

# Radiological Diagnosis and Differential Diagnosis of Headache

## Radiologische Diagnostik und Differenzialdiagnostik bei Kopfschmerzen im Erwachsenenalter

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### Abstract

▼  
Headache is very common and affects almost everyone at some point. It is one of the most common disorders that leads patients to see their physician. All different forms have the nociception via trigeminal nerve fibers in common. Beside the clinical course headaches are classified as either primary or secondary, with the latter having an identifiable structural or biochemical cause. Imaging has a low diagnostic yield in primary headache but play an important role in the differential diagnosis of secondary forms. An overview of different forms of secondary headache is given, outlining diagnostic procedures and the morphologic imaging features of each syndrome.

#### Key points:

- ▶ Headache can be differentiated in primary and secondary forms.
- ▶ Imaging plays an important role in differential diagnosis of secondary forms.
- ▶ Imaging should be performed in patients with concomitant systemic or neurologic symptoms.

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### Zusammenfassung

▼  
Kopfschmerzen sind eine der häufigsten Beschwerden, die zu einem Arztbesuch führen, und betreffen nahezu jeden einmal in seinem Leben. Gemeinsame pathophysiologische Endstrecke ist die Schmerzwahrnehmung über Trigemina-äste. Neben der Verlaufsform kann hinsichtlich der Ursache zwischen primären und sekundären Kopfschmerzen unterschieden werden, wobei die

sekundären Kopfschmerzen Ausdruck einer zugrunde liegenden Erkrankung sind. Ist die diagnostische Ausbeute bei primären Kopfschmerzen gering, so spielt die Bildgebung bei den sekundären Formen eine entscheidende Rolle in der Differenzialdiagnostik. Verschiedene sekundäre Kopfschmerzformen werden vorgestellt und die zur Verfügung stehenden diagnostischen Verfahren sowie die bildmorphologischen Korrelate der einzelnen Krankheitsbilder beschrieben.

### Introduction

▼  
Almost everyone gets headaches at least once in life; they are among the most common complaints resulting in a visit to a physician. In the literature, their prevalence is indicated to be between 31 – 96% among adults [1]. This wide variance is due to varying definitions, age as well as gender-related and ethnic differences. Thus headaches are reported more frequently in North America and Europe compared to Asia and South America [1]. Kolmann et al. showed that women exhibited a 40 – 70% 7-day/12-month prevalence of headaches, compared to men who exhibited a 20 – 50% prevalence [2]. Brain tumors, with an incidence of 46 per 100 000 are rarely the cause; however, in adults, headaches are the initial symptom in 65% of affected adults. Even lower is the incidence of subarachnoid hemorrhage (SAH) at 6 – 7 cases per 100 000, but headaches are the cardinal symptom in more than 75% of affected patients [3]. In many patients, when a headache first occurs, there is the fear of a severe underlying illness, and there is high expectation that this will be ruled out using imaging. The current classification of the International Headache Society [4] distinguishes between

**Table 1** Classification of headache according to the International Headache Society (IHS) (modified according to [7]).

1. **primary headaches**
  - migraine
  - tension-type headache
  - trigeminal autonomic headache
  - other primary headaches
2. **secondary headaches**
  - headache attributed to head and/or neck trauma
  - headache attributed to head and/or neck trauma
  - headache attributed to cranial or cervical vascular disorder
  - headache attributed to a substance or its withdrawal
  - headache attributed to infection
  - headache attributed to disorder of homeostasis
  - headache or facial pain attributed to disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structures
  - headache attributed to psychiatric disorder
3. **cranial neuralgias and central causes of facial pain**
  - trigeminal neuralgia
  - Tolosa-Hunt syndrome

primary and secondary headache as well as cranial neuropathies and facial pain (► **Table 1**). The pathophysiology of headaches is not fully understood in all cases; however, the common final pathway of all symptom clusters is the perception of pain via trigeminal branches.

This review article will discuss primary and frequent secondary headache in adults as well as examination protocols and strategies for individual symptom patterns.

### Examination Protocols and Strategies

The necessity of imaging and selection of examination modality are primarily dependent on the expected symptom cluster. Additional factors may also include local availability of different examination modalities, the age of the patient and general clinical situation, such as accompanying unconsciousness.

In every case, imaging is indicated in the presence of red flags, including appearance of headaches in cancer or HIV-positive patients, if headaches first appear in a patient after 50 years of age as well as headaches with increasing intensity, accompanying systemic reaction such as fever or focal neurological deficit [5, 6].

In emergency situations when patients have potentially life-threatening causes, e.g. in cases of subarachnoid hemorrhage or headache with accompanying unconsciousness, CT is the preferred diagnostic method due to the shorter examination time as well as better access to the patient and easier monitoring of the patient during the examination.

A CT-based examination protocol (► **Table 2**) should include a non-enhanced CT with an orbital-meatal angulation from the base of the skull to the vertex. This can use a sequential technique as well as a spiral CT with newer equipment. Image data should be reconstructed in the soft tissue and bone window.

If there is suspicion of a vascular process, an arterial or venous CT angiography can follow. An arterial angiography is performed after intravenous administration of 60 ml of an

**Table 2** Basic examination protocol for CT and MRI in cases of headache.

CT	
technique	indication/attribute
Non-enhanced CT	exclude hemorrhage, mass, infarction
– incremental CCT with orbital-meatal layer orientation	
– 4.5 mm slice thickness, supra- and infratentorial	
– reconstruction in soft tissue and bone window	
arterial CT angiography (CTA)	caudocranial scan direction, 60 ml contrast agent with 4/6 ml flow rate, semiautomatic bolus tracking at cervical vertebrae 5/6; scan region aortic arch to vertex
venous CT angiography (CTV)	craniocaudal scan direction; 80 ml contrast agent with 3 ml flow rate, 45 sec scan delay
contrast-enhanced CCT	primarily for infectious process, tumor
MRI	
technique	indication/attribute
– examination using head coil; min. two mutually orthogonal planes	
T1w	anatomy, exclude hemorrhage, mass; if possible, as 3 D T1w MPR, otherwise axial T1w
DWI	axial layer orientation; exclude infarction; indication of encephalitis
T2w FLAIR	axial layer orientation; exclude SAH or demyelinating/inflammatory foci
T2w	sagittal layer orientation; evaluation of aqueduct, midbrain, cerebellum
T2*w (“hemo”)	assessment for microhemorrhage, older hemorrhage
arterial TOF angiography	assess brainstem arteries
arterial CE MRA	assess craniocervical arteries; 0.1 mmol Gd/kg bodyweight; Care bolus technique, start measurement when contrast agent is in aortic arch
Venous CE MRA (MRV)	assess veins and arteries

T1w = T1-weighted images; DWI = diffusion-weighted imaging; T2w = T2-weighted images; T2\*w = T2-weighted gradient echo images; CTA = arterial CT angiography; CTV = venous CT venography; MRA = MR angiography; MRV = MR venography; CE = contrast enhanced

iodine-based contrast agent with a concentration of 300 mg iodine/ml with a flow rate of 6 ml/sec (64/128-slice MDCT) or 4 ml/sec (8/16-slice MDCT). The scan direction is caudocranial; the examination uses semiautomatic bolus tracking started at cervical vertebrae 5/6 if a threshold of 150 HE has been exceeded. In order to simplify planning of potential endovascular therapy, the scan region should extend from the aortic arch to the vertex. Venous CT angiography is performed in craniocaudal scan direction after intravenous administration of 80 ml contrast media using a scan delay of 45 sec with a 3 ml/sec flow rate. Using a shorter scan delay increases the risk of insufficient contrast of the brain nerves.

