REVIEW OF RECENT MEDICAL LITERATURE

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New Views on Headache*

Subtle Disk Damage May Explain Cervicogenic Headache[1]

Diagnosis of chronic headache continues to be a challenge, as the search to establish standard criteria to determine the source of headache remains elusive among medical specialists. A percentage of chronic headache (cervicogenic headache) cases can be caused by damage to cervical intervertebral disks and can go undetected by magnetic resonance imaging, (MRI) or conventional imaging technologies.

The proceedings of the 18th Annual Scientific Meeting of the American Pain Society reported novel approaches to efficacy in narrowly defining causative factors in chronic headaches which are often due to undetected damage to the cervical intervertebral disk. These cervicogenic type headaches can also reveal injuries such as the separation of disks from the bony endplates of the vertebra. Brain lesions involving considerable pain can result from the separation of the annulus from the periosteum.

Damage to the deep structures of the spine, including the nerve roots, joints, and disks can confine patients as to the origin of their pain. Damage to C1-C2 joint generates pain that radiates over the top of the head known as "occipital headache." Elderly patients suffer from this condition due to degenerative joint damage and as a result of trauma as children and young adults. The C1-C2 joint is anatomically located near the C2 dorsal root ganglion. When the ganglion is irritated and the C1-C2 joint is injured, a C2-pattern headache or neuralgia may result. A C1-C2 trauma case study is associated with bleeding occurring directly into the capsule and irritation of the C2 ganglion. Selectively over-anesthetizing just the C2 nerve can help refine the diagnosis.

Damage to C2-C3 accounts for 52% of whiplash headaches. Whiplash damage is usually due to deep injuries to the cervical spine followed by a reactive secondary event of myofacial damage. Radiculopathy or neuralgia, pain in the temporal region or behind the eyes, can result from hemorrhage in the C3 dorsal root ganglion. The delicate procedure of administering anesthesia to block the C3 ganglion can relieve the pain and confirm the diagnosis. The vertebral artery lies adjacent to the C2-3 joint. If the artery is punctured accidentally, seizures can result; if steroids are injected stroke can result.

Two sets of somewhat conflicting diagnostic criteria exist for cervicogenic headache. According to standards set by the International Headache Society, it requires an abnormality in the neck, yet this is not usually found. Cervicogenic headache without related radiologic abnormalities has been seen in medical practice. The 18th Annual Scientific Meeting of the American Pain Society reported a more useful definition that includes head or unilateral facial pain without a side shift (as opposed to migraine) but with postural and mechanical triggers restricting the range of movement of the neck; or "with nonradicular ipsilateral neck, shoulder, or arm pain." Compared to traditional migraine treatments, the headache is thought to be refractory. Moreover, pharmacologic treatments for cervicogenic headache such as antiepileptic drugs (AEDs), tricyclic antidepressants, anti-inflammatory drugs (NSAIDs), and muscle relaxants, have yet to be proven effective across patient populations. Anesthetic blockade is generally considered the treatment method of choice and surgery a last treatment option because of the risks involved.

Migraine Headache

The incidence of posttraumatic migraine and post-head trauma syndrome (formerly known as post concussion syndrome) are reported with frequency. Any mild or severe trauma can result in episodic (typical migraine), or chronic tension-type headache that may be experienced daily. In fact, posttraumatic headache can follow a cervical trauma without direct injury to the head. Migraine can develop after an initial encounter with a traumatic event. Symptoms of trauma usually subside within one to three

Continued on Page 10

«Review, cont'd from Page 5»

weeks, although some affected individuals do not present with headaches until well after the traumatic incident, sometimes beginning two, three to even 12 months later.[2]

The etiology of posttraumatic headache does not include a textbook pathway. It is thought that shearing forces that compromise the function of nerves in the brain, brain stem, and spinal cord are probably involved, as well as other causes such as alteration in neuroendocrine function due to biochemical changes.[3] Posttraumatic migraine may have diagnostic, therapeutic, and legal implications according to subtype in different contexts. It can also be a part of a much broader category of symptoms known as "posttraumatic syndrome" which is associated with near-daily tension-type headaches that are in contrast to footballer's migraine, which is more episodic and usually accompanied by depression, dizziness, impaired cognition or concentration, and personality changes. Posttraumatic syndrome may also have neurological, psychological, and cognitive symptoms with or without associated dizziness, vertigo, or light-headedness. Irritability, difficulty in concentrating, and anxiety with depression and personality changes are also reported.[4]

