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Vertebral and Carotid Artery Dissections

Lucian Maidan, MD

Although only 10% of the 750,000 people who annually suffer an ischemic stroke in the U.S. are younger than 50, those younger individuals have a higher mortality. Dissection of carotid arteries (CAD) and vertebral arteries (VAD) is second only to cardioembolic strokes in young people, with an incidence estimated at up to three per 100,000 people for carotid artery dissection and 1.5 per 100,000 for the vertebral arteries.

The second most common lesion of the cervical arteries after atherosclerosis, dissection may be either spontaneous or secondary to major trauma. The majority of dissections occur extracranially—only 10% are intracranial—and in 16% of cases multiple arteries are involved.

Dissection is most likely due to multiple environmental and genetic risk factors. Also, recent infections (mainly respiratory) have been associated with dissection.

A genetic predisposition may be present for an underlying structural defect affecting the media and adventitia of the outer wall of superficial temporal arteries of patients who have had dissections. Also, absence of a demonstrable intimal tear on some histologic examinations suggests that at least some dissections are caused by a primary intramural hematoma. Several connective tissue disorders have been associated with dissection, including vascular Ehlers-Danlos syndrome (type IV), Marfan syndrome, and osteogenesis imperfecta.

Dissection is most likely due to multiple environmental and genetic risk factors. Also, recent infections (mainly respiratory) have been associated with dissection.

The most important environmental risk factor appears to be major or minor trauma caused by events such as chiropractic manipulation, yoga poses, coughing, and sneezing. Trauma produces an intimal tear through which blood enters the wall and forms an intramural hematoma.

Clinical manifestations of a cervical artery dissection include both local signs and symptoms and ischemic and even hemorrhagic cerebral events.

Local manifestations of CAD include Horner syndrome (ipsilateral), neck pain, headache, tinnitus, facial pain, and cranial nerve palsies (IX to XII most commonly).

Local manifestations of a VAD include severe occipital headache, nuchal pain, cervical root involvement (most commonly C5-C6 level), and lower brainstem compression if the dissection extends in the intradural space.

Ischemic manifestations of CAD include stroke, most commonly in the middle cerebral artery distribution, amaurosis fugax, ischemic optic neuropathy, and retinal infarction.

An estimated 85% of patients with a VAD will develop ischemic manifestations involving the posterior circulation sometimes after days or weeks delay. Most often the presentation is a lateral medullary syndrome (Wallenberg syndrome), which consists of ipsilateral facial pain and numbness, contralateral loss of pain and temperature, dysartria, hoarseness, hiccups, vertigo, nausea, vomiting, ataxia and diplopia. If other areas of the brainstem are involved, a medial medullary syndrome with characteristic contralateral weakness and numbness, Horner syndrome or, if the cerebellum is


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Idiopathic Intracranial Hypertension

Peter T. Skaff, MD

Idiopathic intracranial hypertension (IIH), first described by Heinrich Quincke in 1893 as "serous meningitis," is an unusual cause of chronic headache. Also referred to as "benign intracranial hypertension," this disorder can result in permanent blindness if not recognized and treated promptly and should not be regarded as benign.

The common name, pseudotumor cerebri, refers to the presence of papilledema without a demonstrable, intracranial mass, while IIH is used specifically when no other secondary cause of raised, intracranial pressure (ICP) is discovered. Secondary causes of pseudotumor cerebri include cerebral venous thrombosis, steroid withdrawal, Addison disease, obstructive sleep apnea, hypoparathyroidism, and hypervitaminosis A.

The annual incidence of IIH is approximately 1 in 100,000. However, IIH affects approximately 1 in 25,000 women of childbearing age, and the incidence increases to 1 in 5,000 for those with co-morbid obesity. In particular, weight gain over the preceding 12 months is the most predictive and well-established risk factor.

Patients with IIH generally present in one of two ways. Either papilledema is incidentally discovered at the time of optometric or ophthalmological examination, or clinical evaluation of chronic headache is sought. In the first case, further investigation with neuroimaging and referral to a neurologist is assured. In the second case, the clinician must maintain a high index of suspicion, make specific inquiry into related symptoms and secondary causes, and perform an appropriate physical exam including vision, eye movement, and fundoscopy.