If in the non-enhanced CT there is suspicion of an infectious or inflammatory event, a contrast-enhanced examination should be performed 5 minutes after administration of the contrast agent. It should be noted, however, that MRI is superior to CT as verification of inflammatory changes.

MR-based diagnosis (► **Table 2**) should be performed using a head coil. Care should be taken that examinations be performed in at least two mutually orthogonal planes using two different weightings.

Using default 3 T, the authors acquire a T1-weighted (T1w) 3-D data record with a 1 mm isotropic resolution and sec-

**Table 3** Special examination protocols for CT and MRI.

migraine	SAH	RCVS	pituitary apoplexy	dissection	sinus/venous thrombosis	intracranial cysts	infections	cerebral pseudotumors	CSF leak syndrome	trigeminal neuralgia
MRI	CT	CT	MRI	MRI	MRI	MRI	MRI	MRI	MRI	MRI
primary modality	primary modality	often primary modality for SAH	primary modality	primary modality	primary modality	standard program	primary modality for encephalitis	primary modality	primary modality	primary modality
standard protocol	non-enhanced CT	non-enhanced CT	standard protocol	standard protocol	standard program	sag. CISS to assess positional relationship of aqueduct, midbrain	standard program	ax T2w to assess ventricle, orbits, optic nerve pathway and bulbar configuration	head: ax T2w to assess possible hygroma	ax T2w to exclude demyelinating/inflammatory foci
for structural lesion T1w (5 min) post-contrast on at least 2 planes	arterial CTA	arterial CTA	cor T2w to distinguish optic chiasma	ax T1w to assess wall hematoma	CE-MRV		T1w post-contrast on at least 2 planes	cor T2w to assess optic nerve sheath	spine: sag T2w fs to assess epidural fluid cor T2w to assess meningeal diverticula	TOF MRA ax CISS to detect neurovascular compression
			T1w post-contrast cor and sag	CE MRA of craniocervical arteries	CT	CT		CE MRV to assess transverse sinus		
CT	MRI	MRI			CCT	primarily for colloid cysts	CT			<b>Tolosa-Hunt syndrome</b>
only if CI for MRI, or MRI not available	if neg. CCT	primary modality for follow-up	CT	CT	CTV		primary modality to exclude CI prior to LP			MRI
	+ TOF angiography	standard protocol	only if CI for MRI, or MRI not available	CCT CTA						primary modality
		+ TOF angiography								standard program
	DSA									cor and ax T1w post-contrast to detect inflammatory lesions
	If CTA ambiguous for endovascular therapy									

CI = contraindications; LP = lumbar puncture; ax = axial layer orientation; cor = coronal layer orientation; fs = fat-saturated imaging.

ondary reconstruction on the axial and coronal plane. Using 1.5T MR systems, axial T1w images are acquired with a 4 mm slice thickness. These are each followed by an axial T2-weighted (T2w) FLAIR sequence with a 3 mm slice thickness. In this sequence particular attention is paid to the complete signal suppression of the CSF. If this is lacking, it can be an indication of subarachnoid hemorrhage or an inflammatory event. However, it should be noted that CSF signal suppression might be due to additional pathological causes such as acute stroke, leptomeningeal metastasis, administration of supplemental oxygen, or physiological processes such as CSF pulsation [7]. In addition, images taken of all the patients include sagittal T2w images with 3 mm slice thickness to assess the midbrain and brain stem, axial T2\*w

sequences to detect microhemorrhages as well as axial diffusion-weighted images with 5 mm slice thickness and two diffusion factors ( $b = 0/1000 \text{ s/mm}^2$ ).

If there is a related clinical suspicion of a vascular process, arterial angiography employing the TOF (time-of-flight) technique can be used or performed as a contrast-enhanced MR angiography (CE MRA). Routinely, representation of the intracranial vessels uses TOF due to the higher spatial resolution than CE MRA for extracranial vessels [8]. As a rule, time-resolved CE MRA offers a diagnostic advantage only when there is suspicion of intracranial arteriovenous malformation, and therefore has not been established in clinical practice.

Likewise, various techniques are available for MR venography. Due to the brief required examination time and low susceptibility to artifacts, contrast-enhanced MR angiography is regularly performed in clinical practice. The procedure involves injection of 0.1 mmol Gd per kg of body-weight.

Depending upon the medical issue, the examination protocol can be modified just as in CT imaging (◉ Table 3).

Intra-arterial angiography using DSA does not play a role in the primary diagnosis of headache.

### Primary Headache



Primary headache is the cause of 84% of reported cases. Pain itself is the disorder and can have a number of causes. Such headaches include tension headache, cluster headache and migraine. Imaging only rarely yields indicative findings [3–5]. Due to the possibility of incidental findings that are not necessarily causal for headache, but which may have therapeutic consequence, the case for imaging should be strongly made [5].

Structural abnormalities in cases of primary headache are generally found among patients with a migraine and can best be identified using MRI [9, 10]. This particularly concerns women and patients with migraine and accompanying aura. It could thus be shown that patients with migraine exhibit an increased incidence of lesions of the white matter [11]. These concern changes which appear hyperintense in T2w or FLAIR images, and which in T1w images do not exhibit enhancement after contrast agent is administered. Although over the course of time these lesions can increase, the clinical relevance of this change is not understood [11, 12].

In addition, by using functional MR imaging such as resting-state fMRI or VBM (voxel-based morphometry) analysis, it could be shown that, compared to healthy subjects, patients with primary headache can exhibit changes in the connectivity of the brain and regional differences in the distribution of gray matter [13]. Nevertheless, these results require further research and have yet to be integrated into routine clinical practice.

For migraine patients, imaging is primarily indicated when there are changes in the type, duration and frequency of previously identified headaches or if there is a prolonged aura. In this case an MRI should be performed (◉ Table 3). If a non-enhanced examination does not reveal abnormalities, then administration of contrast agent can be omitted. As a rule, imaging of the intracranial vessels is not indicated.

