Chapter 105

Acute Posttraumatic Headaches

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International Headache Society (IHS) code and diagnosis:

5.1 Acute posttraumatic headache
  5.1.1 Attributed to moderate or severe head injury
  5.1.2 Attributed to mild head injury
5.3 Acute headache attributed to whiplash injury
5.5 Headache attributed to traumatic intracranial hematoma
  5.5.1 Attributed to epidural hematoma
  5.5.2 Attributed to subdural hematoma
5.6.1 Acute headache attributed to other head and/or neck trauma
5.7.1 Acute postcraniotomy headache

World Health Organization (WHO) codes and diagnoses:

G44.880 Acute posttraumatic headache
  G44.880 Attributed to moderate or severe head injury
  G44.880 Attributed to mild head injury
G44.841 Acute headache attributed to whiplash injury
G44.88 Headache attributed to traumatic intracranial hematoma
  G44.88 Attributed to epidural hematoma
  G44.88 Attributed to subdural hematoma
G44.88 Acute headache attributed to other head and/or neck trauma
G44.880 Acute postcraniotomy headache

Short description: New headache occuring for the first time in close temporal relation to a known trauma to the head, neck, or brain frequently embedded as a prominent complaint in a posttraumatic syndrome and recovering within 3 months. Trauma related worsening of a preexisting headache is possible. Most frequently the acute posttraumatic headache has the characteristics of a tension-type headache. In some cases the trauma triggers a migraine; in rare cases it is followed by a clusterlike syndrome.

ACUTE POSTTRAUMATIC HEADACHE

Headache is the cardinal symptom of the posttraumatic syndrome that follows head trauma (HT) and cervical spine whiplash injury (WI). Knowledge of the different types of posttraumatic headache (PTH) (a) guides in the choice of headache-specific treatment; (b) helps in avoiding chronification of the headache and hastening remission; and ultimately, (c) provides the basis for improving the clinical outcome of the patient (57,58). This chapter provides an overview of the definition, epidemiology, classification, pathogenesis, clinical picture, diagnosis, course, and treatment of acute PTH. Chronic PTH (80) is discussed in a subsequent chapter of this book.

The International Classification for Headache Disorders-II (ICHD-II) requires that PTH occur in close temporal relationship with HT or WI of varying severity and states that late-acquired headaches (2,16), occurring later than 1 week up to 3 months after a traumatic event, are not sufficiently validated (41).

EPIDEMIOLOGY

PTH is not uniform in its epidemiology or clinical presentation. Headaches characteristic of tension-type headache, migraine, cluster headache, cervicogenic headache, headache of intracranial hemorrhage, or elevated intracranial pressure headache all have been described following HT.

The frequency of the different types of PTH is as follows: tension-type headache, 85% (27,28,34,36); postwhiplash (unilateral) cervicogenic headache, 8% at 6 weeks, 4% at 6 months, and 3% at 1 year (female:male ratio of 3:2) (24,25); and migrainelike headache, 2.5% (more common in children and teenagers) (33,97,103). The lifetime prevalence of an HT-associated migraine is 1.4% (86). The exact frequency of posttraumatic clusterlike headache is not

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known but this type of headache remains rare (82,102). Basilar migrainelike PTH (36,43) and PTH associated with sexual activity (21) have been reported anecdotally. Analgesic overuse is described in 19% of all cases of chronic PTH and in 25% of chronic posttraumatic tension-type headache (36).

The incidence of head trauma is180 to 220 per 100,000 in North America and 350 per 100,000 in Europe (30), which is comparable to that of cerebrovascular ischemia, 10 times the incidence of Parkinson's disease (20 of 100,000), and 500 times that of myasthenia gravis (0.4 of 100,000) (55,56).

Approximately 2 million HTs occur per year in the United States, of which 80% are mild, 10% moderate, and 10% severe (46,55,56).

