

CAN'T MISS CONDITIONS

SAH
Arterial Dissection
Cerebral Venous Sinus Thrombosis
Posterior Reversible Encephalopathy Syndrome (PRES)

NON-LIFE-THREATENING CONDITIONS

Reversible Cerebral Vasoconstriction Syndrome (RCVS)
Intracranial Hypotension

Table 5.2 Symptoms associated with diagnoses presenting with thunderclap headache

Diagnosis	Associated symptoms
Carotid artery dissection	Ipsilateral neck pain of gradual onset, up to two weeks prior to headache or neurological symptoms. Onset with minor neck trauma
Vertebral artery dissection	Occipitonuchal pain of gradual onset, vertigo, posterior circulation neurological findings. Onset with minor neck trauma
Aortic dissection	Chest pain
RCVS	Recurrent abrupt headaches over 2–3 weeks
Intracerebral hemorrhage	Vomiting, decreased level of consciousness, focal neurological findings. "Focal findings trump headache" [6]
Cerebral sinus thrombosis	Diplopia, blurred vision, focal headache, seizures, decreased mental status
SAH	Nausea, vomiting, syncope, photophobia, nuchal rigidity. "Headache trumps focal findings" [6]
PRES	Seizures, decreased mental status, visual symptoms
Intracranial hypotension	Orthostatic symptoms, recent dural puncture
Temporal arteritis (vasculitis)	Polymyalgia rheumatica, xerostomia, masseter claudication, abrupt loss of vision from one eye, non-contiguous focal deficits
Hydrocephalus	Nausea, vomiting, decreased mental status

Stroke. 2007;38:1216-1221.

Missed Diagnosis of Subarachnoid Hemorrhage in the Emergency Department

Marian J. Vermeulen, MHSc; Michael J. Schull, MD, MSc, FRCPC

Subarachnoid hemorrhage (SAH) can be devastating, yet its initial presentation may be limited to common symptoms and subtle signs, potentially leading to misdiagnosis.

Authors studied persons admitted with a **nontraumatic SAH** to all **Ontario hospitals** over 3 years (April 2002 to March 2005).

Of 1507 patients diagnosed **with SAH**, **5.4%** (95% CI, 4.3 to 6.6) had a **missed diagnosis**. The risk was significantly higher among patients triaged as low acuity (odds ratio 2.65; 95% CI, 1.46 to 4.80).

About 1 in 20 SAH patients are missed during an ED visit. **Lower acuity patients are at higher risk of misdiagnosis**, suggesting the need for heightened suspicion among patients with minimal clinical findings.

Warning, or sentinel leaks associated with aneurysmal expansion, may precede major, clinically devastating SAH in up to half of cases.
Typically the **thunderclap headache is the only symptom in such sentinel events, and can resolve within hours to days**.
It is important to recognize that a thunderclap headache related to a sentinel bleed **may have resolved** by the time the patient is seen **in the ED** – such patients must not be ignored despite their normal appearance and absence of headache at the time of their evaluation.

Original Investigation

Clinical Decision Rules to Rule Out Subarachnoid Hemorrhage for Acute Headache

JAMA September 25, 2013 Volume 310, Number 12

Jeffrey J. Perry, MD, MSc; Ian G. Stiell, MD, MSc; Marco L. A. Sivilotti, MD, MSc; Michael J. Bullard, MD; Corinne M. Hohl, MD, MHSc; Jane Sutherland, MEd; Marcel Émond, MD, MSc; Andrew Worster, MD; Jacques S. Lee, MD, MSc; Duncan Mackey, MD; Merril Pauls, MD; Howard Lesiuk, MD; Cheryl Symington, RN, ENCC; George A. Wells, PhD

Of the 2131 enrolled patients, 132 (6.2%) had SAH.

The decision rule including any of age 40 years or older, neck pain or stiffness, witnessed loss of consciousness, or onset during exertion had 98.5%(95%CI, 94.6%-99.6%) sensitivity and 27.5%(95%CI, 25.6%-29.5%) specificity for subarachnoid hemorrhage.

Adding "thunderclap headache" (ie, instantly peaking pain) **and "limited neck flexion on examination"** resulted in the **Ottawa SAH Rule**, with **100%** (95%CI, 97.2%-100.0%) **sensitivity** and **15.3%** (95%CI, 13.8%-16.9%) **specificity**.

Box 2. The Ottawa SAH Rule

For alert patients older than 15 y with new severe nontraumatic headache reaching maximum intensity within 1 h

Not for patients with new neurologic deficits, previous aneurysms, SAH, brain tumors, or history of recurrent headaches (≥3 episodes over the course of ≥6 mo)

Investigate if ≥1 high-risk variables present:

1. Age ≥40 y
2. Neck pain or stiffness
3. Witnessed loss of consciousness
4. Onset during exertion
5. Thunderclap headache (instantly peaking pain)
6. Limited neck flexion on examination

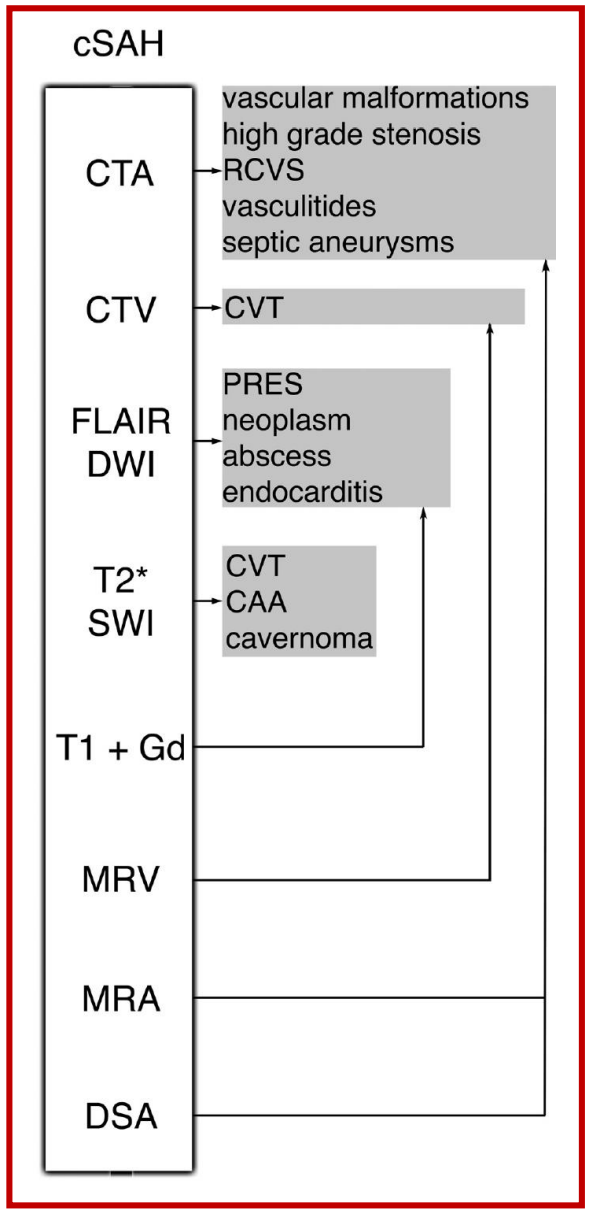
SAH indicates subarachnoid hemorrhage.

Isolated Acute Nontraumatic Cortical Subarachnoid Hemorrhage

AJNR Am J Neuroradiol 31:1355–62 | Sep 2010

REVIEW ARTICLE

V. Cuvinciuc
A. Viguier
L. Calviere
N. Raposo
V. Larrue
C. Cognard
F. Bonneville



Etiology of cSAH

Etiology
Pial arteriovenous malformations
Dural arteriovenous fistulas
Arterial dissection
Dural/cortical venous thrombosis
Vasculitides
RCVS reversible cerebral vasoconstriction syndrome
PRES posterior reversible encephalopathy syndrome
High-grade stenosis
Endocarditis
CAA cerebral amyloid angiopathy
Coagulation disorders
Abscess
Cavernoma
Primary and secondary brain tumors

ABBREVIATIONS: CAA = cerebral amyloid angiopathy; cSAH = cortical subarachnoid hemorrhage; CTA = CT angiography; CTV = CT venography; CVT = cerebral venous thrombosis; DSA = digital subtraction angiography; DWI = diffusion-weighted imaging; FLAIR = fluid-attenuated inversion recovery; Gd = gadolinium; GRE T2 = gradient echo T2-weighted imaging; MRA = MR angiography; MRV = MR venography; PRES = posterior reversible encephalopathy syndrome; RCVS = reversible cerebral vasoconstriction syndrome; SAH = subarachnoid hemorrhage; SWI = susceptibility-weighted imaging; TIA = transient ischemic attack; TOF = time of flight

Ann Emerg Med. 2013;62:1-10

Nontraumatic Subarachnoid Hemorrhage in the Setting of Negative Cranial Computed Tomography Results: External Validation of a Clinical and Imaging Prediction Rule

Dustin G. Mark, MD; Yun-Yi Hung, PhD; Steven R. Offerman, MD; Adina S. Rauchwerger, MA; Mary E. Reed, DrPH; Uli Chettipally, MD, MPH; David R. Vinson, MD; Dustin W. Ballard, MD, MBE; for the Kaiser Permanente CREST Network Investigators

In this case-control study performed in 21 emergency departments over a 12-year period (2000-2011), **55 cases of SAH** subarachnoid hemorrhage were identified, 34 (62%) of which were attributed to cerebral aneurysms.