### Thunderclap Headache

The thunderclap headache (TCH) is a sudden headache reaching maximum intensity within a few minutes. The term was originally used to characterize headache among patients with SAH (see Secondary Headache) which occurs in only 11% of patients with TCH, however [14]. There are also a number of additional causes of TCH (◉ Table 4) and primary TCH is a diagnosis by exclusion [4].

### Secondary Headache



In a minority of cases, secondary headache is symptomatic of a different underlying disorder. Following are descriptions of frequent symptom patterns and related changes disclosed by imaging.

#### Subarachnoid Hemorrhage

##### Etiology

In 75–85% of cases [15], non-traumatic SAH is related to a rupture of an aneurysm of vessels supplying the brain. So-called perimesencephalic nonaneurysmal subarachnoid hemorrhage accounts for 15% of cases [15]; the cause of this is abnormal venous drainage into the subarachnoid space. In approx. 5% of cases, SAH has a different cause, such as dissection of the intracranial vertebral artery [16].

##### Symptomology

Thunderclap headache is the cardinal symptom in more than 75% of patients. Here, patients with perimesencephalic SAH frequently exhibit a less fulminant onset of headache. In addition, clinical symptoms can range from minor complaints to coma. Patients with perimesencephalic SAH generally experience a more favorable clinical outcome.

##### Imaging

When there is clinical suspicion, the imaging method of choice (◉ Table 3) is CCT with CTA [15, 16]. In non-enhanced CCT, the subarachnoid space appears hyperdense in cases of SAH, whereas aneurysmal SAH is typically localized in the basal and ambient cisterns (◉ Fig. 1). In comparison, perimesencephalic SAH is localized in the interpeduncular and prepontine cisterns (◉ Fig. 1). Since blood distribution is not pathognomic, a CTA must be performed in cases of perimesencephalic SAH, however. The literature describes CT sensitivity for the detection of SAH as 100% within the first 6 hours after the event; after 24 hours, sensitivity decreases to 93%, and after 4 days, it declines to 50% [17].

FLAIR imaging during MRI is suitable for the detection of SAH. Subarachnoid blood causes the intrathecal space to appear hyperintense in FLAIR [18].

In the primary diagnosis of SAH, CTA has replaced angiography using DSA, which as a rule is only still employed in cases of ambiguous CTA findings or as part of endovascular therapy [15, 16].

If there is clinical suspicion of SAH, and in the absence of evidence of intracranial blood in the CT or MRI, the suspected diagnosis must be excluded or confirmed via lumbar puncture, since in such cases, spinal SAH must be considered [19].

#### Reversible Cerebral Vasoconstriction Syndrome

##### Etiology

Reversible cerebral vasoconstriction syndrome (RCVS) should be considered as part of a differential diagnosis of patients with TCH and unremarkable CSF as well as indication of segmented diameter variations of the intracranial vessels in the CT or MR angiography (◉ Fig. 1). This is a heterogeneous group of disorders associated with reversible segmental vasospasm of the intracranial vessels [20]. The primary differential diagnosis is the isolated angiitis of the CNS, where patients with RCVS typically exhibit reversibil-

ity of the vascular changes within 4 weeks after symptom onset [14, 21].

### Symptomology

Just as in cases of SAH, thunderclap headache is the leading symptom in 95% of patients; patients with RVCS frequently complain of recurring headaches.

### Imaging

Due to the clinical symptom profile, patients with RVCS are frequently examined using CCT or CTA. Unlike SAH, the basal cisterns are usually hypodense. Using CTA, narrow vessels with significant diameter variations can be demonstrated (◉ Fig. 1). With such symptoms, a differential diagnosis should also consider the possibility of SAH with consecutive vasospasms no longer detectable in the CT, so that lumbar puncture should be recommended [19].

Follow-up should entail MR imaging (◉ Table 3), since in such cases diffusion-weighted images can reliably detect infarcts as a possible complication [22]. Due to method-related higher sensitivity required to detect diameter variations, imaging of the intracranial arteries should employ TOF angiography compared to contrast-enhanced angiography (CE MRA) [23]. However, it should be considered that the resolution of MRA is less than that of CTA.

**Table 4** Thunderclap headache differential diagnosis (modified according to [12]).

primary TCH
secondary TCH
subarachnoid hemorrhage
reversible vasoconstriction syndrome
pituitary apoplexy
dissection of craniocervical arteries
sinus or venous thrombosis
colloid cysts of third ventricle
spontaneous CSF suppression syndrome
hypertensive crisis
stroke
primary headache related to coughing/gender

The primary differential diagnosis is isolated angiitis of the CNS, which likewise includes multiple stenosis of the intracranial vessels. However they are distally and eccentrically localized in the arterial lumen, and after contrast is administered, enhancement of the arterial wall can be observed. In addition, transient ischemia and/or hemorrhages can frequently be detected [24]. Typically, patients with RVCS show reversibility of the vascular changes within four weeks.

### Pituitary Apoplexy

#### Etiology

Pituitary apoplexy is a rare but potentially life-threatening disorder which can be caused by ischemic or hemorrhagic necrosis. Very frequently, affected patients have a macroadenoma undergoing drug therapy or are taking oral anticoagulants; however, in the majority of cases, no cause can be ascertained [25].

#### Symptomology

This involves a sudden headache accompanied by progressive vision loss [25].

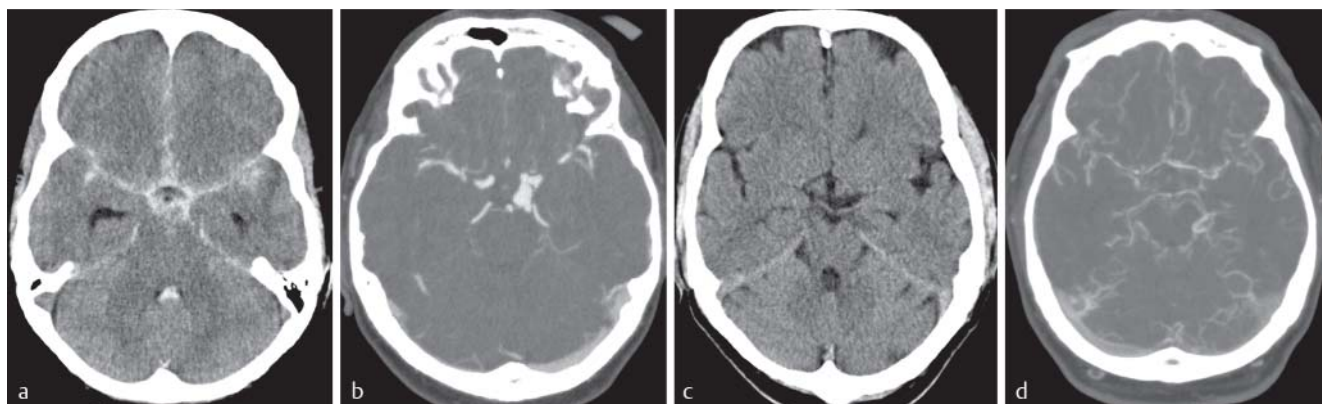
#### Imaging

Neuroimaging should be promptly performed on patients when pituitary apoplexy is suspected (◉ Table 3). MRI is the procedure of choice, whereby the pituitary signal is dependent upon the time point of the examination after the event. Differentiation from optic chiasma is best based upon T2w images. Contrast-enhanced images should be obtained in order to differentiate non-infarcted tissue. However, evidence of hemorrhage can also be obtained via CT (◉ Fig. 2).