The frequency of PTH in HT varies between 31% and 90% (15, 20,59,68). This implies that the relative incidence of acute PTH is up to 200 per 100,000 in the United States and 315 per 100,000 in Europe (specifically, Germany). The absolute rates of newly diagnosed PTH per year are estimated as 1,800,000 in the United States and 270,000 in Germany.

The severity of the head injury is inversely proportional to the occurrence of acute PTH (72% in mild head injury, 33% in severe head injury [106]) and to chronic PTH (18). There is also an inverse relation between the duration of loss of consciousness (14) or the duration of posttraumatic amnesia (16) and the incidence of PTH.

Acceleration injuries of the cervical spine (i.e., WI) are the most common mechanisms of cervical trauma in traffic accidents (94). In one study, 88% of patients complained of headache after a mild WI (Quebec Task Force, QTF Grade II) without bony injuries or accompanying neurologic deficits (47,50,52). Other studies have indicated incidence rates of 40 to 97% (24,44,52). These wide ranges may be related partially to differences in registers used for analysis (insurance, police, hospital) and are lower in a police register (72) or an insurance register (17).

Exact figures for the incidence of PTH following moderately severe WI (with neurologic deficits; Quebec Task Force, Grade III [99]) or severe injury of the cervical spine (with osseous lesions and neurologic deficits; Quebec Task Force, Grade IV) are not available because of inadequacies in reporting the severity of the injury and difference in patient populations.

PATHOPHYSIOLOGY

The exact pathophysiology of acute PTH is unknown. Different mechanisms of pain are likely to play a role in acute PTH because the clinical presentation could mimic migraine, tension-type headache, or other forms of primary headaches. The mechanism(s) of primary headache may help us understand the corresponding subtype of acute PTH.

Acute posttraumatic cervicogenic headache (following WI or HT combined with neck sprain) is likely the result of the multisegmental pain impulses generated from nociceptive afferents in stretched muscles, ligaments, and intervertebral disks as well as from sympathetic nerve fibers of the arterial vessels entering the cervical spinal cord via C-fibers of the C2–C5 dorsal rami (74,75). The convergence between these upper cervical roots and the spinal nucleus of the trigeminal nerve provides a pathway for referral of posttraumatic neck pain to the frontal region (and vice versa [11,53]). Rarely, direct traumatic compression of C2 fibers in the lateral atlantoaxial joint provides another explanation for cervicogenic headache as demonstrated postmortem (9). Sympathetic vertebral nerve irritation (6) and ischemia of the vertebral artery (4,5) are other postulated, although debated (10), causes of posttraumatic cervicogenic headache.

An alteration of the antinociceptive inhibitory temporalis reflex in acute PTH of the tension type following WI suggests a transient dysfunction in central pain processing with an impairment of the serotonergic descending inhibitory pain system (48). Similar reflex abnormalities also have been described for the idiopathic tension-type headache (95). Acute PTH of tension type is accompanied by a varying degree of increase in general pain sensitivity (70,100). Subsequent chronification of the acute posttraumatic pain syndrome may be related to a windup phenomenon of central pain sensitization. The impact of the accompanying vegetative disturbances, subjective impediment, or disturbances in mood and well-being on pain intensity and duration is not fully elucidated (46,52).

In head injury, a variety of posttraumatic neurochemical, neurohumoral, and neuroelectrical changes are described (107). They include elevated extracellular K^+ and intracellular Na^+, Ca^+, and Cl^-, reduced intracellular and total brain Mg, influx of extracellular Ca^{2+} (in axolemmas), accumulation of platelet-derived 5-hydroxytryptamine in the central nervous system, increased release of excitatory amino acids (e.g., glutamate), cortical spreading depression, increased levels of endogenous opioids, and increased nitric oxide activity (73,107). Some authors consider this altered neurochemical environment to be responsible for the acute manifestation of PTH or aura, and suggest a mechanistic cascade for the syndrome that is similar to primary migraine (73,107,108).