Negative CT result for tests performed within 6 hours of headache onset **does not rule out subarachnoid hemorrhage**; so this study **failed to validate the six-hour rule**.

Head CT performed within six hours demonstrated a **sensitivity of 97.1%**, a **specificity of 22.7%**, and a negative likelihood ratio of 0.13 for a diagnosis of subarachnoid hemorrhage.

11 of 55 subarachnoid hemorrhage cases (20%) had negative cranial CT results for tests performed within 6 hours of headache onset.

Volume 51, NO. 6 : June 2008

NEUROLOGY/ORIGINAL RESEARCH

Is the Combination of Negative Computed Tomography Result and Negative Lumbar Puncture Result Sufficient to Rule Out Subarachnoid Hemorrhage?

Jeffrey J. Perry, MD, MSc
Alena Spacek, MD
Melissa Forbes, MD

From the Department of Emergency Medicine (Perry, Spacek, Mortensen, Symington, Fortin, Stiell), Department of Family Medicine (Forbes), and Department of Epidemiology and Community Medicine (Wells), University of Ottawa, Ottawa, Ontario, Canada.

In a prospective study of nearly 600 patients, the **combination of CT followed by LP** was found to have a **sensitivity of 100 percent** and a **negative likelihood ratio of 0 for SAH**.

This study validates current clinical practice that **a negative CT result with a negative lumbar puncture result is sufficient to rule out SAH** subarachnoid hemorrhage in patients presenting to the ED with an acute headache.

the bmj | BMJ 2015;350:h568 | doi: 10.1136/bmj.h568

Differentiation between traumatic tap and aneurysmal subarachnoid hemorrhage: prospective cohort study

Jeffrey J Perry,[1] Bader Alyahya,[1] Marco L A Sivilotti,[2] Michael J Bullard,[3] Marcel Émond,[4] Jane Sutherland,[5] Andrew Worster,[6] Corinne Hohl,[7] Jacques S Lee,[8] Mary A Eisenhauer,[9] Merril Pauls,[10] Howard Lesiuk,[11] George A Wells,[12] Ian G Stiell[1]

WHAT IS ALREADY KNOWN ON THIS TOPIC

It is often difficult to differentiate blood in cerebrospinal fluid from subarachnoid hemorrhage or as a result of traumatic tap

WHAT THIS STUDY ADDS

A red blood cell count of less than 2000 × 10^6/L and no xanthochromia reasonably rules out an aneurysmal subarachnoid hemorrhage

In the right clinical setting, the presence of **xanthochromia** or **red blood cells in the CSF** raises concern for SAH. **The absence of both of these findings excludes the diagnosis**. However, relying on presence of **red blood cells** results in **false positive rates as high as one in three**.

The sensitivity of **xanthochromia is time-dependent: if fewer than six hours have elapsed since the headache began, xanthochromia is frequently not present** and one must rely on the presence of red blood cells to exclude disease .

Table 2 | Characteristics of 641 patients with acute non-traumatic headache with abnormal results on lumbar puncture. Figures are numbers (percentage) of patient unless otherwise specified

	Aneurysmal subarachnoid hemorrhage (n = 15)	No subarachnoid hemorrhage (n = 626)	P value
Mean (SD) age (years)	49.6 (14.2)	45.0 (16.2)	0.28
Women	10 (66.7)	359 (57.3)	0.47
Median (IQR) time of headache onset to peak (seconds)	38 (3–210)	10 (1–120)	0.64
Median (IQR) time from headache onset to lumbar puncture (hours)	30.0 (14.0–120)	18.7 (9.0–50.8)	0.13
Positive xanthochromia	46.6	2.6	< 0.001
Median (IQR) red blood cells count in last tube (× 10^6/L)	28 741 (12 468–74 789)	20 (7–110)	< 0.001
Median (IQR) cerebrospinal fluid protein concentration (g/L)	0.56 (0.38–0.92)	0.40 (0.31–0.54)	0.033
Angiography rate	15 (100)	404 (64.5)	0.004

*IQR = interquartile range.

AJNR Am J Neuroradiol 26:820–824, April 2005

Clearing of Red Blood Cells in Lumbar Puncture Does Not Rule Out Ruptured Aneurysm in Patients with Suspected Subarachnoid Hemorrhage but Negative Head CT Findings

D. Cressler Heasley, Mona A. Mohamed, and David M. Yousem

In evaluating the results of lumbar puncture (LP), **a decrease in the number of red blood cells (RBCs)/mm3 between the first and fourth tubes collected (clearing) has often been assumed to indicate a traumatic puncture** rather than the presence of subarachnoid hemorrhage (SAH).

There are reports of patients who were ultimately diagnosed with aneurysmal SAH with clear CSF and as few as 100 red cells in tube 4.

A 25% reduction in RBC concentration between the first and fourth tubes of CSF in patients with suspected SAH but negative CT findings occurs even in cases of ruptured aneurysms. Formal evaluation for the presence of an aneurysm is still necessary in this scenario.

TABLE 1: Patients with negative head CT findings and aneurysms on angiography

Patient	Time from Symptom Onset to Head CT	Time from Head CT to LP	Tube 1 (RBCs/mm^3)	CSF Description	Tube 4 (RBCs/mm^3)	CSF Description	% Change from Tube 1 to 4	Angiographic Result
1	**4 days**	**5 hours**	**3550**	**Cloudy**	**2550**	**Slightly bloody**	**−28.2**	**Basilar artery dissecting aneurysm**
2	**3 days**	**5 hours**	**24,686**	**Cloudy**	**17,842**	**Bloody**	**−27.7**	**Right posterior communicating artery aneurysm**
3	8 days	2 hours before CT	14,325	Xanthochromic	11,207	Xanthochromic	−21.8	Anterior communicating artery aneurysm
4	7 days	1 hour	2249	Cloudy	2129	Bloody	−5.3	Left vertebral artery dissection
5	3 hours	3 hours	29,710	Cloudy	33,700	Bloody	13.4	Anterior communicating artery aneurysm
6	2 days	6 hours before CT	69	Clear	100	Clear	45	Right middle cerebral artery aneurysm
7	24 days	2 hours	192	Clear	1150	Slightly bloody	499	Anterior communicating artery aneurysm
8	3 hours	9 hours	80	Clear	6225	Slightly bloody	7700	Right anterior communicating artery aneurysm

Stroke. 2009;40:994-1025.

AHA/ASA Guideline

Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage

A Statement for Healthcare Professionals From a Special Writing Group of the Stroke Council, American Heart Association

The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists.

Joshua B. Bederson, MD, Chair; E. Sander Connolly, Jr, MD, FAHA, Vice-Chair; H. Hunt Batjer, MD; Ralph G. Dacey, MD, FAHA; Jacques E. Dion, MD, FRCPC; Michael N. Diringer, MD, FAHA; John E. Duldner, Jr, MD, MS; Robert E. Harbaugh, MD, FAHA; Aman B. Patel, MD; Robert H. Rosenwasser, MD, FAHA

Sensitivity for aneurysm of the neurovascular imaging modalities, CTA (computed tomography angiography), MRA (magnetic resonance angiography) and angiography, varies by aneurysm size.

Using **Angiography** as the gold standard, for **aneurysms ≥ 5 mm, CTA has a sensitivity of 95– 100 %,** while for aneurysms **< 5 mm**, sensitivity ranges between **64 and 83 %**.

For **non-contrast time-of-flight MRA (magnetic resonance angiography), sensitivity for aneurysms ≥ 5 mm is 85–100 %**, while for aneurysms **< 5 mm** sensitivity is **56 %**.