### Dissection

#### Etiology

In a dissection there is a tear in the arterial wall and subsequent subintimal hemorrhage. The result can be consecutive narrowing of the true lumen or formation of a pseudoaneurysm if the tear is subadventitial [26, 27]. In the



**Fig. 1** **a** 28-year-old female patient with thunderclap headache with maximum pain within a few minutes; plain CCT with identification of extensive subarachnoid hemorrhage (SAH) in the basal cisterns. **b** Axial 10 mm MIP-reconstruction of the same patient revealed a saccular aneurysm of the posterior communicating artery as the underlying cause of the hemor-

rhage. **c** 56-year old patient with thunderclap headache two days before admission and now dull headache. Plain cranial CT without evidence of SAH. **d** CT angiography of the same patient demonstrates extensive vasospasm of the intracranial vessels. CSF analysis was without evidence of SAH supporting the diagnosis of reversible cerebral vasoconstriction syndrome.

majority of cases, dissection is spontaneous; additional risk factors include trauma or manipulation of the cervical spine and pre-existing connective tissue disease [26].

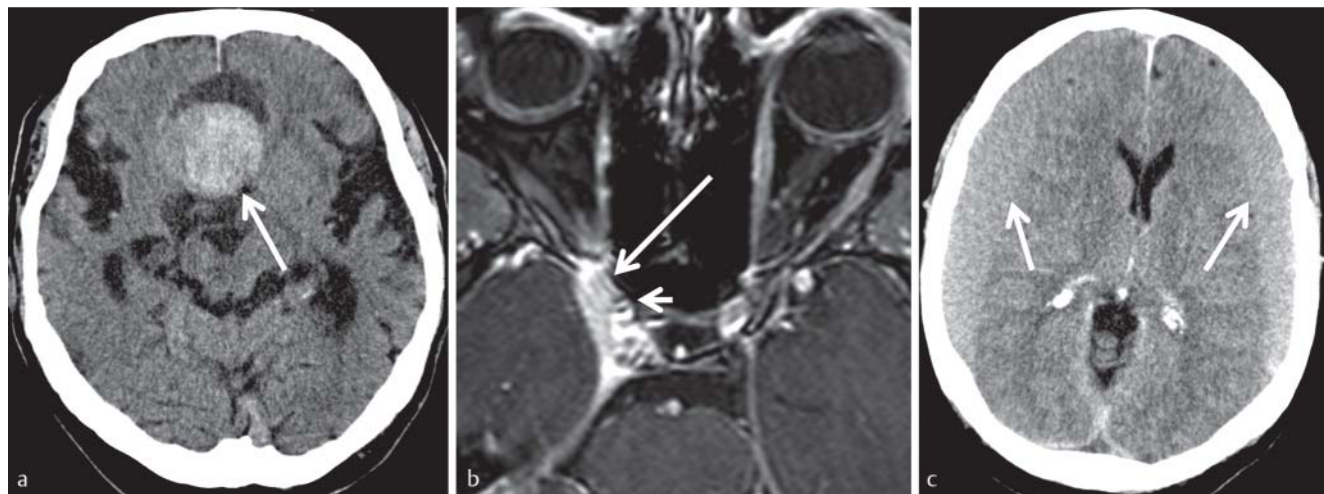
### Symptomology

Headaches are the most common symptom of patients with dissection and as a rule they appear prior to focal-neurological symptoms [26]. They occur more frequently during dissection of the internal carotid artery compared to lesion of the vertebral artery (◉ Fig. 3) Although headache generally exhibits gradual increase, up to 20% of affected patients present with TCH. Typically pain is unilateral on the same side as the affected vessel and radiates from the neck into the head. In dissection of the vertebral artery, pain is prima-

rily localized occipitally; in carotid dissection, pain radiates into the jaw, to the temple as well as around the eyes [26].

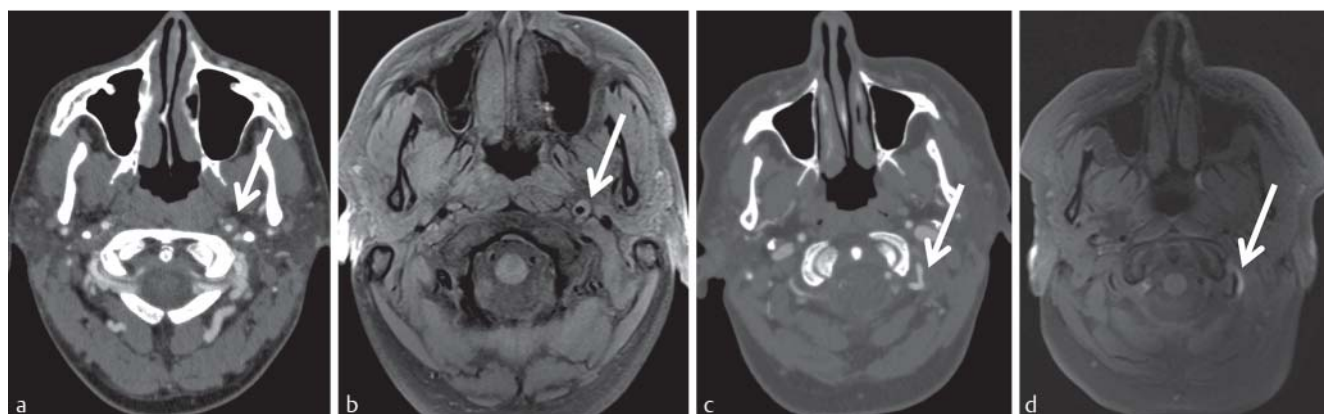
### Imaging

MRI is the preferred imaging modality (◉ Table 3). Non-enhanced T1-weighted images with fat saturation should be used to detect mural hematoma as confirmation of dissection, since after the second or third day, the mural hematoma appears hyperintense compared to the perfused lumen due to methemoglobin formation [26, 27]. In the acute phase, mural hematoma appears isointense. Vessel imaging can be performed using either TOF MRA or CE MRA; the latter is preferred to display craniocervical vessels [26, 27]. As



**Fig. 2** Rare causes of secondary headache. **a** Plain axial cranial CT; 64-year-old patient with known pituitary macroadenoma; TCH and sudden loss of vision. The intra- and suprasellar adenoma (arrow) is hyperdense indicating hemorrhagic pituitary apoplexy. **b** Contrast-enhanced axial T1-weighted sequence with fat suppression; 48-year-old female patient with right-sided orbital pain and paralysis of the third and fourth cranial nerve. Strongly enhancing granulomatous tissue within the right cavernous sinus, superior

orbital fissure and dura of the middle cranial fossa (long arrow) and concomitant narrowing of the right internal carotid artery (short arrow) as diagnostic landmark of Tolosa-Hunt-Syndrome. **c** Plain axial cranial CT three weeks after a mild traumatic brain injury; 74-year-old patient with oral anticoagulation treatment and two weeks history of headache. Subacute subdural hematoma above both hemispheres; the cortical surface (arrow) is displaced from the inner aspect of the cranium.