Another subtype includes the onset of the first attack of migraine in susceptible people. Initial and subsequent migraine attacks are not caused only by head trauma. In the inter-migraine period, non-migrainous and neurologic symptoms are absent. Predisposing factors may be a family history of migraine, childhood precursors of migraine (e.g., bouts of unexplained abdominal pain, nausea, vomiting) motion or car sickness, and comorbid conditions (e.g., epilepsy, anxiety disorders, depression). One speculative theory accounts for posttraumatic headache due to trauma-induced alterations in the brain.[5]

The diagnosis of most cases of posttraumatic headache includes symptoms more closely related to tension-type headache, though not as severe as migraine, steady and nonthrobbing, not worsening with movement, bilateral, and unassociated with photophobia, phonophobia, nausea, or vomiting.

Critical Care Management and Severe Traumatic Brain Injury

The management and care of traumatic brain injury (TBI) has evolved into a health care industry with complex, medical, economical, and social responses. Developmental strategies to restore normal brain function to traumatic brain injury (TBI) patients is critical to measurable positive outcomes determined by the expertise of the prehospital care provider, the emergency physician, the critical care management team as well as the health care facility. Each TBI is as different as the patients who are affected by the condition. The brain may suffer damage at the onset of the injury or may develop after exposure to the healthcare triage center. Special care is taken to reduce the rate of morbity and mortality beginning in the field. Head injuries require that more than 400,000 people visit hospitals each year with an unknown number who do not seek medical intervention. The mortality rate includes 75,000 people each year in the United States. In addition, on any given day 15,000 people lie comatose in North America.[6]

Few techniques existed for the diagnosis and treatment of TBI in the first half of the 20th Century other than clinical and radiological survey for surgical lesion. Some treatments such as corticosteroids have been discontinued whereas osmotic therapy continues to be utilized. Perhaps one of the most important developments in medical treatment is the organization of intensive care units in which physiologic outcomes could be monitored and reported. In the 1950s, physiologic parameters could be measured by intraarterial, intravenous, and central catheters. In the 1960s, intracranial pressure (ICP) was first measured by a rontal intraventricular catheter, currently the most cost-effective and accurate method of ICP measurement.[7] The device was fluid-coupled and upon calibration, the fluid pressure could be measured with a strain gauge, and cerebrospinal fluid (CSF) could be drained to reduce elevated pressure. Parenchymal monitors followed the development of fiberoptic technology; though easier to work with they lose accuracy over time and cannot drain CSF.

Before the 1970s, surgical decisions relied on neurologic examination assisted by the technologies of radiography, cerebral angiography, and air ventrivulography. In the mid-1970s, technology made a

quantum stride with the development of computerized tomography (CT scan). Prompt diagnoses, including those of severe TBI, became available with the introduction of fast scanners. Jennett and Teasedale described the clinical procedure that became to be known as the Glasgow Coma Scale (GCS) in 1974. It was developed primarily for use by nonneurosurgeons and neurosurgeons alike. It is also used currently to group patients into mild, moderate and severe TBI.

A landmark study in 1977 by Becker and others confirmed the general experience that early intervention and diagnosis and aggressive treatment of severe brain injury results in improved outcomes.[8] Aggressive treatment includes ICP monitoring, early surgical decision making, and treatment of elevated ICP. Becker et al. reduced the previously studied fatal outcome and severe disability of 63% of severely brain injured patients to an outcome to 43%.

In the 1980s, the implication was made that impairment of cerebral perfusion is at least as important to outcome as the onset of injury. Seelig et al. showed improved outcomes for those patients whose subdural hematoma were evacuated within 4 hours of injury.[9] Furthermore, a study of 1,030 of severely brain-injured patients provided data to participating centers of the Traumatic Coma Data Bank confirmed the earlier work of Jenkins in 1986 that showed the vulnerability of the traumatized brain to ischemia and hypoxia as compared to the normal brain. Study of the database assisted care providers in realizing that an episode of prehospital hypoxia or hypotension was the most powerful predictor of poor outcome.[10] Moreover, cerebral edema, hypotension, low systemic blood volume, intracranial mass lesions, and pulmonary disease all acquired new importance as parameters of neurologic outcome because of their effects on cerebral perfusion and oxygenation. Today, traumatic brain injury has a time frame that may require moment-to-moment critical care management.