If the CT + LP are negative, the job is not done:
consultation with a neurologist or radiologist should be the next step, in order to discuss which form of cervicocephalgic vascular imaging should be performed.

In a patient presenting with a **thunderclap headache** the data showing that at least **30–40 % of these conditions will not be identified with a CT + LP approach**.

Table 5.1 Differential diagnosis of thunderclap headache

Identifiable with CT	Identifiable with LP	Normal CT and LP
Subarachnoid hemorrhage	Subarachnoid hemorrhage	Arterial dissection extracranial -Carotid -Vertebral Intracranial
Intracerebral hemorrhage	Meningitis -bacterial -viral	Symptomatic aneurysm with mass effect
Intraventricular hemorrhage		Reversible cerebral vasoconstriction syndrome
Acute subdural hemorrhage		Posterior reversible encephalopathy syndrome
Ischemic stroke		Cerebral venous sinus thrombosis
Tumor (third ventricle colloid cyst, posterior fossa tumor)		Pituitary apoplexy
Hydrocephalus (aqueductal stenosis, Chiari type 1 malformation)		Intracranial hypotension (usually unidentified CSF leak)
		Ischemic stroke in first three hours
		Temporal arteritis
		Myocardial ischemia
		Aortic dissection

Devenney *et al. The Journal of Headache and Pain* 2014, **15**:49

A systematic review of causes of sudden and severe headache (Thunderclap Headache): should lists be evidence based?

Emma Devenney, Hazel Neale and Raeburn B Forbes*

Table 3 Sudden AND severe headache causation by group and type of publication

Cause	Cohort (N)	%	Case (N)	%	Case series	%	All (N)	%
Idiopathic thunderclap headache	265	16	87	27	107	29	459	20
Other, not specified	457	28	18	6	0	0	475	20
Primary headache	447	27	0	0	0	0	447	19
Cerebrovascular	281	17	153	47	198	53	632	27
Infection	119	7	17	5	24	6	160	7
Unknown	49	3	0	0	0	0	49	2
Non-neurovascular	27	2	46	14	46	12	119	5
Sudden death with headache	4	0.2	0	0	0	0	4	0
Total	**1,649**	**100**	**321**	**99**	**375**	**100**	**2345**	**100**

In patients with thunderclap headache who have no evidence of hemorrhage on CT or in CSF, vascular imaging is indicated to exclude other serious vascular causes which are often or always undetectable on CT/ CSF.

These include **RCVS (reversible cerebral vasoconstriction syndrome)**, which has emerged as **one of the most common causes of thunderclap headache**.

Since RCVS may be associated with **intracranial hemorrhage within the first week** or **ischemic stroke within the second week after onset**, **prompt diagnosis is urgent**. It is important to remember that even in patients with documented RCVS, the **initial non-invasive vascular imaging is normal in up to 20 percent of patients**. It is therefore important to ensure that these patients have outpatient **follow-up and imaging is indicated within 7– 10 days**. In addition, non-invasive vascular imaging (CTA/ MRA) may identify carotid or vertebral artery dissection or cerebral venous sinus thrombosis.

Headache and neck pain in spontaneous internal carotid and vertebral artery dissections

NEUROLOGY 1995;45:1517-1522

Peter L. Silbert, MBBS, FRACP; Bahram Mokri, MD; and Wouter I. Schievink, MD

Neck pain is the most prevalent symptom for **vertebral or carotid artery dissection**.

It may be the **presenting symptom in 50– 60 %** of patients with **vertebral artery dissection (VAD)**, and can precede any other symptom by up to two weeks.

Thunderclap headache as the initial symptom occurs about **20 %** of the time.

The **headache** is classically **occipital** and may be bilateral in those with unilateral VAD, often associated with **vertigo**.

In those with **carotid artery dissection (CAD)**, the **pain** may involve the **neck**, jaw, face, and periorbital and/ or temporal region on the **ipsilateral** side, and is invariably **unilateral**.

Diagnosis must be rapid: the **mean time from headache onset to the occurrence of ischemic stroke may be as short as 15 hours**.

Carotid artery dissection often occurs after seemingly trivial trauma – hyperextension of the neck, rapidly shaking the head, even massage and manipulation.

Unless complicated by hemorrhagic or ischemic stroke, cervical arterial dissection will not result in abnormal findings on either noncontrast head CT or LP .

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

N ENGL J MED 352;17 WWW.NEJM.ORG APRIL 28, 2005

CURRENT CONCEPTS

Thrombosis of the Cerebral Veins and Sinuses

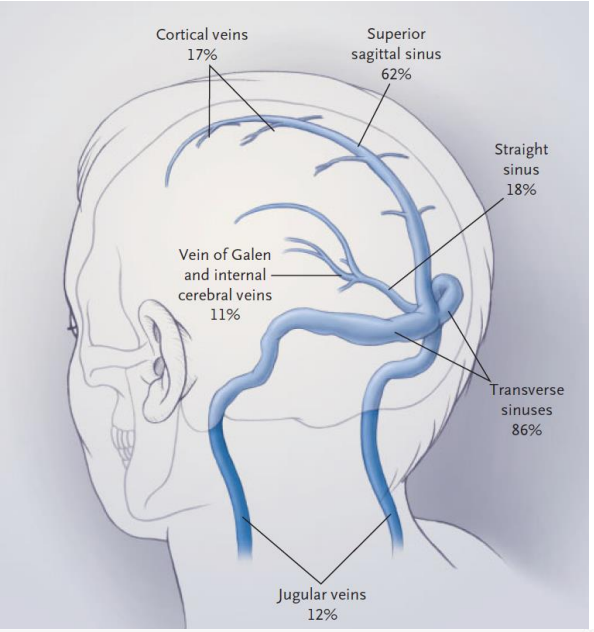
Jan Stam, M.D., Ph.D.

The headache may be **thunderclap** in approximately **15 %** of cases; **headache** of some form will be present in up to **90 %** of cases and may be the sole manifestation of the condition in as many as 10 %.

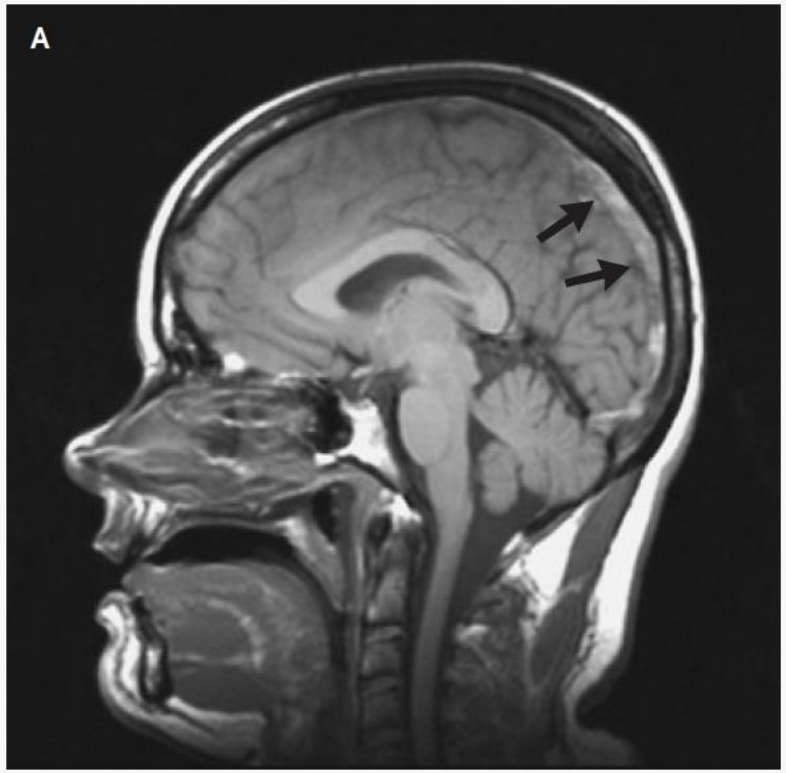
Initial early CT (with or without contrast) may be normal. Non-invasive **venography (CT or MR)** is often diagnostic.