**Fig. 3** **a** 27-year-old female patient with numb pain in the neck radiating into the temporal region after physical exercises. CT angiography demonstrates circular mural hematoma of the internal carotid artery (ICA). **b** Plain axial T1-weighted image with fat saturation of the same patient demonstrating the hyperintense mural hematoma of the ICA (arrow). The hematoma can be very nicely separated from the hypointense flow void causing

discrete narrowing of the lumen. **c** 37-year-old female patient with new onset of headache after playing with her toddler. CT angiography demonstrates discrete luminal irregularities of the dominant left vertebral artery within the horizontal part of atlas loop. **d** Plain axial T1-weighted image with fat saturation of the same patient demonstrates the hyperintense mural hematoma indicating arterial dissection.

a rule, the arterial lumen tapers conically in a vascular occlusion caused by dissection (so-called “string sign”). Alternatively, CT angiography can be performed (◉ Fig. 3). In this case, the intramural hematoma appears as localized wall thickening with accompanying variations in diameter. In clinical practice, Doppler ultrasound is successfully used as a screening examination and for follow-up. However, ultrasound findings frequently require follow-up diagnostic imaging, since only hemodynamic findings can be obtained and the etiology of the flow obstruction observed via Doppler sonography remains unclear [27].

### Cerebral Venous Sinus Thrombosis

#### Etiology

A number of risk factors exist for the formation of cerebral venous or sinus thrombosis [28]. In particular, pregnant women and those in the postpartum phase have an increased risk [29]. No causal factor can be identified in 20–25 % of cases.

#### Symptomology

The clinical symptom pattern is variable and can range from non-specific complaints to focal-neurological deficits through to coma. The guiding symptom, however, is headache [30]. Classically, headache has subacute onset and increases during gradual progression.

#### Imaging

Whereas in non-enhanced CCT, hyperdense interior veins have a high positive predictive value as an indirect reference point for thrombosis [31], the value of the already hyperdense sinus in non-enhanced CCT is debatable (◉ Fig. 4); this is also true of the lack of flow-related signal loss (so-called flow void phenomenon) in T2-weighted images. In MRI, thrombus in the gradient echo appears as tubular signal cancellation due to blood degradation products. Further, susceptibility-weighted sequences, e.g. SWI (susceptibility-weighted imaging) or SWAN (susceptibility-weighted angiography) should be suitable to definitively identify thrombosis of cortical veins [32]. Nevertheless, vascular diagnosis should always follow in cases of doubt. In addition to CT angiography, this can also be in the form of MR angiography; however, contrast-enhanced MR venography (◉ Table 3)

is preferable to other techniques [32]. In both examination modalities, thrombus appears as a contrast defect (◉ Fig. 4).

### Intracranial Cysts

#### Colloid cysts

##### Etiology

Colloid cysts are protein-rich cysts on the roof of the third ventricle (◉ Fig. 5) emanating from the endoderm [33].

##### Symptomology

Colloid cysts can be an incidental finding in cranial imaging or can manifest themselves via headache. The cause can be assumed to be intermittent blockage of the interventricular foramen. Since colloid cysts can exhibit rapid growth progression, they can result in the formation of life-threatening obstructive hydrocephalus [33]. Therefore neurosurgical therapy should be suggested to patients with colloid cysts.

##### Imaging

In more than 90 % of cases, colloid cysts are found in the direct vicinity of the interventricular foramen [33] and in non-enhanced CCT appear as hyperdense round lesions (◉ Table 3). Cyst signal can be variable in an MRI, and depends on the viscosity and protein makeup of the cyst contents [34]. As a rule they appear hypointense in T2w and hyperintense in T1w. Since they can also appear to be CSF-isointense in an MRI, these cysts are frequently better distinguished using CCT.

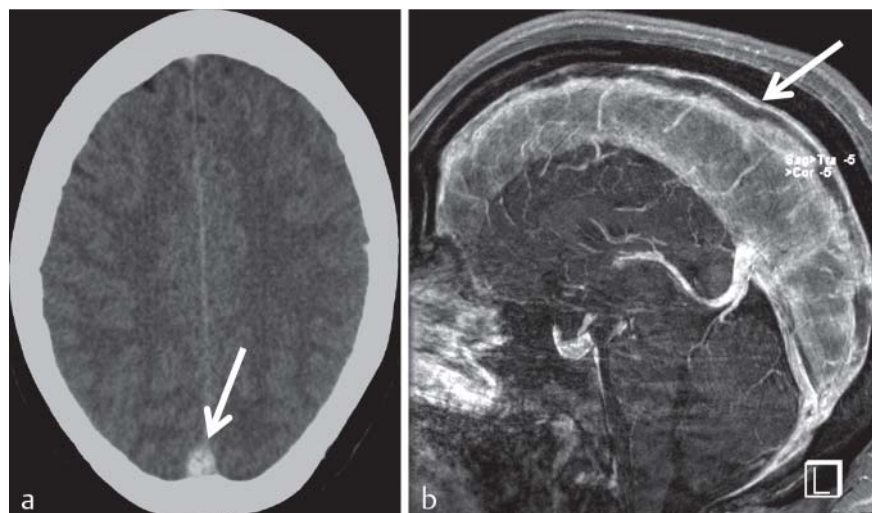
### Pineal Cysts

#### Etiology

Pineal cysts are non-neoplastic cysts in the pineal canal.

#### Symptomology

Generally these are asymptomatic findings (◉ Fig. 5) during imaging of the neurocranium, found in 2.5–10 % of all patients [33]. Because of this, some authors consider them only as a diagnosis of exclusion as a cause of headache [35]. In addition to a disturbance of melanin metabolism, another possible cause under discussion is intermittent compression of the tectal plate and aqueduct caused by very large cysts. Infrequently very large cysts can result in com-



**Fig. 4** a 27-year-old female patient 2 weeks postpartum. Slowly progressive headache which is not responding to therapy. Plain cranial CT demonstrates hyperdense superior sagittal sinus (arrow). b Sagittal MIP reconstruction (3 mm slice thickness) of a contrast-enhanced MR venography of the same patient demonstrating extensive thrombosis of the superior sagittal sinus (arrow).