The vast majority of patients with IIH present with headache that is typically holocranial, occurs daily, is worse upon awakening or when lying down, is better when sitting or standing, and is aggravated by activities that raise ICP, such as coughing, sneezing, bending over, or performing a Valsalva. Associated symptoms include transient visual obscuration, photopsia (light flashes), pulsatile tinnitus (often described as whooshing), retrobulbar pain or pressure, neck pain or stiffness, diplopia, and visual loss. Although formal, automated visual field testing will demonstrate peripheral vision loss (scotoma) in a majority of patients, most will be unaware of the deficit due to preservation of central vision and of visual acuity. Blindness occurs in as many as 5% of cases, and though often gradual, it can be acute. Approximately 10% of patients are asymptomatic, and symptomatic IIH can also present without papilledema if there is chronic optic atrophy with thinning of the retinal neural fiber layer.

The diagnosis of IIH is made on the basis of clinical history, physical examination, imaging studies, and lumbar puncture. The physical examination should be normal except for papilledema and the occasional finding of an abducens nerve palsy, which can be caused by increased ICP.

Modified Dandy Criteria for Diagnosis of IIH:

1. Symptoms of raised ICP (e.g., headache, visual loss, papilledema)
2. No localizing signs other than abducens (6th) nerve palsy
3. The patient is awake and alert
4. Normal brain imaging without evidence of venous thrombosis (e.g., MRI w/ MR Venogram)
5. Lumbar puncture opening pressure > 250 mm CSF with normal composition
6. No other explanation for the raised ICP

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Guillain Barre Syndrome

Ryan Armour, DO

Guillan Barre syndrome (GBS) is the eponym used to describe a group of rapidly progressive immune mediated polyneuropathies. GBS is caused by autoantibodies binding to peripheral nerves causing demyelination and sometimes axonal loss. Many cases are preceded by a relatively benign viral or bacterial illness, vaccination or surgical procedure. Typical patients will develop muscle weakness that worsens over the course of days, beginning in distal lower limb muscles.

Care must be taken to monitor patients for dysautonomia, as this can occur in 70% of patients.

Proximal lower limb muscle, upper limb muscle and facial muscle weakness may develop over a few days. Patients will complain of back pain throughout the clinical course of the disease. The weakness will progress over the course of two to four weeks and then slowly improve, even without treatment.

Clinical examination will demonstrate muscle weakness in the extremities, with distal muscles affected more than proximal muscles. Absence of deep tendon reflexes is seen in nearly all patients with GBS but may be normal early on. Parasthesias involving the distal extremities are common, but patients will often have a normal sensory examination. Severe or variant cases may present with ataxia, severe sensory loss, or weakness of extraocular muscles. It is not uncommon for patients to be seen by multiple providers over the span of several days before the proper diagnosis is made.

Cerebrospinal fluid (CSF) analysis should be performed and typically shows elevated protein levels with normal cell counts. CSF studies are often normal during the first week of symptoms. Nerve conduction studies are not necessary in all cases, but can be helpful when the clinical examination and CSF findings are insufficient to determine the diagnosis.

Care must be taken to monitor patients for dysautonomia, as this can occur in 70% of patients. Cardiac arrhythmias, gastrointestinal dysmotility and rapid and dangerous blood pressure fluctuations may occur suddenly, making patients at risk for significant morbidity and sudden death. Severe respiratory weakness requiring ventilatory support may occur rapidly in up to 30% of patients. For these reasons, patients admitted into the hospital must be monitored with cardiac telemetry. Bedside spirometry with close attention to forced vital capacity and negative inspiratory force is critical.