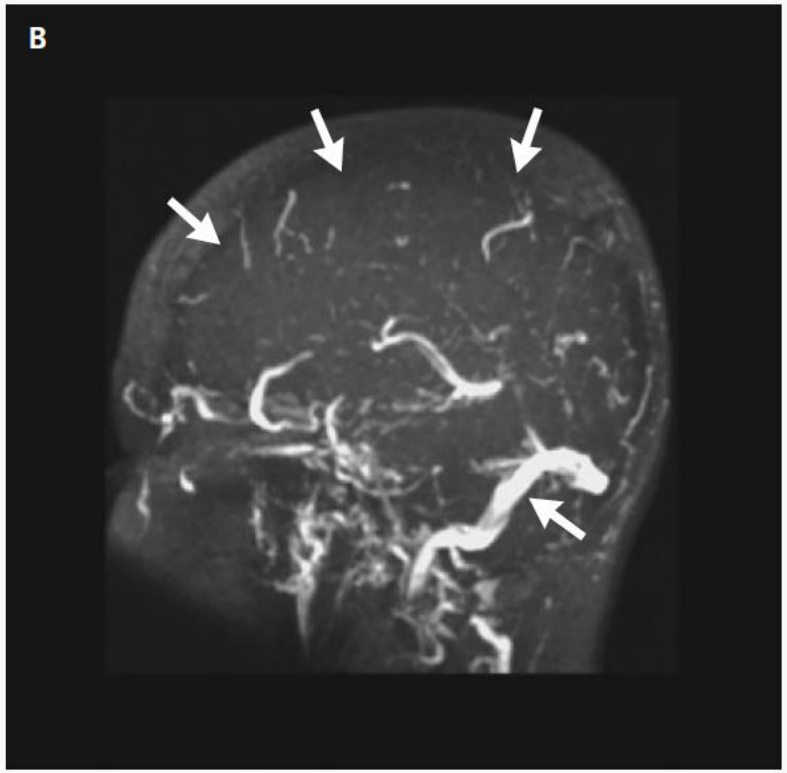
It is felt that the obstruction of venous return leads to a rapid rise in intracranial pressure and thus an abrupt onset headache.

Symptoms can be non-specific, with headache lasting months.
In most, however, the **clinical course is fulminant**, with rapid clinical deterioration involving focal **neurological deficits** and **seizures**.



T1 MRI scan obtained with a hyperintense signal in the thrombosed superior sagittal sinus (arrows).
In Panel B, a MR venogram reveals the absence of a signal in the superior sagittal sinus (upper arrows)





Approach to Pregnant or Lactating Patients with Headache in the ED

Migraine frequency typically decreases early in pregnancy, with approximately half of pregnant women with migraine improving during the first trimester and up to 87 percent during the third trimester. Only 3–7 % of women note the onset of new migraine during pregnancy, typically occurring during the first trimester. At least **half of women experience recurrence of migraine attacks within the first month after delivery and at least two-thirds by six months**.

Since many women limit their exposure to drugs during pregnancy or breastfeeding, a migraine may be undertreated, become severe, refractory, and result in the need for an ED visit.

Migraines during pregnancy linked to stroke and vascular diseases: US population based case-control study

Cheryl D Bushnell,[1] Margaret Jamison,[2] Andra H James[3]

Pregnant women with a history of migraine have a higher risk of developing hypertensive disorders, including **pre-eclampsia** and **stroke**, than those without migraine.

Overweight or **obese migraineurs** (compared to lean non-migraineurs) had a 6.10- fold increased odds of pre-eclampsia.

Late pregnancy is a hypercoagulable state: **pregnant women are four times more likely to develop venous thrombotic disease** than non-pregnant women.

Additionally, the first six weeks postpartum show a 20–80-fold higher risk of venous thrombosis with **a peak of 100-fold higher risk in the first week postpartum** compared to the risk during pregnancy.

BMJ | 4 APRIL 2009 | VOLUME 338

LOGISTIC REGRESSION ANALYSIS OF ASSOCIATIONS WITH MIGRAINE DISCHARGE CODES

Independent variable	Odds ratio (95% CI)	P value
Age	1.03 (1.02 to 1.03)	<0.001
Pre-eclampsia	2.29 (2.13 to 2.46)	<0.001
All strokes*	15.05 (8.26 to 27.4)	<0.001
Venous thromboembolism or pulmonary embolus*	3.23 (2.06 to 7.07)	<0.001
Acute myocardial infarction or heart disease*	2.11 (1.76 to 2.54)	<0.001
Hypertension*	8.61 (6.43 to 11.54)	<0.001

**Pre-eclampsia or eclampsia codes excluded from records*

Acute headache diagnosis in pregnant women

A hospital-based study

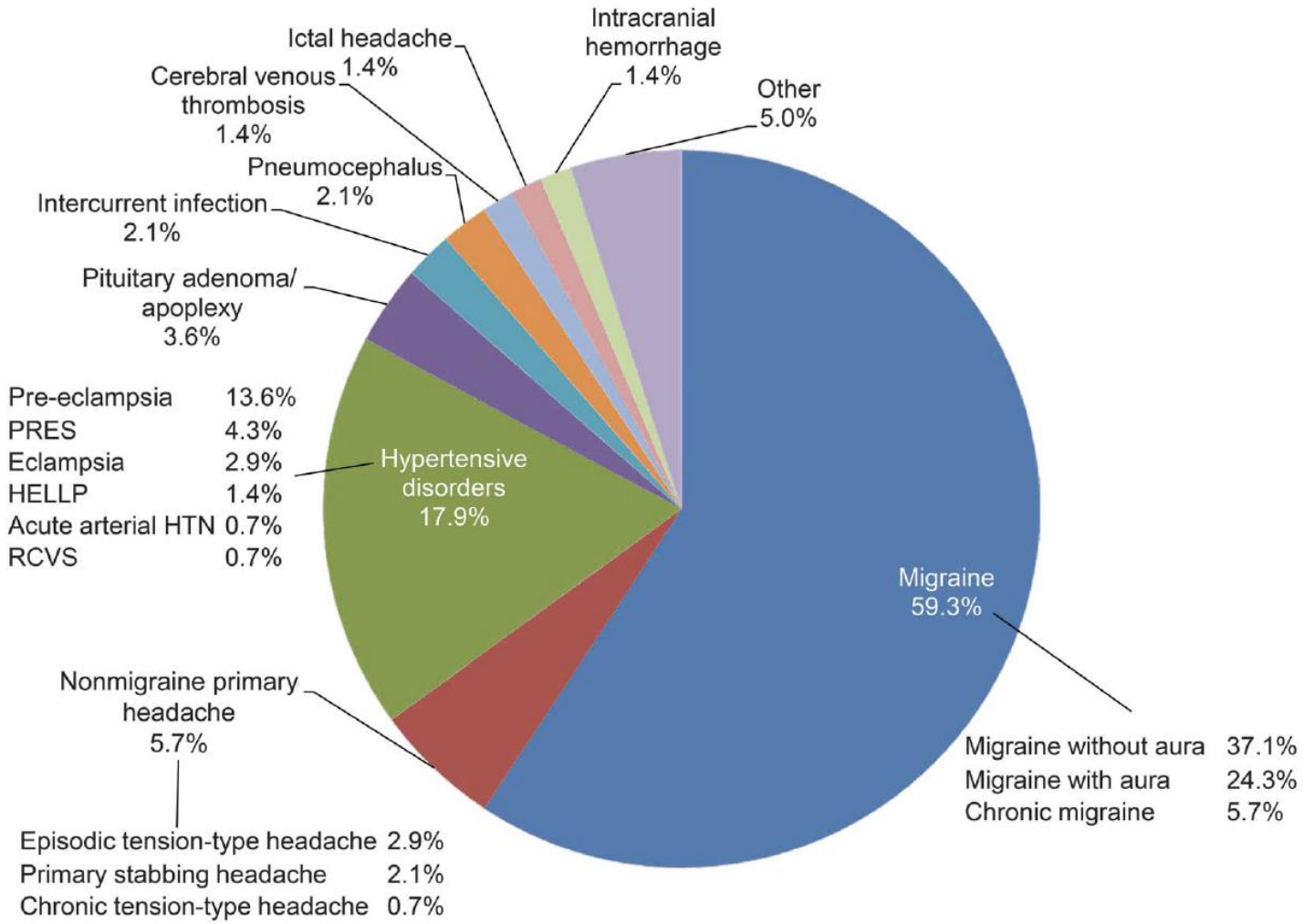
Matthew S. Robbins, MD
Constantine Farmakidis, MD
Ashlesha K. Dayal, MD
Richard B. Lipton, MD

Neurology® 2015;85:1024-1030

In this study of 140 pregnant patients presenting to a single acute care center with headache and receiving neurologic consultation, 56 % of these women were in their third trimester and 35 % were diagnosed with secondary headache.

MRI scans are preferable to CT scans.
CT iodinated contrast dye is likely safe in pregnancy, but gadolinium should be avoided during pregnancy unless absolutely necessary.

Clinical suspicion for a **secondary headache** should be high in the **third trimester** because **primary headaches typically improve but vascular risk increases** in the pregnant patient.