**Fig. 5** **a** 23-year old patient with intermittent holocephalic headache. The colloid cyst in typical location at the roof of the third ventricle (arrow) appears hyperdense on plain cranial CT. **b** Coronal T2-weighted CISS sequence demonstrates the close anatomical relationship of the colloid cyst (arrow)

to both foramen of Monro (dotted arrow). The cyst appears hypointense due the protein-rich content. **c** 25-year-old female fMRI volunteer subject with intermittent morning headache. Sagittal T2-weighted CISS image demonstrates a large pineal gland cyst extending just in front of the aqueduct.

pression of the tectal plate accompanied by development of Parinaud's Syndrome.

Since the majority of cysts are asymptomatic and consistent in size or can even exhibit size reduction, follow-up is recommended; neurosurgical therapy is recommended only for cysts > 10 – 14 mm [31].

### Imaging

Imaging can employ either CT or MRI (Table 3); MRI should always be used for follow-up. In CT, the cyst is hypodense; in T1w and T2w images it likewise indicates a hypointense signal. Cyst contents in FLAIR images appear hyperintense. Sagittal high-resolution T2w sequences (e.g. using CISS) should be acquired to accurately determine positional relationship of the cyst to the tectum and aqueduct (Fig. 5).

Administration of contrast agent is not required for diagnosis. In up to 40% of cases, in both CT and MRI a linear marginal enrichment can be detected [33].

### Intracranial infections

#### Etiology

In cases of cerebral infection, a distinction can be made between inflammation of the cerebral parenchyma (encephalitis) and cerebral membranes (meningitis). In the majority of cases, infections are either viral or bacterial in nature [36], and the path of infection is either hematogenic (e.g. during sepsis), through neighboring tissue (as a result of otitis media or sinusitis), directly or iatrogenically.

#### Symptomology

The guiding symptom of intracranial infection is headache and fever. Depending on the site and extent of the infection, additional neurological symptoms – including death – may occur.

Particular attention, however, should be paid to immune system-compromised and HIV-positive patients [37]. Intracranial pathology is found in cases of new-onset headache in up to 82% of these patients [38, 39] (Fig. 6). Therefore, cer-

ebal imaging should be performed after new-onset headache, or if existing symptoms change in type or intensity.

### Imaging

Meningitis is a clinical diagnosis supported by testing cerebrospinal fluid. The use of imaging, therefore, is to exclude contraindications for a lumbar puncture [40]. Performing non-enhanced CCT is sufficient here. In rare cases, moreover, complications or entry points for an inflammatory process can be identified (Fig. 6).

If encephalitis is suspected, an MRI should be performed (Table 3). Inflammatory changes appear as hyperintense signal alterations in FLAIR images, and in diffusion-weighted images can be distinguished earlier as hyperintense signal alteration. Administration of contrast agent is required, and contrast-enhanced images should be acquired on at least two planes.

### Cerebral Pseudotumors

#### Etiology

CSF circulatory dysfunction can likewise be a source of headache. A particular form of this is the cerebral pseudotumor, also characterized as idiopathic intracranial hypertension [41]. This is understood to be an increase in intracranial pressure without evidence of an intracranial mass, hydrocephalus or edema.

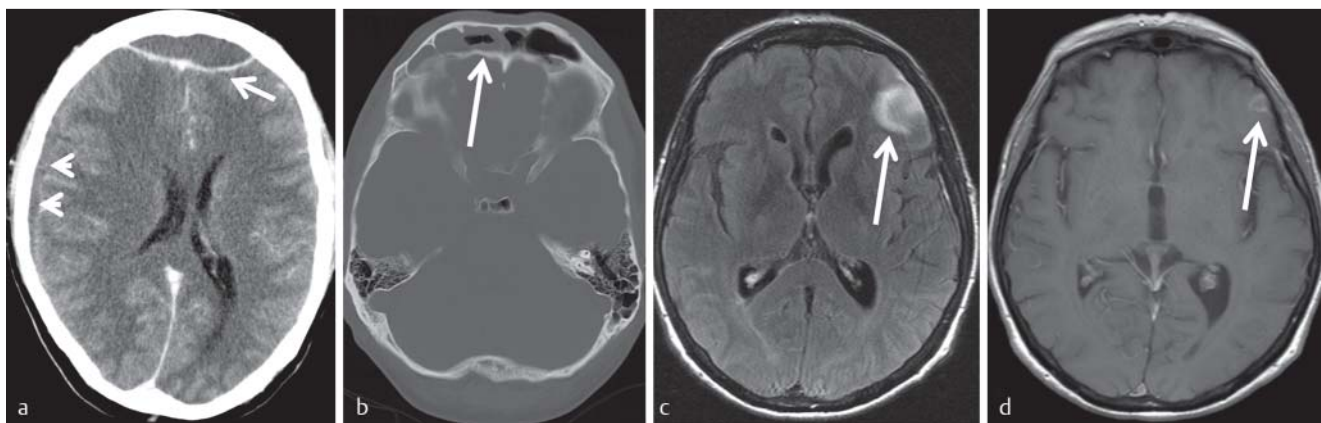
#### Symptomology

Typical symptoms are headache and loss of visual acuity. Additional common symptoms include tinnitus as well as cranial nerve paresis; an ophthalmological examination may reveal papilledema. Typically young, overweight women and older slender men are affected, whereas among women, headache is in the forefront, and vision loss predominates among men [41].

### Imaging

MRI is the examination modality of choice. Typical imaging findings are flattening of the papilla in axial T2w images as





**Fig. 6** **a** 43-year-old patient with new onset of headache and fever. CSF analysis demonstrated bacterial meningitis. Neurological worsening two days after onset of symptoms. Contrast-enhanced CT demonstrates frontal epidural abscess (arrow) and subdural empyema (arrow heads). **b** 29-year-old female patient with bacterial meningitis. Plain cranial CT in bone window level setting demonstrates extensive frontal sinusitis as possible un-

derlying cause. **c** 37-year-old HIV-positive patient (T4 cell count <200/ $\mu$ l) with new onset of headache. Axial T2-weighted FLAIR image demonstrates left frontal cortical and subcortical lesion. **d** Contrast-enhanced T1-weighted image of same patient demonstrates linear cortical enhancement. Histologically-proven cerebral toxoplasmosis.