Treatment is indicated for patients who have progressively worsening weakness. Symptoms may relapse after some initial improvement in the eight weeks after initial onset of weakness. Intravenous immunoglobulin (IVIG) or plasmapheresis have both been shown to improve recovery time. Physical therapy should be started early in the course of treatment and continued with a multidisciplinary acute rehabilitation team if available. The majority of patients with GBS are able to walk independently six months after onset of disease. However, about 50% of patients will report some weakness or fatigue that is persistent for one year. Immunizations are not recommended during the acute phase. It is generally suggested that immunizations be held for one year, then resumed normally. Decisions to withhold vaccines for a longer period of time can be made on an individual basis. ■

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Medical treatment of IIH is appropriate for patients with mild or no vision loss and includes weight loss, salt restriction, carbonic anhydrase inhibitors (e.g., acetazolamide, topiramate), and diuretics (e.g., furosemide). Surgical treatment is generally reserved for patients who do not improve with medical therapy or who present with significant visual loss. Options include serial lumbar punctures, CSF shunting, venous sinus stenting, optic nerve sheath fenestration, and gastric bypass. All patients should be followed ophthalmologically with dilated fundoscopy, automated visual field testing, and ocular coherence tomography to assess treatment response.

For references, please email us at DignityHealthNeuro@DignityHealth.org. ■

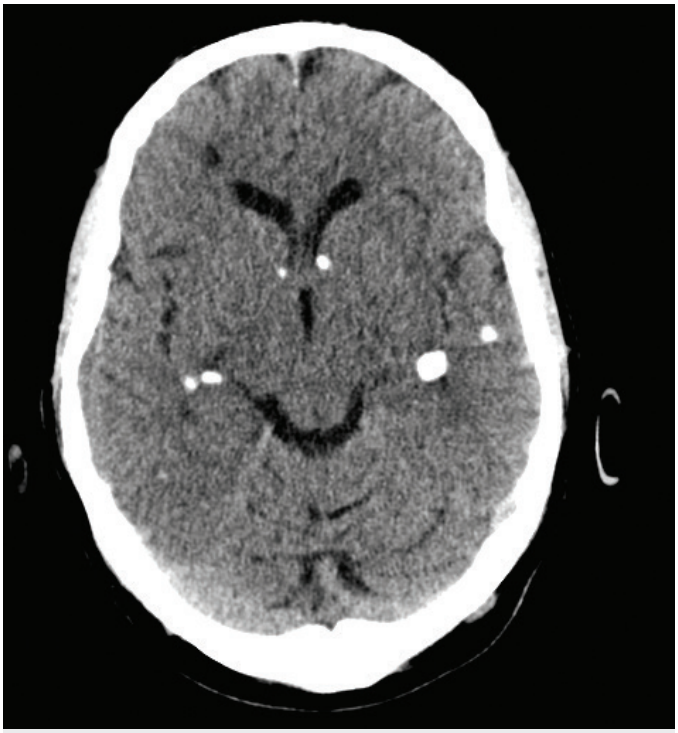
Neurocysticerocosis

Sanaz Abderrahmane, MD

Cysticercosis is caused by the larval stage (metacestode) of the pork tapeworm Taenia solium. Tapeworm eggs are spread through food, water, or surfaces contaminated with feces. Humans swallow the eggs when they eat contaminated food or put contaminated fingers in their mouth.

Cysticerci develop in the skeletal muscle, the subcutaneous tissue, and mainly in the central nervous system (CNS), where they lead to a clinical pleomorphic disorder known as neurocysticercosis (NCC). It can also cause ophthalmic cysticercosis, where the larvae can lodge in the retina or in the vitreous humor.

It is a disease endemic throughout Latin America, East Africa, India, China and Indonesia. This disease represents a serious public health problem in developing countries and is considered a disease of poverty. In the U.S., it is most often seen in immigrants from Mexico and other Latin American countries.



Calcifications identifying cerebral cysticercosis

Neurocysticercosis is a complex disease. Its symptoms depend on the number, type, size, localization and stage of development of cysticerci in the CNS and meninges, as well as on the degree of inflammatory response and the host conditions. Seizures are the most common symptom, occurring in over 70% of patients with NCC. In countries where the disease is endemic, there are a lot of cases of epilepsy in adults whose underlying cause is the cysticercosis (CC).

When cysticerci lodge within the ventricular system, acute intracranial hypertension (ICH) secondary to hydrocephalus may develop. Cysts in the subarachnoid space may invade the Sylvian fissure and grow to large size and can cause hemiparesis, partial seizures or other focal neurological signs.