Reversible cerebral vasoconstriction syndrome

Lancet Neurol 2012; 11: 906–17

Anne Ducros

Emergency Headache Centre, Head and Neck Clinic, Lariboisière Hospital, Paris, France (A Ducros MD)

Until roughly ten years ago, 70 % or more of patients with thunderclap headache were felt to have a benign headache of unknown origin. They were often felt to occur in patients already suffering from tension-type headache or migraine.

In 2012, a systematic review by Ducros highlighted that reversible cerebral vasoconstriction is more common than previously thought. **RCVS presents as isolated headaches in about 75 % of cases**.

More than half the cases occur **post partum** or after exposure to **adrenergic or serotonergic drugs**.

Many patients with this disorder may have **several thunderclap headaches over several weeks**.

As would be expected, CT and cerebrospinal fluid (CSF) are normal in most, although roughly 20 % have been found to have a small sulcal subarachnoid hemorrhage.

Panel 2: Diagnostic criteria for reversible cerebral vasoconstriction syndrome

- Acute and severe headache (often thunderclap) with or without focal deficits or seizures
- Uniphasic course without new symptoms more than 1 month after clinical onset
- Segmental vasoconstriction of cerebral arteries shown by indirect (eg, magnetic resonance or CT) or direct catheter angiography
- No evidence of aneurysmal subarachnoid haemorrhage
- Normal or near-normal CSF (protein concentrations <100 mg/dL, <15 white blood cells per µL)
- Complete or substantial normalisation of arteries shown by follow-up indirect or direct angiography within 12 weeks of clinical onset

Adapted from the International Headache Society criteria[1] for acute reversible cerebral angiopathy and the criteria proposed in 2007 by Calabrese and coworkers.[2]

Panel 4: Precipitants of reversible cerebral vasoconstriction syndrome

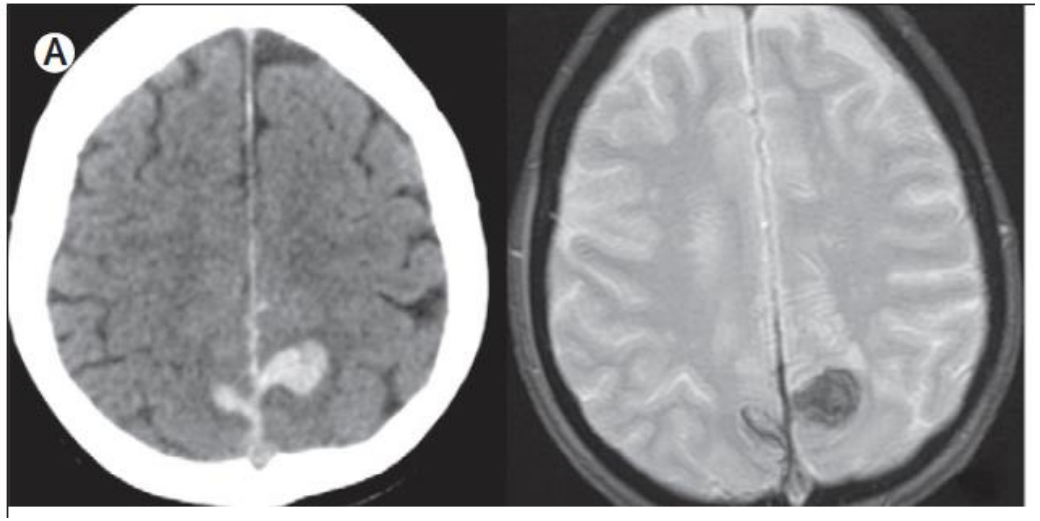
Post partum[2,18,50,97]
- With or without vasoactive substances, with or without eclampsia or pre-eclampsia

Vasoactive drugs[2,5,9]
- Illicit drugs—eg, cannabis,[5,34] cocaine,[105] methylenedioxymethamfetamine,[29] amphetamines, lysergic acid diethylamide
- Antidepressants—eg, selective serotonin reuptake inhibitors,[6,59] serotonin–noradrenaline reuptake inhibitors[9,59]
- α-sympathomimetics—eg, nasal decongestants (phenylpropanolamine, pseudoephedrine, ephedrine),[98,99] norepinephrine[100]
- Triptans[9,41,52,101,102]
- Ergot alkaloid derivatives[50]—eg, methergine, bromocriptine,[103] lisuride[48]
- Nicotine patches[5]
- Ginseng and other herbal medicines[22,53,104]
- Binge drinking[5]

Catecholamine-secreting tumours[15,108]
- Phaeochromocytoma, bronchial carcinoid tumour, glomus tumours

Immunosuppressants or blood products
- Intravenous immunoglobulin,[46] red-blood-cell transfusion,[109] interferon alfa[5]

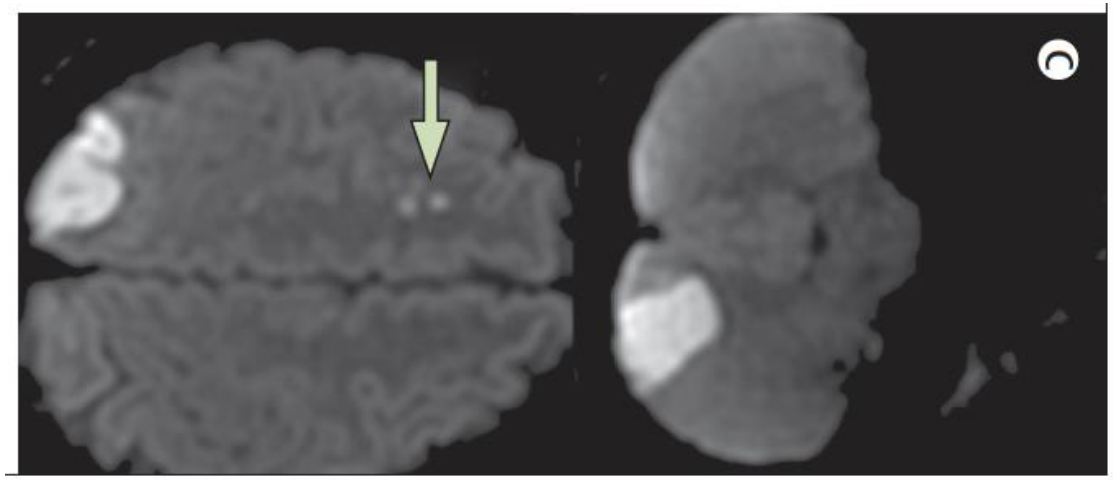
Miscellaneous
- Hypercalcaemia, porphyria, head trauma,[110–112] neurosurgery,[95,113] subdural spinal haematoma, carotid endarterectomy,[55,114] cerebral venous thrombosis,[115] CSF hypotension,[116] autonomic dysreflexia,[117] phenytoin intoxication[118]

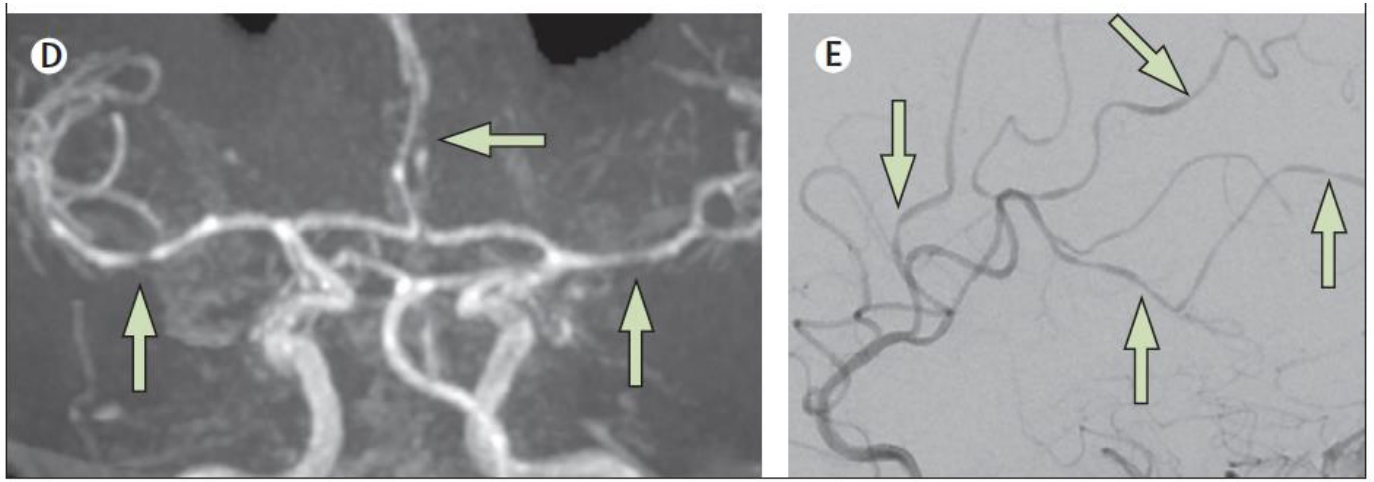


(A) **CT** (left) and **T2*-weighted MRI** (right) scans showing a bilateral occipital haematoma with interhemispheric subarachnoid haemorrhage in a 57-year-old-woman who also had a left capsulothalamic haematoma (not shown).