**Fig. 7** **a** 32-year-old obese female patient (BMI 32 kg/m<sup>2</sup>) with headache and progressive loss of vision. Coronal T2-weighted sequence demonstrates dilated optical nerve sheaths on both sides (arrow) as indirect sign of pseudotumor cerebri. **b** Sagittal T2-weighted image of the same patient

demonstrates “empty sella”-phenomenon. **c** Venous TOF angiography of the same patient delineating stenosis of the lateral transverse sinus (arrow). **d** The stenosis of the transverse sinus is reversible (arrow) after spinal tap test highly suggestive for the diagnosis pseudotumor cerebri.

well as a gyrate optic nerve. Coronal images reveal an enhancement of the optic nerve sheath; this phenomenon can be easily assessed using T2w images with fat saturation (Table 3). Additionally, the pituitary is flattened (so-called empty sella syndrome). Venous angiography frequently reveals stenosis in the transverse sinus [42]. Due to shorter examination time and reduced susceptibility to artifacts, contrast-enhanced MR venography is preferable to TOF venography (Table 2). Morphological changes in the image can be reversed after lumbar puncture, a diagnostic indicator (Fig. 7)

### CSF Leak Syndrome

#### Etiology

In contrast, the CSF leak syndrome is a relatively recent diagnosis that is underdetected. The pathophysiological basis is that the loss of CSF results in caudal displacement of the brain with subsequent strain on the richly innervated dura, causing headache [43]. As it progresses, pain symptoms become chronic. A distinction can be made between a primary

idiopathic form and a secondary form. Causes of the secondary form can be meningeal diverticula generally occurring along the nerve root, or dural defects. The latter can be iatrogenic or caused by degenerative osteophytes. As many as 2/3 of patients additionally exhibit connective tissue disease.

#### Symptomology

The disease pattern is marked by orthostatic headache normally occurring after 15 minutes in the standing position and improving in the reclining position. Women are more frequently affected than men, and peak age is about 40 [43]

#### Imaging

Diagnosis must be performed using MRI (Table 3) [43]. Intracranial subdural hygromas and sinus dilation are typically found, and after administration of contrast agent, the meninges exhibit significant contrast accumulation (Fig. 8). Spinal epidural fluid deposits can be detected. These are easily detected in sagittal T2-weighted images with fat satura-



**Fig. 8** **a** 43-year-old female dancer with progressive headache in upright position. Axial T2-weighted image demonstrates bilateral frontal hygroma (arrow). **b** Contrast enhanced coronal T1-weighted image of the same patient demonstrating thickened and strongly enhanced meninges and tentorium. **c** Sagittal T2-weighted image with fat suppression of the spine de-

monstrates epidural effusion (arrow) as indirect spinal sign of intracranial hypotension. **d** Coronal T2-weighted MR-myelogram delineates multiple spinal nerve root diverticula as possible cause of the CSF leakage. Complaints resolved after epidural blood patch at the level of the largest diverticulum.

tion, since in these sequences, a distinction can be reliably made between epidural fluid and fat. Coronal T2-weighted images are suitable to identify nerve root sleeve cysts as a possible cause of CSF loss (► Fig. 8). In up to 20% of cases, the MRI is unremarkable, however. In addition to diagnosis, radiology also offers the possibility of interventional radiological therapy using an epidural blood patch [44].

### Sinusitis Etiology

There are a number of triggering factors for sinusitis. With respect to their temporal progression, a distinction can be made between an acute (less than 8 weeks duration or fewer than 4 episodes per year) and a chronic form (more than 8 weeks duration or more than 4 episodes per year) [45].

### Symptomology

Sinus headaches as a rule generally have a dull character and mainly occur with involvement of the sphenoid sinus (► Fig. 9).

### Imaging

Acute sinusitis is generally a clinical diagnosis and does not require imaging. However, imaging should be performed in cases of chronic sinusitis to assess the anatomy with respect to surgical therapy [46], in cases of immune system-deficient patients, new onset focal neurological deficit, or if complications are suspected [45, 47].

Due to their limited sensitivity and specificity, conventional X-ray images are obsolete for the diagnosis of sinusitis; instead a low-dose CT should be performed [47]. Sinusitis is accompanied by swelling of the mucosa, which appears in CT as iso- to hypodense; in MRI it appears hyperintense in T2w images, and isointense to the musculature in T1w images. Chronic cases may result in sclerosis of the sinus walls

which is easily detectable in CT, and which can reduce the volume of the affected sinus.

If there is suspicion of intracranial or orbital complications (► Fig. 9), MRI is superior to CT as a diagnostic modality. These can be well-differentiated in coronal T2-weighted images with fat saturation, as well as in fat-saturated T1-weighted images after administration of contrast agent [47].

### Trigeminal Neuralgia Etiology

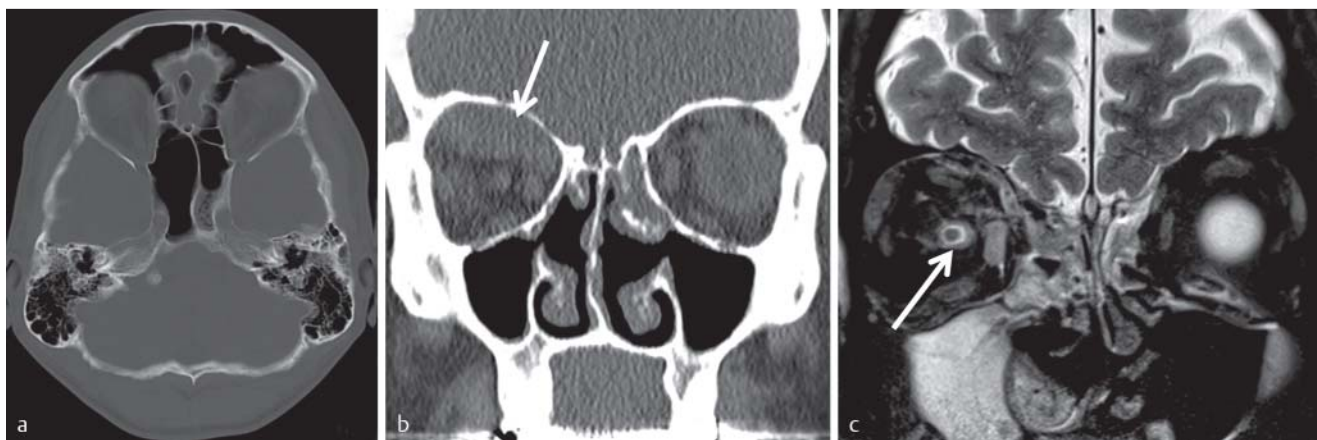
Trigeminal neuralgia (TN) is a neurovascular compression syndrome; according to the IHS classification of headache, it belongs to the group of “cranial neuralgias, central and primary facial pain and other headaches.” The cause is considered to be compression of the nerve in its root entry zone by an artery [48], generally the superior cerebellar artery or the anterior inferior cerebellar artery (► Fig. 10).

### Symptomology

Typically patients complain of intense pain lasting but a few seconds in the region of the second or third trigeminal branch; in only 5% of cases is the pain in the territory of the ophthalmic nerve (first branch). Pain can occur spontaneously or can be provoked by contact with trigger points [48].