Animal models and functional brain imaging studies in HT and WI show various structural, blood flow, and metabolic changes, which do not necessarily translate into mechanisms of acute PTH. Factors likely to be involved in the pathophysiology include:

1. Referred pain from nociceptive input caused by lesions of musculoskeletal, discoligamentous, and other soft-tissue structures (including vessels, perivessel sheets, and nerves)

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2. Activation of meningeal nociceptive afferents due to traumatic epidural, subdural, and subarachnoidal bleeding
3. Stretching of pain-sensitive intracranial structure from increased intracranial pressure
4. Intracranial hypotension (79,81)
5. Activation of the trigeminovascular system by posttraumatic sinus venous thrombosis

CLINICAL FEATURES

The IHS diagnostic criteria (5.1.1) for acute PTH attributed to moderate or severe head injury (Headache Classification Committee, 2004) (40) are as follows:

A. Headache, no typical characteristics known, fulfilling criteria C and D
B. Head trauma with at least one of the following:
  1. Loss of consciousness for >30 minutes
  2. Glasgow Coma Scale (GCS) <13
  3. Posttraumatic amnesia for >48 hours
  4. Imaging demonstration of a traumatic brain lesion (cerebral hematoma, intracerebral and/or subarachnoid hemorrhage, brain contusion and/or skull fracture)
C. Headache develops within 7 days after head trauma or after regaining consciousness following head trauma.
D. One or other of the following:
  1. Headache resolves within 3 months after head trauma.
  2. Headache persists but 3 months have not yet passed since head trauma.

The IHS diagnostic criteria (5.1.2) for acute PTH attributed to mild head injury (1) (Headache Classification Committee, 2004) (40) are as follows:

A. Headache, no typical characteristics known, fulfilling criteria C and D
B. Head trauma with all the following:
  1. Either no loss of consciousness or loss of consciousness of <30 minutes' duration
  2. Glasgow Coma Scale (GCS) = 13
  3. Symptoms and/or signs diagnostic of concussion
C. Headache develops within 7 days after head trauma.
D. One or other of the following:
  1. Headache resolves within 3 months after head trauma.
  2. Headache persists but 3 months have not yet passed since head trauma.

The IHS diagnostic criteria (5.6.1) for acute headache attributed to other head and/or neck trauma (Headache Classification Committee, 2004) (40) are as follows:

A. PTH, no typical characteristics known, fulfilling criteria C and D
B. Evidence of head and/or neck trauma of a type not described above
C. Headache develops in close temporal relation to, and/or other evidence exists to establish a causal relationship with, the head and/or neck trauma.
D. One or other of the following:
  1. Headache resolves within 3 months after the head and/or neck trauma.
  2. Headache persists but 3 months have not yet passed since the head and/or neck trauma.

The IHS diagnostic criteria (5.3) for acute headache attributed to whiplash injury (Headache Classification Committee, 2004) (40) are described elsewhere in the book.

Headache (with or without accompanying neck pain) is the cardinal symptom of the posttraumatic syndrome (108). Direct HT and WI cause a similar syndrome. In the acute phase, it is characterized by vegetative symptoms (dizziness, nausea, vomiting, orthostatic dysregulation, and thermodysregulation), a neurasthenic depressive syndrome (subjectively reduced cognitive performance sometimes with overt neuropsychologic deficits, mood alteration, nervousness, and irritability), and a sensory syndrome characterized by excessive sensitivity to light and noise (49).

Following WI, the PTH is associated with a feeling of heaviness of the head in almost half of the patients. It usually follows a complaint-free interval of a few hours to 1 day (52). The headache is mainly occipital, dull pressing, or dragging and reaches maximum intensity in the evening (47,49,50). Headaches of the tension type following head or neck injury (i.e., WI) have similar features (36,50,52). Some patients with the posttraumatic stress disorder (PTSD) report headache shortly after the trauma (a mild or trivial head injury in some cases) but the DSM-IV diagnostic criteria for PTSD do not include headache as a prominent symptom (3). Furthermore, acute PTH and PTSD are distinct in temporal profile despite the overlap in vegetative symptomatology. Acute PTH usually resolves within 3 weeks (47,49,52). In contrast, PTSD is only diagnosed safely when symptoms persist for more than 2 months.