(C) **Diffusion-weighted MRI** showing a left cerebellar infarction (top), a right occipital infarction (bottom), and patchy small areas of restricted diffusion at the border zone between the right anterior and middle cerebral arteries (arrow) in a 33-year-old female cannabis smoker.

(D) **MR angiogram** showing segmental narrowings (arrows) of the middle and anterior cerebral arteries in the patient shown in (A). (E) Transfemoral angiogram showing segmental narrowings of the branches of the anterior cerebral artery (arrows) in a 58-year-old woman with a left frontal haematoma and SAH in several sulci. A follow-up angiogram at 2 months was normal.





Diagnosis requires non-invasive vascular imaging (**CTA/MRA**) demonstrating **diffuse, segmental, multifocal vasoconstriction**.

This diagnosis is often missed initially – hence the previous belief of thunderclap headache to be of unknown origin – as the **vasoconstriction may take 1– 2 weeks to appear after the first headache** and usually **peaks at week three**.

RCVS-associated **vasoconstriction normally reverses within 12 weeks of onset** .

J Neurol (2017) 264:1608–1616
DOI 10.1007/s00415-016-8377-8

REVIEW

Posterior reversible encephalopathy syndrome

Marlene Fischer[1] · Erich Schmutzhard[2]

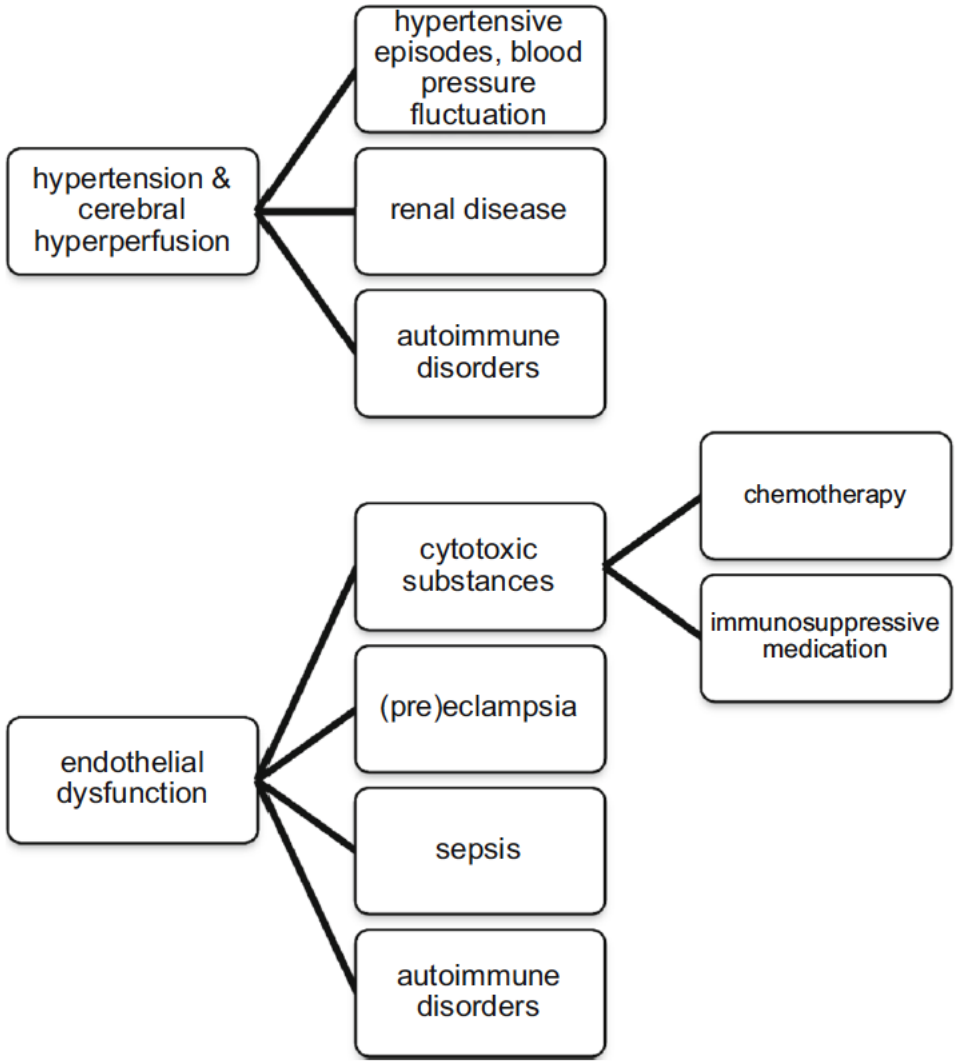
This **almost always occurs** in relationship to a **hypertensive crisis** (eclampsia or hypertension emergency), though other provoking factors have been identified, such as certain **immunosuppressive medications**.

Posterior reversible encephalopathy syndrome can also occur in normotensive people who develop acute hypertension from illicit drug use (**cocaine**), other types of **sympathomimetics** (nasal decongestants containing pseudoephedrine), **reversible cerebral vasoconstriction syndrome**, or **pheochromocytoma**.

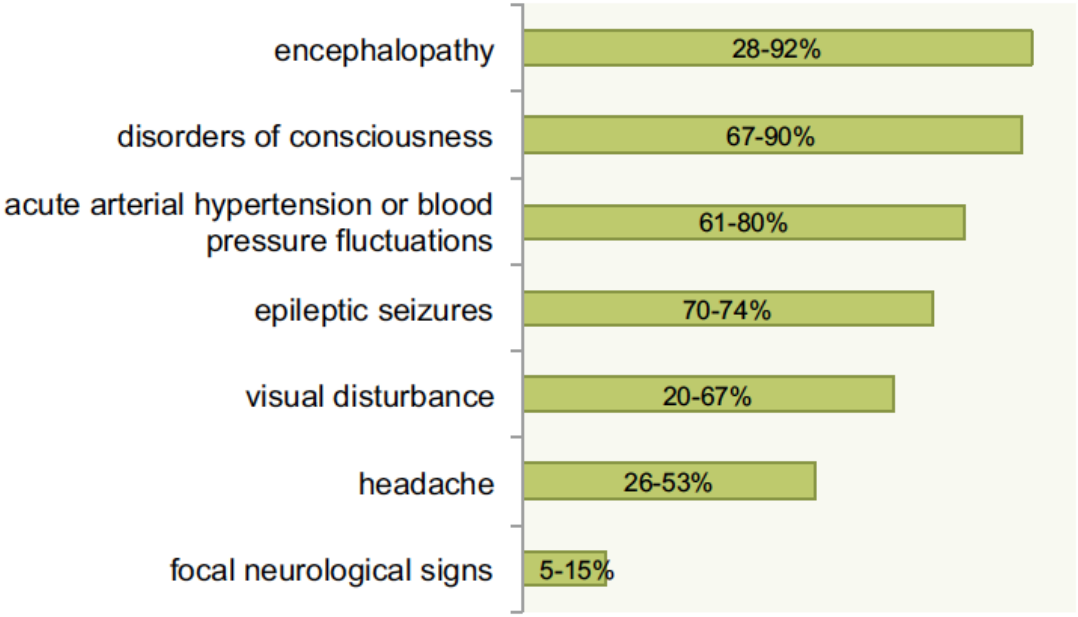
The treatment is symptomatic and is determined by the underlying condition.

The overall prognosis is favorable, since clinical symptoms as well as imaging **lesions are reversible in most patients**.

However, neurological sequelae including **long-term epilepsy** may persist in individual cases.



Rapid neurological deterioration may follow the thunderclap headache – seizures, decreased mental status, and visual symptoms.