The most severe clinical presentation of NCC is racemose cysticercus, which is the presence of multilobular cysts in the ventricular and subarachnoid spaces, causing obstructive hydrocephalus and arachnoiditis that might be life threatening. They are known to undergo disproportionate growth with extensions of membranes that group in clusters resembling bunches of grapes, which cause inflammatory reaction, fibrosis, and progressive thickening of the leptomeninges at the base of the brain. Mortality rate can be very high from hydrocephalus secondary to cysticercotic meningitis even after VP shunt.

NCC can be active or inactive. The active form is associated with living parasites that cause arachnoiditis, hydrocephalus, and stroke. Inactive NCC presents with parenchymatous calcifications or meningeal fibrosis. The symptoms of the disease are a result of the granulomatous inflammatory (active form) process associated with the immune response against the parasite.

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The diagnosis of NCC is based upon clinical features, epidemiologic aspects, neuroimaging studies, laboratory analysis of the CSF and antibody detection in the serum. Serologic tests can be helpful but not always necessary.

Radiographic imaging is an important diagnostic tool for diagnosis of NCC and extraneural cysticercosis. CT is better for identifying calcifications and often sufficient to identify parenchymal cysticerci. However, MRI is better for detection

of extra-axial cysts. Spinal fluid may show mild elevation of white cells, sometimes eosinophils, and elevation of protein may be present in inflammatory forms. Lumbar puncture may be contraindicated when increased intracranial pressure is present. In some cases are if the radiologic findings are diagnostic. Visualizing the parasite in the ocular fundi is pathognomonic for cysticercosis.

Therapeutic measures include antiparasitic drugs, surgery, and symptomatic medication. Praziquantel and albendazole are effective antiparasitic drugs against T. solium cysticerci. Between the second and fifth days of antiparasitic therapy, there is usually an exacerbation of neurological symptoms, attributed to local inflammation due to the death of the larvae. For this reason, albendazole and praziquantel are generally given simultaneously with steroids and with anti-seizure medications in order to control the edema and intracranial hypertension that may occur as a result of therapy.

Between 65% and 85% of parenchymal brain cysticerci are killed after standard courses of treatment, with most trials showing a higher parasiticidal effect of albendazole. Because it has better penetration into cerebrospinal fluid, its concentrations are not affected when given with steroids, and it does not decrease the concentration level of anti-seizure medications (phenytoin and carbamazepine).

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The role of surgical therapy in the management of neurocysticercosis has significantly decreased over time and is now mainly restricted to placement of ventricular shunts for hydrocephalus secondary to neurocysticercosis. The high prevalence of shunt dysfunction is problematic.

Family members may be tapeworm carriers. Identification and treatment may prevent further cases. Improving sanitation and educating people are all major ways to discontinue the cycle. Cooking of pork or freezing it and inspecting meat are effective means to cease the life cycle. Meat products need to be well cooked. The importance of hand washing after handling meat cannot be overemphasized. When visiting a developing country, use special caution with food and drink to avoid infection. ■

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affected, ataxia and nystagmus are noted. Ischemic infarcts infrequently affect the spinal cord, and if the intracranial segment of the vertebral artery is involved, subarachnoid hemorrhage (SAH) can occur.

The diagnosis of arterial dissection can be made by CT angiography (CTA) or MR angiography (MRA). Magnetic resonance imaging with high resolution 3Tesla magnets has the advantage of showing the intramural thrombus. If these noninvasive studies are inconclusive a catheter cerebral angiogram should be done.

In patients with symptomatic CAD or VAD, intravenous t-PA within 4.5 hours of onset has been shown to be safe and effective.

In the 2015 Cervical Artery Dissection in Stroke Study (CADISS) trial, the 90-day recurrence rate for ischemic stroke was only 2%, and it occurs early after the dissection. That trial randomized 250 patients with extracranial arterial dissection within seven days of developing symptoms to antiplatelet therapy or anticoagulation (heparin and warfarin) and found no difference in efficacy of either treatment in preventing stroke or death.

Endovascular surgical treatment for symptomatic arterial dissection is reserved for patients who continue to have ischemic events despite antithrombotic therapy or for intracranial dissection that causes SAH.