The clinical features of PTH overlap with those of the primary headache disorders (i.e., migraine, cluster, tension, etc.) (see Table 105-1). Posttraumatic tension-type headache (90%) is characterized by a dull-pressing, dragging, or pulling pain, which is mainly holocephalic, band- or helmetlike, usually nucho-occipital, seldom episodic, and often continuous (101). Tension-type PTH must be differentiated from posttraumatic cervicogenic headache (8% after WI [24,25]), which is mainly occipitonuchal and typically dragging. The pain of posttraumatic cervicogenic headache radiates from occipital to frontal. It is not holocephalic, strongly unilateral without side change, and commonly associated with limited mobility of the cervical

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TABLE 105-1 Type, Incidence, Characteristics, and Differential Diagnosis of Posttraumatic Headache Following Head Trauma and Whiplash Injury

Type	*Incidence*	*Localization*	*Characteristics*	*Differential Diagnosis*	*Differentiating Features*
Tension-type headache	90%	Holocephalic, mainly occipital	Dull pressing, dragging	Increased intracranial pressure	Level of consciousness, vegetative signs
				Subarachnoid bleeding	Signs of meningeal irritation, focal neurologic signs
				Drug-induced (analgesia) headache	History of medication, drug abuse
				Intracerebral bleeding	Unilateral preponderance, focal neurologic signs
Cervicogenic headache	8%	Nuchal, unilateral	Dragging, triggerable	Vertebral artery dissection	Brainstem symptoms (also transient)
				Prolapsed disk	Brachialgia, radicular signs and symptoms
				Subarachnoid bleeding	Bilateral neck pain, signs of meningeal irritation, altered mentation
Migrainelike	2.5%	Hemicranial, side changing	Pulsating	Head contusion	Circumscript, cranial vault localization
—				Cranial vault fracture	X-ray
—				Scalp injury (subgaleal hematoma)	Clinical inspection, cranial computer tomography
—				Unilateral intracranial bleeding	Focal signs, cranial computer tomography
Cluster-type	Unknown	Periorbital, frontotemporal	Stabbing, pulsating, dragging	Facial skull fracture	Hematoma, x-ray
				Carotid artery dissection	Cervical/facial pain

spine. The pain can be triggered by turning the head, sometimes in a position resulting in pressure on the occipital nerve entry points. The diagnosis is confirmed by a trauma-related bony injury of the cervical spine and by pain relief after local anesthetic infiltration of the tender greater occipital nerve or C2 root (75).

Posttraumatic migrainelike headache (34–36,104,105) is mainly felt as a pulsating, hemicranial, side-changing headache. Accompanying vegetative complaints such as nausea, vomiting, dizziness, photophobia, and phonophobia are common. Posttraumatically isolated aura symptoms also may occur. Rarely, posttraumatic basilar migraine is described with vertigo, nausea, vomiting, and cranial nerve disorders of variable severity (36,43). Cluster-type PTH does not differ clinically from primary cluster headache (82,102). The clinical picture is characterized by unilateral periorbital and frontotemporal pressing, stabbing or throbbing head or facial pain, accompanied by local autonomic signs such as ptosis, miosis, enophthalmos, lacrimation, rhinorrhea, and conjunctival injection.

Clinically important—because it is often misdiagnosed as posttraumatic tension-type headache—is drug-induced persisting headache, which can develop after HT or WI due to prolonged intake of analgesics. Drug-induced headache occurs daily, is prominent in the morning, has a dull pressing character and holocephalic distribution, and is exacerbated by physical activity (22). Clinically, it is best differentiated from chronic PTH or tension-type headache only after successful withdrawal of analgesic intake.