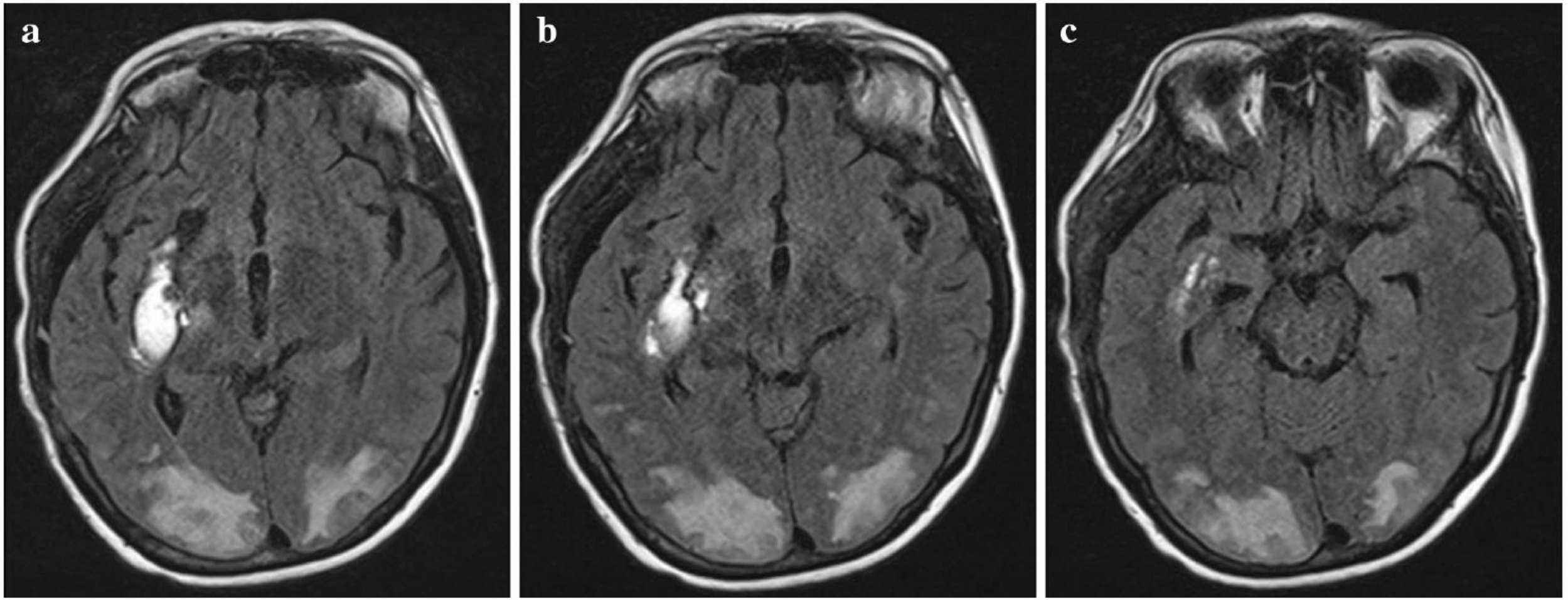


suggested diagnostic criteria

- neurological symptoms of acute onset
- (focal) vasogenic edema on neuroimaging
- reversibility of clin ical and/or radiological findings

Diagnostic tool	Finding
Laboratory data	Hypomagnesemia
	Lactate dehydrogenase ↑
	Liver function parameters ↑
	Creatinine ↑
	Albumin ↓
Cerebrospinal fluid	Albumin ↑
	Albuminocytologic dissociation
EEG	Diffuse theta slowing
	Delta slowing
	Rhythmic delta activity
	Sharp-slow wave activity
	Periodic lateralizing epileptiform discharges
	Diffuse or focal (symmetric) slowing of background activities
CT and MRI	Vasogenic edema
	Watershed distribution
	Parieto-occipital pattern
	Frontal and temporal lobe involvement
	Subcortical white matter lesions
	Bilateral, frequently symmetric distribution
	Hyperintense T2-weighted and FLAIR sequences
	Iso-, hypo-, or hyperintense lesions on DWI
	Facultative contrast enhancement
	Microbleeds, intracerebral hemorrhage possible
	Increased or decreased ADC values depending/indicating (ir)reversibility of lesions
Angiography	Vasoconstriction, vasospasm (diffuse or focal)

There may be **segmented vasoconstriction** identified on **MRA**.



Axial MR image demonstrates **extensive vasogenic edema in the occipital region bilaterally** and **right insular hemorrhage**

Post-traumatic headache

Headache onset within seven days of trauma is required for the diagnosis of post-traumatic headache, though the **vast majority** of headaches that occur as a result of trauma occur **at the onset or within 24 hours** of the head trauma.

Other physical symptoms commonly seen include nausea, photophobia and phonophobia, dizziness, and unsteadiness; difficulties concentrating, sleepiness, insomnia, and psychiatric symptoms including depression, anxiety, and irritability.

The prevalence of persistent/recurrent headache after traumatic brain injury varied substantially in published reports, with a weighted average of more than 50 %.

Table 4.3 ACEP recommendations to obtain or to consider obtaining non-contrast CT of the head following minor trauma

Level A ACEP Recommendation **to obtain** non-contrast head CT for mild head trauma:	Level B ACEP Recommendation **to consider** non-contrast head CT for mild head trauma:
Loss of consciousness **OR** post-traumatic amnesia, AND one or more of the following:	**NO** loss of consciousness **AND NO** post-traumatic amnesia, AND one or more of the following:
• Headache • Vomiting • Age older than 60 years • Intoxication (drug or alcohol) • Short-term memory deficits • Evidence of trauma above the clavicle • Post-traumatic seizure • GCS score less than 15 • Focal neurologic deficit • Coagulopathy	• Focal neurologic deficit • Vomiting • Severe headache • Age 65 years or greater • Signs of a basilar skull fracture • GCS score less than 15 • Coagulopathy • Dangerous mechanism of injury (including ejection from a motor vehicle, struck pedestrian, and a fall from a height of more than 3 feet or 5 stairs)

ACEP (American College of Emergency Physicians)

Journal of Trauma and Acute Care Surgery. 79(2):310–313, AUGUST 2015
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Issn Print: 2163-0755
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Print

Delayed intracranial hemorrhage in the anticoagulated patient: A systematic review

Joseph Miller;Leedor Lieberman;Bashar Nahab;Gina Hurst;Jayna Gardner-Gray;Aaron Lewandowski;Shaw Natsui;Jill Watras;

One normal head CT excludes intracranial hematoma in the vast majority of patients. **Head TC does not need to be repeated**, ***even among those using anticoagulants***, **if symptoms do not progress**.

The studies included data on 1,257 patients, the incidence of **delayed intracranial hemorrhage** ranged **from 5.8 to 72 per 1,000 cases** of patients on **warfarin** with **minor head injury**.

Association for Academic Surgery

Delayed Intracranial Hemorrhage After Blunt Head Injury With Direct Oral Anticoagulants

Tahereh Soleimani, MD, MPH,[a,] Benjamin Mosher, MD,[b] Laura Ochoa-Frongia, MD,[a] Penny Stevens, DNP, RN,[b] and John P. Kepros, MD[c]*

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Multiple studies of **delayed intracranial hemorrhage** have placed the risk among the patients taking **warfarin** at the time of head injury in the range of **0.6-6.0%**.
However, data regarding the risk of delayed intracranial hemorrhage in patients with **Direct-Acting Oral Anticoagulants (DOACs)** remains limited.
In this study **of the 314 patients**, **three** were found to have **delayed intracranial hemorrhage** on the **repeated head CT (0.95%)**.
Two of these three patients were on **concomitant antiplatelet medication**. None of the three individuals required neurosurgical intervention.

Table 2 – Characteristics of the patients with delayed intracranial hemorrhage.

Patient	Sex	Age	Anti coagulant	Mech of injury	Loss of consciousness	Physical signs of head injury	INR	Platelet count	Anti-platelet
Pt #1	M	81	Rivaroxaban	Fall out of bed	No	None	1.3	144K	None
Pt #2	M	88	Apixaban	Fall from standing	Yes	Facial laceration	1.1	66K	Low-dose aspirin
Pt #3	F	92	Apixaban	Fall from standing	No	Scalp laceration	1.0	162K	Clopidogrel

Conclusion: for patients who sustain a blunt head injury while taking only DOACs, that is, **without concurrent antiplatelet medication**, admission, and **repeat head CT may not be necessary after confirming a negative initial CT scan**.