Antithrombotic therapy is usually given for 4–6 months or until follow-up neuroimaging shows resolution of the dissection. A rare complication of arterial dissection is the formation of a pseudoaneurysm that can become symptomatic causing ischemic stroke or even subarachnoid hemorrhage (SAH).

In conclusion, CAD and VAD are a common cause for stroke in young adults and may result in significant disability and death. The Mercy San Juan Comprehensive Stroke Center and the outpatient Cerebrovascular Disease Clinic have the expertise in offering the best treatment and follow-up for these patients. ■

Clinical Trials in Treatment of Neurological Disorders

Dignity Health Neurological Institute is proud to be a part of multiple multicenter, national and international clinical research trials. As a comprehensive stroke center, it is of the utmost importance to bring the latest advancements to patients. Clinical trials provide an opportunity to improve stroke care, increase quality of life, and decrease mortality. Some of the current trials detect biomarkers of acute ischemic strokes, while others evaluate the effectiveness of medications with neuroprotective properties or of SMART coils on cerebral aneurysms. Another trial evaluates the reduction of intracerebral clots in hemorrhagic strokes using tPA. Through continued participation in these clinical trials, Dignity Health Neurological Institute is helping to discover new preventive care, new treatments for stroke, and to improve overall quality of life for patients.

ACTION 2

Acute Ischemic Stroke Trial for the Investigation of Natalizumab (Action 2) is a multicenter, double-blind, placebo controlled, randomized, parallel group, dose ranging clinical trial with the primary objective of assessing neuroprotective effects of Natalizumab in acute ischemic stroke, and looking at clinical measures of independence and activities of daily living. *Principal investigator: Lucian Maidan, MD*

BASE

Biomarkers of Acute Stroke Etiology (BASE) is a clinical trial that looks at the validity of the clinical use of new biomarker blood tests to identify blood components that may differentiate between diverse stroke etiologies and clinical outcomes. In cases of unknown etiology or cryptogenic strokes, the study evaluates the biomarkers in blood to help determine if the etiology of the stroke is cardioembolic or atheroembolic. *Principal investigator: Lucian Maidan, MD*

MISTIE III

Minimally Invasive Surgery plus rt-PA for ICH Evacuation Phase III (MISTIE III) is a clinical trial that seeks to improve patient long-term outcomes after suffering from an intracerebral hemorrhage by removing the formed clot from the brain through minimally invasive surgery, intermittent dosing of rt-PA and drainage of the broken down clot. The study premise is that by removing the blood clot faster, the injury to the brain will be reduced. *Principal investigator: Alex Nee, MD*

POINT

Platelet-Oriented Inhibition in New TIA and minor ischemic stroke (POINT) is a randomized, double-blind study to evaluate the effectiveness of aspirin and clopidogrel in patients with minor ischemic stroke and high risk TIA over 90 days. Patients will receive aspirin and either a loading dose clopidogrel 600mg or placebo followed by a daily dose for 90 days. *Principal investigator: Lucian Maidan, MD*

DEFUSE 3

Endovascular Therapy following Imaging Evaluation for Ischemic Stroke 3 (DEFUSE 3) is a prospective, randomized, multi-center, Phase III, adaptive, blinded endpoint, controlled trial. Subjects with acute ischemic stroke will be randomized 1:1 and treated between 6 and 16 hours of symptom onset with endovascular therapy plus medical management or medical management alone. *Principal investigator: Lucian Maidan, MD*

SMART

This is a prospective, multicenter registry assessing the embolization of neurovascular lesions using the Penumbra SMART coil system. The new Penumbra SMART Coil not only addresses improvements in the delivery system, but importantly incorporates new technology into the coil implant itself. It enables an individual coil to become progressively softer as it is deployed, utilizes advanced materials to improve stretch resistance, and incorporates new processes to create accurate complex and helical shapes. *Principal investigator: Lucian Maidan, MD*

HEAT

HEAT is a randomized study that compares the effectiveness of new generation hydrogel coated coils versus the bare platinum coils in the endovascular treatment of aneurysms. Hydrogel coils are coated with a biosynthetic polymer that expands in body fluid. The hypothesis is that they will provide the highest degree of occlusion and packing density of the aneurysm, leading to increased protection against recanalization and need for retreatment. *Principal investigator: George Luh, MD*

In addition to inpatient stroke trials, outpatient Dignity Health Neurological Institute trials are underway to evaluate new treatments for multiple sclerosis, epilepsy and headache.