PTH due to traumatic intracranial bleeding in traumatic brain injury (TBI) such as epidural, subdural, subarachnoid, intracerebral, or intraventricular hematoma can mimic PTH of the tension type. In most cases, hematoma-related headache can be clinically differentiated from tension type by a concomitant altered level of consciousness, some confusion, altered mentation, cognitive impairment, vegetative signs, signs of meningeal irritation, or additional focal neurologic signs (see Table 105-1). In acute and subacute hematomas headache occurs in 11 to 53% of cases (40). Elderly patients, patients on antiplatelet medication or anticoagulants, or those that abuse alcohol are at an increased risk for developing a traumatic intracranial hematoma underlying PTH.

The IHS diagnostic criteria and clinical features for headache attributed to traumatic epidural hematoma (5.5.1; Headache Classification Committee, 2004) (40) are as follows:

A. Acute-onset headache, no other typical characteristics known, fulfilling criteria C and D
B. Neuroimaging evidence of epidural hematoma

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C. Headache develops within minutes to 24 hours after development of the hematoma
D. One or other of the following:
  1. Headache resolves within 3 months after evacuation of the hematoma.
  2. Headache persists but 3 months have not yet passed since evacuation of the hematoma.

The IHS diagnostic criteria and clinical features for headache attributed to traumatic subdural hematoma (5.5.2; Headache Classification Committee, 2004) (40) are as follows:

A. Acute or progressive headache, no other typical characteristics known, fulfilling criteria C and D
B. Neuroimaging evidence of subdural hematoma
C. Headache develops within 24–72 hours after development of the hematoma.
D. One or other of the following:
  1. Headache resolves within 3 months after evacuation of the hematoma.
  2. Headache persists but 3 months have not yet passed since evacuation of the hematoma.

According to ICHD-II, headache attributed to traumatic intracerebral and/or subarachnoid hemorrhage or to traumatic intracerebral hematoma is classified and coded as acute PTH attributed to moderate or severe head injury (5.5.1).

DIAGNOSIS

The investigation of first choice following head injury is cranial computed tomography (CCT) with bone window images. CCT is used to screen for any fracture (vault or base of skull) and to identify potential sequelae of intracranial trauma, such as hematoma, focal contusion, or hydrocephalus (51). Also, CCT guides the decision of a neurosurgical operative procedure, when appropriate. Cranial magnetic resonance imaging (MRI) is more sensitive for showing nonhemorrhagic focal contusions. In HT with accompanying cervical spine distortions or in isolated WI, plain cervical spine multiplanar x-rays are necessary to identify fracture, luxation, or kinking of the spine. In addition, functional x-rays with cervical spine flexion and extension views help to delineate indirect signs of a ligamentous lesion or traumatic structural damages such as spondylolisthesis. A dens fracture, luxation, or atlantodental loosening can be excluded with supplementary dens views. Further investigational studies could be conducted depending on the symptom complex and the neurologic signs that accompany PTH (47,52). Repeated studies may be necessary if (a) the character of the headache or its localization change; (b) there is a change or appearance of new, nonheadache symptoms; and (c) new focal neurologic signs emerge.

PROGNOSIS

Most young patients with a mild cerebral concussion (e.g., without loss of consciousness but with vegetative symptoms and PTH) fully recover within a few days (7). The classic cerebral concussion patient with a momentary loss of consciousness and amnesia of less than 60 minutes' duration generally recovers completely within 6 to 12 weeks (32,59). The posttraumatic syndrome after a severe cerebral concussion (e.g., with loss of consciousness greater than 10 minutes and a loss of memory greater than 4–6 hours) usually recovers within months to years (41,83). In an investigation of its course, Denker (20) showed that 90% of patients continued to complain of PTH several months following mild HT, 35% had PTH after 1 year, 22% after 2 years, and only 20% after 3 years. Generally, however, more than 80% of patients become headache free after 6 months (20). These figures are quite consistent from one study to another (8,15,21,26,59,62,63,68,87,88). Prevalence rates of PTH at 6 and 24 months are reported as 27% and 24% after head injury (16) and as 27% and 15% after WI (76,77), respectively. Up to 15% of patients show an incomplete remission from the posttraumatic syndrome (12,23,65,67,88).