VIEWS & REVIEWS

Headache attributed to autonomic dysreflexia

An underrecognized clinical entity

Julio C. Furlan, MD, MBA, MSc, PhD

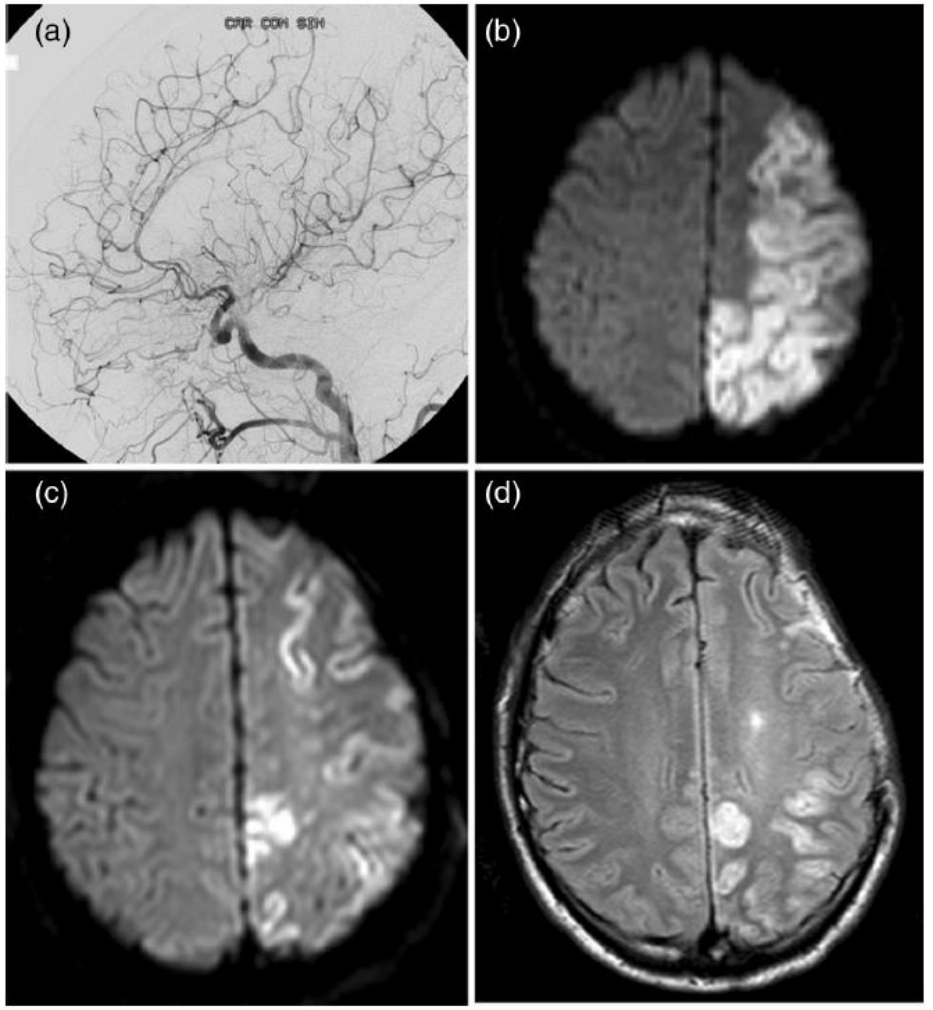
Neurology® 2011;77:792–798

In patients with **acute or chronic spinal cord injuries**, and in those who have recently undergone a **carotid procedure** (e.g., endarterectomy, angioplasty/stenting), a severe, sudden-onset **thunderclap headache with a paroxysmal rise in blood pressure** suggests **autonomic dysreflexia or baroreceptor dysfunction**.

Clinical signs may include diaphoresis, tachycardia as well as blood pressure well above baseline.

The usual **triggers** include bladder distension or infection, bowel distension, or pressure ulcers.

Reversible cerebral vasoconstriction syndrome (RCVS) has been associated with autonomic dysreflexia.

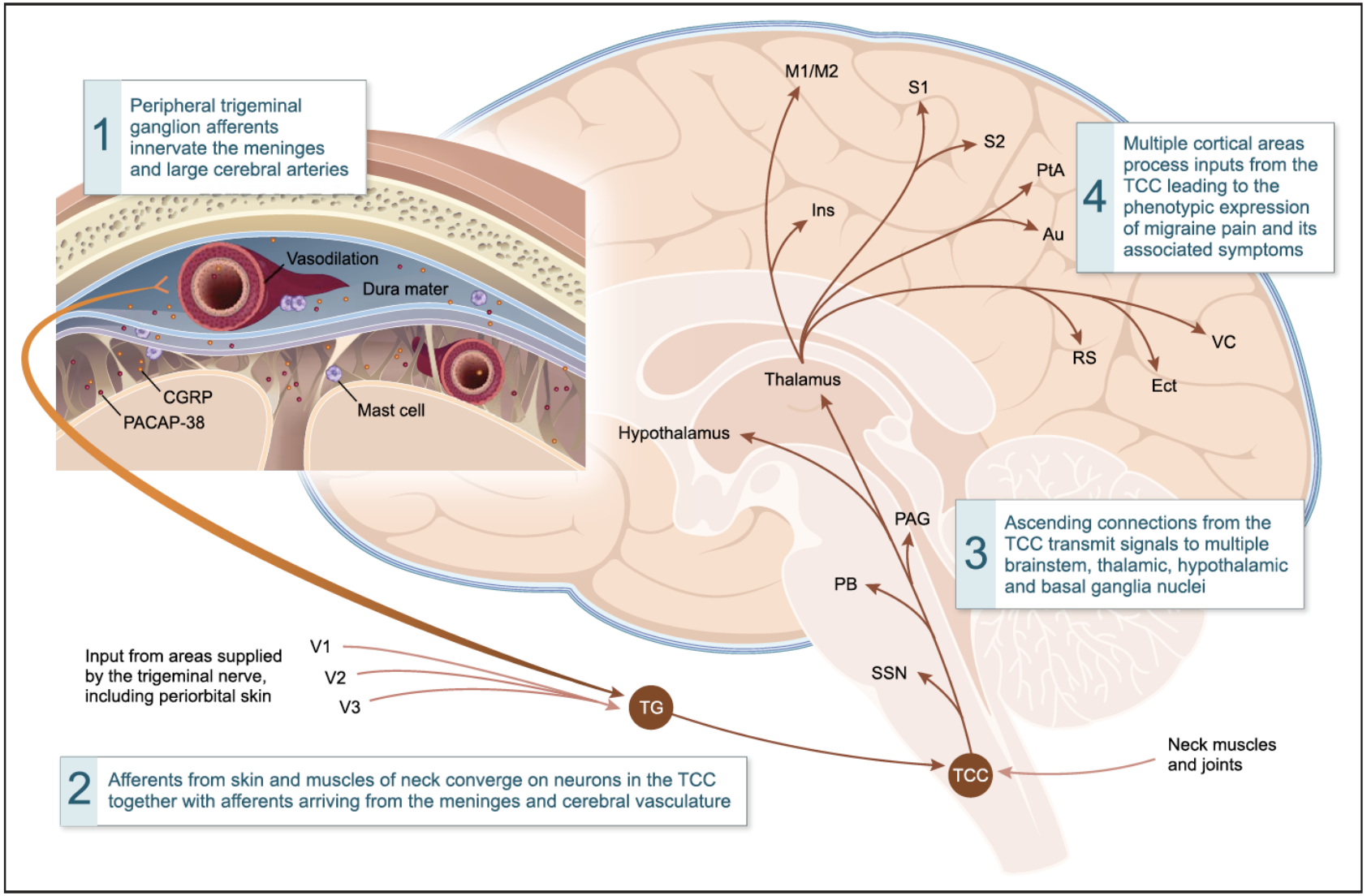


(a) Multiple intracranial blood vessel caliber changes on a selective cerebral angiogram in a patient with spastic quadriparesis from a remote C5– C6 fracture, presenting with reversible cerebral vasoconstriction syndrome in the context of an episode of autonomic dysreflexia.
(b) Diffusion-weighted MRI sequence performed two weeks after the patient's admission showing acute ischemia in the left hemisphere.
(c) Repeat diffusion-weighted MRI sequence three weeks after admission.
(d) T2- weighted MRI sequence three weeks after admission showing expected evolution of the prior ischemic changes.

Cervicogenic Headache

Neck pain is a common symptom in patients with migraine, but occasionally headache may result from cervical spine disease.

The structures of the neck innervated by the first three cervical nerves can be associated with migraine through the convergence of nociceptive afferents at the level of the **caudal part of the trigeminal nucleus in the brainstem** and sensitization of trigeminocervical neurones.



Convergence of trigeminal (dura, skin) and cervical (muscle, joints, skin) afferents on to the same nociceptive second-order neuron in the trigeminocervical complex at the level of C2.