### Imaging

If TN is suspected, diagnosis should be performed using MRI (► Table 3). The success of surgical therapy correlates with the preoperative verification of neurovascular compression. The examination protocol to substantiate this should include axial high-resolution T2-weighted images (e.g. CISS sequence) in addition to arterial TOF angiography. It should be noted, however, that among healthy subjects, vessel-nerve contact can likewise be observed; therefore there



**Fig. 9** **a** 33-year-old patient with new onset of holocephalic headache. Plain cranial CT in bone window level setting demonstrating opacification of the left sphenoid sinus indicating acute sinusitis. **b** 43-year-old patient with chronic sinusitis and recent exacerbation. Two-day history of pain in the right eye and diplopia. Coronal CT reconstruction demonstrates a subperiosteal abscess at the roof of the orbit. **c** 47-year-old female patient with chronic sinusitis. Acute exacerbation with new onset of headache and pain in the right eye. Coronal T2-weighted images demonstrate subtle inflammatory changes around the optic nerve and extra- and intraconal fat tissue.

istial abscess at the roof of the orbit. **c** 47-year-old female patient with chronic sinusitis. Acute exacerbation with new onset of headache and pain in the right eye. Coronal T2-weighted images demonstrate subtle inflammatory changes around the optic nerve and extra- and intraconal fat tissue.

must always be a correlation between image findings and clinical symptoms [48]. A differential diagnosis of multiple sclerosis should be considered, particularly in cases of young women with new-onset TN or new-onset atypical facial pain (◉ Fig. 10).

### Tolosa-Hunt Syndrome

#### Etiology

Together with trigeminal neuralgia, Tolosa-Hunt syndrome (THS) is counted among the group of cranial neuralgias. The disorder is characterized by inflammatory granulomatous changes of unknown etiology in the cavernous sinus and superior orbital fissure which can extend into the orbit and continue intracranially [49].

#### Symptomology

THS is an episodically recurring pain in the orbit, accompanied by paresis of the oculomotor nerve and trochlear nerve and/or the abducens nerve [49].

#### Imaging

MRI is the modality of choice. In addition to prompt improvement of symptoms within 72 hours after administration of cortisone, detection of inflammatory changes (◉ Fig. 2) is one of the essential diagnostic criteria [49]. To demonstrate granulomatous changes, contrast-enhanced images with fat saturation should be acquired using axial and coronal layer orientation (◉ Table 3).

### Post-traumatic Headache

#### Etiology

Headache is one of the most common symptoms after traumatic brain injury (TBI), and the most frequent form of secondary headache [4, 50]. The exact pathophysiological mechanism is not understood in the majority of cases. Paradoxically, mild TBI is more frequently associated with chronic post-traumatic headache than severe TBI [50].

### Symptomology

According to guidelines, post-traumatic headache appears within seven days after the trauma and is considered acute if it lasts less than 3 months [4]. Post-traumatic headache does not exhibit specific characteristics, and current studies have shown that in the majority of cases, post-traumatic headaches have a migrainoid character [50]. In addition they can be associated with impairment of cognitive function, leading to a significant reduction in the quality of life.

#### Imaging

In rare cases there is a correlation in morphological imaging [50]; therefore, initial imaging can use non-enhanced CCT. In the differential diagnosis, a subdural hematoma should be considered, particular in older patients undergoing anticoagulant therapy (◉ Fig. 2). CCT is the imaging modality of choice. Hematoma appears hyperdense compared to the adjoining cerebral parenchyma. However, with progression it reduces in density, so that the hematoma can appear isodense or hypodense with respect to the brain. Therefore it is important to note whether the cortical relief touches the calvaria, which is otherwise an indication of subdural hematoma.

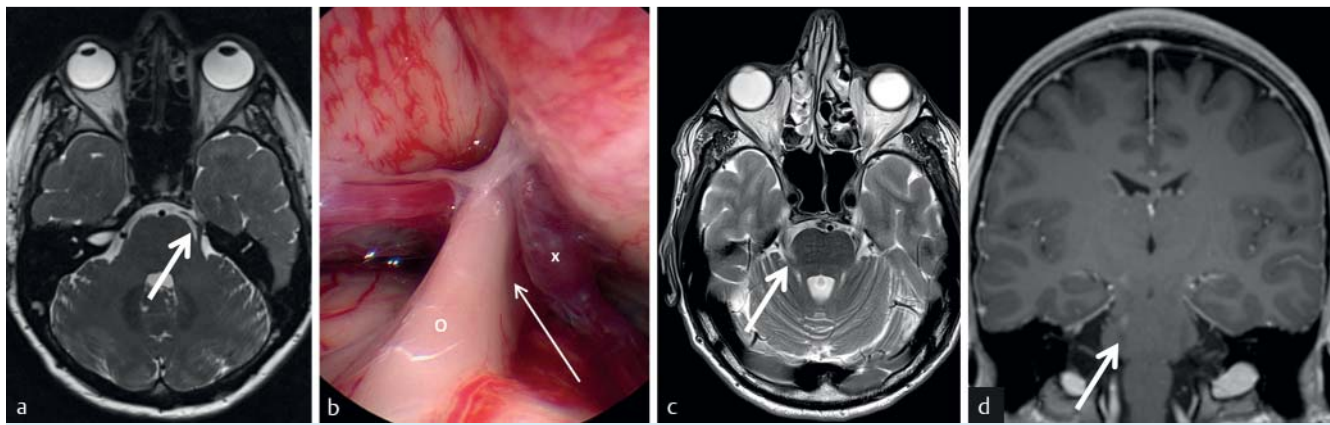
### Summary



The majority of patients with headaches suffer from primary headache for which imaging only rarely yields indicative findings.

In contrast there are a number of less common secondary headaches that are an expression of an underlying disorder requiring additional clarification.

Even in cases in which insufficient evidence-based data is available, at the least a non-enhanced CCT to exclude intracranial pathology should be performed on patients above 50 years of age with new-onset or altered headache. Further, imaging is indicated if previously identified headache changes in character, if accompanying focal-neurological deficit or systemic secondary reactions are present, as well



**Fig. 10** **a** 57-year-old female patient with 7-month history of recurrent pain in the territory of the second trigeminal branch. Axial T2-weighted CISS image demonstrates neurovascular compression of the root entry zone of the left trigeminal nerve by a loop of the left superior cerebellar artery. **b** Intraoperative photograph of the same patient. The nerve (o) is flattened (arrow) by the pulsatile compression of the vascular loop (x).

**c** 24-year-old female patient with 2-week history pain in the territory of the first trigeminal branch. Axial T2-weighted image demonstrates demyelinating lesion at the root exit zone. **d** Coronal reconstructed contrast-enhanced T1-weighted MPR image demonstrates enhancement of the lesion indicating active inflammation.

as in cases of immune system-compromised patients with new-onset headache. Selection of the imaging modality is based on the suspected underlying disorder and the general clinical situation of the patient.

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