The average length of PTH after mild WI without neurologic deficits or bony cervical spine injuries is 3 weeks. In this group, the headache generally disappears within 3 months (47,52). A delayed remission of PTH following WI is found in those patients with an initially severe headache with marked limitation of passive cervical spine mobility (particularly flexion), in patients with poor general well-being and depressive mood with somatic–vegetative complaints, and in the elderly (47,52). Central hypersensitivity may be a risk factor for a poor recovery from posttraumatic pain (19,70,100). Posttraumatic de novo cervicogenic headache after WI is rare and has a good prognosis. The prevalence decreases from 8.3% within the first 6 weeks to 4.5% after 6 months and to 3.5% 1 year following WI (24,25).

MANAGEMENT

The treatment of acute PTH varies according to the type of headache and its duration. Treatment guidelines are based on clinical experience and controlled studies (47,52,78,90). First-choice treatment for the acute posttraumatic tension-type headache is analgesics such as acetylsalicylic acid (ASA; 500 to 1000 mg daily, maximum 1500 mg daily after excluding intracranial hemorrhage) or paracetamol (500 to 1000 mg, maximum 1500 mg daily). Alternatively, ibuprofen in retard form or delayed release (400 to 600 mg

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daily) or naproxen (500 to 1000 mg daily) can be used. The use of combination preparations is discouraged. Should the headache continue for longer than 4 weeks, no further analgesics should be given because of the danger of developing drug-induced persisting headache.

The posttraumatic migraine-type headache is treated during the attack, with an analgesic (e.g., ASA 1000 mg in effervescent tablet form) together with an antiemetic (e.g., domperidone or metoclopramide, 10 to 20 mg orally). In the case of severe, long-lasting, or frequent migrainelike attacks, prophylactic drug treatment with a beta-adrenergic blocker such as metoprolol or propranolol could be used. Migrainelike PTH responds to propranolol or amitriptyline (25 to 150 mg daily) as monotherapy or in combination. The seldom seen PTH of the cluster type is treated in the same way as primary cluster headache.

Acute posttraumatic (cervico) cephalic pain following WI (QTF Grade II) is treated with early active interventions with frequent active cervical rotation and physiotherapy (e.g. McKenzie's principle) (84,96). This treatment is more effective in pain relief than initial rest, soft collar, or gradual self-mobilization (13,64,66,90). This regimen is of importance, as acute PTH is correlated with low neck mobility (44). Cervical immobilization wearing a soft collar should be avoided (13). However, when neck and head pain is stronger in the morning, wearing a temporary and short-term cervical collar at night can be considered to avoid the additional cervical soft-tissue straining with head movement in deep sleep due to nocturnal-dependent hypotonia of the neck musculature.

Supplementary physical measures with heat applications should be considered. Dry heat (infrared light, arc light, warm air, and heat pillows) or moist heat (hydrocollator packs) have all proved useful (MK, personal observation). If there is no therapeutic benefit from physical measures, additional therapy with muscle relaxants, antiinflammatories/antirheumatics, and if necessary, analgesics may be necessary (49,52). In order to prevent medication overuse and subsequent drug-induced headache, the drug treatment of acute PTH following WI should include strict control of medication intake.