Myofascial Pain Syndrome[11]

Although controversy exists as to the validity of the distinction between myofascial pain and fibromyalgia, most specialists underscore the existence of myofascial pain syndrome. A survey of 1,663 members of the American Pain Society attending the 18th Annual Meeting reported 88.5% of respondents affirming the legitimacy of myofascial pain syndrome, and 81% would diagnose the condition as distinct from fibromyalgia. Note that consensus among specialists is clustered by specialty. The range varied from 50% of surgical specialists (neurosurgeons, orthopedic surgeons) to 100% of pediatricians, obstetricians/gynecologists, and rheumatologists.

Symptoms generally thought to be consistent with myofascial pain syndrome confirmed by 80% respondents include tender points, muscle ropiness, a decreased range of motion, pain exacerbated by stress, palpable nodules, descriptions of regional pain as achy, dull, or deep in nature. The top symptoms confirmed by 90+% of respondents included trigger points, pain decreasing after local injection, taut bands, and pain decreasing with spray and stretch.

Not all specialists agreed on which factors should be considered essential in diagnosis although most felt that reflex abnormalities, scar tissue, sensory abnormalities, and the results of most tests (thermography, erythrocyte sedimentaton rate, and algometry) as unimportant. Neurosurgeons as a group specialty were unique in their consideration of representative criteria for diagnosis of mysofascial pain syndrome, and did not consider "trigger points" an essential signal to diagnosis.

Traumatic Brain Injuries and HMO Guidelines[12]

"Treatment Guidelines" (a misnomer) determine the level of access a patient has with his managed health care insurer and health care provider. Although it is assumed that HMOs establish their own guidelines, in actuality health care guidelines are published by relatively unknown companies. These companies amass the power to market and sell specific practice "guidelines" to HMOs. Included in the guidelines are estimates of length of stay and the possible complications a person may experience for each illness.

Most guidelines are strict policies that can impact "life or death" situations for patients, particularly those with prior medical history and catastrophic illness or severe injury such as traumatic brain injury (TBI). The initial thrust of treatment guidelines centered around recommending recovery time for

categorical medical illness. Alternatively, it has evolved into a useful tool that HMOs use to deny treatment and/or reimbursement. Therefore, the guidelines in many cases have constructed barriers to the health care delivery system.

Some of the constraints of external practice guidelines include but are not limited to the fact that guidelines vary significantly from insurance plan to insurance plan; insurance companies modify the guidelines or restrict them according to their needs; HMO guidelines are not made available to the insured, the treating doctor, or other health care professional.

TBI cases can be denied based on the following criteria:

- The requested treatment is not medically necessary
- The requested treatment is experimental
- The individual has plateaued and ongoing care is custodial in nature
- Given the person's condition, the most appropriate care has been provided
- There is a limit to what the insurers can do
- If every appeal was granted, insurers would go out of business

Further restrictions in HMO guidelines account for several reasons for denying care or premature discharge from a health care facility, including:

- Unfair and unnecessary medical documentation required before approval for inpatient rehabilitation
- Overly restrictive guidelines requiring unrealistic progress before acceptance into a coma stimulation/coma awareness TBI program
- Directives limiting the number of home care visits, and type and frequency of therapies for individual with brain injury
- Primary care providers are encouraged to limit testing and referral of seriously ill and injured person under a capitated system
- Discharge from hospital emergency rooms without treatment or admission

Research studies have been published that determine the effect of restricted treatment and HMO guidelines have on the health of the individual and recovery time from medical illness. One study has shown that neonates discharged one day after birth face increased risk of developing jaundice, dehydration, and dangerous infection. A National Cancer Institute study found that women treated for mastectomies in an outpatient setting may have a higher risk of complications related to surgery, including infection and embolism.

Denial of benefits by HMOs have mobilized state legislation to endorse laws for the use of external reviews which provide the insured an unbiased review of utilization decisions by an impartial panel. Moreover, appealing an HMO decision requires excellent legal counsel and verified insurance coverage and attention to the Appeals process which is often unsuccessful due to restrictive time limits for filing; treatment guidelines and regulations are not "set in stone" in a written document; the hearing is often done over the phone; substantive information used as *prima facie* evidence may not be read prior to the hearing; the doctor as well as the employer may be unwilling to support a disabled person for fear of sanctions by the HMO.

The Appeals process can be successful. In 1999, a court in California ordered an HMO to pay in excess of $100 million for not providing appropriate care in a timely manner; in a Pennsylvania court, a decision in a landmark case ruled that some HMOs are providers of healthcare services, not just payors of services, and therefore may be found liable for rendering improper medical care under the theory of "corporate negligence."

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