Acute PTH following HT or WI should not be treated, as a rule, with opioid analgesics, because of the possibility of dependency and substance overuse. Similarly, prolonged use of benzodiazepines as muscle relaxants should be avoided. Although the short-term use of a peripherally acting analgesic is reasonable in the initial acute stages, longer use (i.e., longer than 4 weeks) should be disallowed to avoid drug-induced headache. Similarly, drug-induced headache also can develop with regular use of combination analgesics. Other obsolete therapies for the pain of acute PTH include antihistamines, steroids, neuroleptics, barbiturates, and ergot preparations. Manual traction or pulling of the cervical spine with Glisson slings in the acute phase of the cervicocephalic pain syndrome following cervical spine distortion is obsolete. The same is true for immobilization with a plaster collar (Minerva plaster). Nonbeneficial and often pain-intensifying maneuvers include massage of the stretched musculature and reflex zone; locally invasive anesthetic measures such as subcutaneous, perineural, or intraarticular infiltration; neural therapy; acupuncture; and acupressure. Fresh cell and ozone therapy are obsolete in posttraumatic tension-type headache. The benefit of local unguent treatment for the nuchal pain that often accompanies the headache is not proven. A 24-hour clinical observation period is usually sufficient after cerebral contusion but prolonged bed rest may increase the risk of delayed remission.

The management of PTH after intracranial hematoma is covered elsewhere in the book.

ACUTE POSTCRANIOTOMY HEADACHE

EPIDEMIOLOGY

Up to 80% of patients report moderate to severe pain after craniotomy. The incidence of headache after infratentorial (suboccipital) craniotomy is higher than that reported for supratentorial craniotomy (45).

Supratentorial Craniotomy

In a series of 126 patients who underwent supratentorial craniotomy (with anterior temporal lobectomies for intractable epilepsy), 17% developed postcraniotomy headaches (PCH) persisting beyond 2 months postoperatively. In 6% the headaches lasted more than 2 months but less than 1 year. Twelve percent suffered from chronic ongoing postcraniotomy headaches 1 year after surgery (45). A comparable PCH incidence of 19% in supratentorial craniotomy is described in a controlled retrospective study selecting patients who did not complain of headache preoperatively (31).

Infratentorial Craniotomy

Soumekh et al. reported that 16% (7/56) of patients who underwent suboccipital craniectomy (craniotomy without primary cranioplasty) for cerebellopontine angle tumors, microvascular decompression, or vestibular nerve section developed PCH (98). In a retrospective study, 54% (37) of 228 patients who underwent suboccipital craniotomy for removal of cerebellopontine angle tumors complained of PCH and 27% had PCH for more than a year after surgery (37).

In suboccipital (noncranioplastic) craniectomy with a retrosigmoid approach to acoustic neuroma removal, the incidence of PCH was reported as 23% (of 331 patients) at 3 months, 16% at 1 year, and 9% at 2 years (38). In a questionnaire study, 64% (of 58 patients) experienced PCH due to acoustic neuroma excision by suboccipital

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craniotomy (92). The incidence of PCH subsequent to resection of acoustic neuroma is reduced by the performance of a craniotomy with primary cranioplasty (4% vs. 17% [39,89]). Suboccipital minicraniectomy (2 × 2 cm) resulted in significantly diminished PCH severity from the third month postsurgery of acoustic neuroma as compared with wide craniectomy including dissection of the upper cervical musculature (89). Within the first postoperative year the pain severity of PCH is significantly less in patients having undergone a translabyrinthine resection instead of a suboccipital resection of acoustic neuroma (85,92).

Removal of vestibular schwannoma was associated with a PCH in 34% (52 of 155 patients) 3 months after retrosigmoid craniotomy (91). Immediately after surgery for vestibular schwannoma 61% (154 of 251 patients; 89 (35%) with, 65 (26%) without preoperative headache) reported PCH (60). PCH persisted in 37% for an average of 8.9 years; after this period 11% suffered from a disabling PCH (60). If PCH continues for at least 1 year, the prognosis is not favorable.

Retrosigmoid approach, small tumor size, postoperative gait problems, and preoperative headache are risk factors for PCH (60). Also, PCH is more common in patients with direct dura closure, with bone flap replacement, or with a laboratory-proven aseptic meningitis (91). Jackson et al. described a PCH incidence of 54% of patients with retrosigmoid vestibular schwannoma resection with intradural drilling compared to a PCH incidence of just 5% of patients with vestibular nerve section without intradural drilling (42).

PATHOPHYSIOLOGY

Adherence of cervical muscles to the dura with consequent traction of the dura is suggested to be responsible for PCH in suboccipital craniectomy (craniotomy without primary cranioplasty) (54,91,93). Occipital nerve entrapment or neuralgia may explain PCH in other cases (61). Furthermore, a trigeminal system–mediated cause for PCH is suggested based on the effect of sumatriptan on PCH (61). Single case reports of cerebrospinal fluid leak from the craniotomy site may point to intracranial hypotension as causative of posture-related PCH (69,79). Finally, intradural drilling and the use of fibrin glue may cause aseptic meningitis and PCH after suboccipital/retrosigmoid craniectomy for tumor resection of the nervus octavus (42,91).

CLINICAL FEATURES

The IHS diagnostic criteria (5.7) for acute PCH (ICHD-II) (40) are as follows:

A. Headache of variable intensity, maximal in the area of the craniotomy, fulfilling criteria C and D.
B. Craniotomy performed for a reason other than head trauma
C. Headache develops within 7 days after craniotomy.
D. One or other of the following:
  1. Headache resolves within 3 months after craniotomy.
  2. Headache persists but 3 months have not yet passed since craniotomy.

When the craniotomy is performed for complications of head trauma, the postoperative headache is classified as Acute PTH attributed to moderate or severe head injury (5.1.1).

Postcraniotomy headache is clinically characterized as a site-of-injury-headache with pain maximally experienced over the surgical site with or without an accompanying tension-type–like headache (31). In general, PCH resembles PTH following HT or WI. In rare cases, a cerebrospinal fluid leak from the craniotomy site resulting in an alteration of intracranial pressure can cause a posture-related headache following surgery (69,79). Other types of secondary headaches caused by complications of surgery such as septic meningitis (ICHD-II: 9.1), meningoencephalitis (9.1), intracranial hemorrhage (6.2), cerebral ischaemia (6.1), and cerebral venous thrombosis (6.6) have to be differentiated from PCH (see Table 105-1).

MANAGEMENT

The treatment options of PCH do not differ from those of PTH due to HT or WI (47,49,50,80). Controlled studies investigating the efficacy of specific therapeutic strategies in PCH are lacking. Anecdotally, the severity of acute PCH can be reduced by a scalp nerve block with ropivacaine performed before awakening from anesthesia (71). The majority of patients that complain of headache can be adequately treated with nonsteroidal antiinflammatory drugs (NSAIDs). Patients with PCH unrelieved by NSAIDs may benefit from treatment with divalproex sodium and verapamil (37). Sumatriptan may alleviate pain in some patients with PCH (61).

PREVENTION

Osteoplastic suboccipital craniotomy replacing the bone flap between muscles and dura at the time of craniectomy significantly reduces the incidence of PCH compared to a simple craniectomy (29,39,54,98). Consequently, primary cranioplasty is recommended as a surgical strategy to minimize the risk of developing postoperative headache due to craniotomy (29,39,54,89,91,93,98). In surgery of acoustic neuroma, "mini" craniectomy seems to be superior to wide craniectomy (89) and translabyrinthine resection seems to be superior to suboccipital resection (85,92) in preventing PCH.

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Prevention of PCH due to suboccipital craniotomy includes (a) prevention of the use of fibrin glue; (b) prevention of extensive drilling of the posterior aspect of the internal auditory canal (42,91); (c) duraplastic instead of direct dural closure (91); and (d) primary replacement of bone flap (42,91).

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