Multiple Sclerosis: OPERA Trail

This randomized, double-blind study is to evaluate the efficacy and safety of Ocrelizumab, a monoclonal antibody targeting B cells, in comparison to Interferon Beta-1a (Rebif) in patients with relapsing multiple sclerosis. Five patients have been enrolled, and we are currently in the follow-up phase. *Principal investigator: John Schafer, MD*

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Telemedicine: A High Tech Solution to Specialty Care Access

John Schafer, MD

Access to specialty care can be a challenge anywhere, but it can be an even greater problem when scarcity of specialists and long travel distances are present, which may occur in rural areas. Telemedicine services are evolving nationwide to provide a solution.

Since 2008 Dignity Health Neurological Institute of Northern California has provided crucial immediate telemedicine consultation to emergency rooms throughout California to quickly evaluate patients for thrombolytic treatment of acute ischemic stroke. This service has filled an enormous gap resulting from the lack of rapid availability of neurologists to the emergency rooms of most hospitals.

Using the technical and organizational resources developed for telestroke service, telemedicine visits for ambulatory care have become possible at Dignity Health facilities in rural areas. Outpatient telemedicine visits have become routine at Dignity Health Clinics in Red Bluff and Grass Valley for multiple sclerosis, movement disorders and neurosurgical visits and are being planned for other areas as well.

This service is generally provided to established patients who have had initial face-to-face evaluations by providers at Sacramento area clinics. Return visits, which usually occur at intervals of four to six months, are scheduled at the distant clinics, saving patients as much as five or six hours of driving. A patient living in the Redding-Red Bluff area goes to the outpatient clinic at St. Elizabeth's Hospital in Red Bluff, and patients in the Nevada County area go to a clinic adjacent to Sierra Nevada Memorial Hospital in Grass Valley. They are registered and roomed by clinic staff in the same way they would be for a face-to-face visit, but the doctor is on the LED screen, almost as if sitting in the room. The audio and visual capabilities of the apparatus are very sophisticated, allowing the doctor to zoom in to examine eye movements or to zoom out and rotate the camera to permit evaluation of walking, for example. Although examination of tendon reflexes and sensation is not possible, the major portions of the examination can be completed just as they would in a face-to-face return visit. In the case of multiple sclerosis care, the major part of every visit is devoted to obtaining the history of new or worsening symptoms, reviewing treatments and ordering tests, all of which can be done as well in a telemedicine consultation as a face-to-face visit.

Currently half-day clinics are scheduled for tele-neurosurgery, tele-MS and tele-movement disorders as well as cardiology. Over 200 multiple sclerosis visits have been conducted over the last several years.


John Schafer, MD

These telemedicine visits differ from Skype and other common online applications not only because the equipment is technically more sophisticated but also because it is HIPAA-compliant. In the future, HIPAA-secure home devices and relaxation of insurance reimbursement restrictions will make telehealth coverage available to even more patients. ■

Clinical Trials in Treatment of Neurological Disorders—continued from page 6

Multiple Sclerosis: CHORDS

This open label study evaluates the effectiveness and safety of Ocrelizumab in patients with relapsing-remitting multiple sclerosis who have had suboptimal course with other disease modifying treatments. *Investigator: Sabeen Lulu, MD*

Multiple Sclerosis: Expanded Access Ocrelizumab

This is an open label expanded access program for Ocrelizumab in patients with primary progressive multiple sclerosis. *Investigator: John Schafer, MD*

Multiple Sclerosis: Ofatumumab

This randomized, double-blind study compares the experimental drug ofatumumab with currently available Teriflunomide in patients with relapsing-remitting multiple sclerosis. *Investigator: Sabeen Lulu, MD*

Epilepsy: ARTEMIS

ARTEMIS is a randomized, double-blind, placebo-controlled study of the safety and efficacy of intranasal midazolam in the outpatient treatment of subjects with seizure clusters. *Principal investigator: Edwin Cruz, MD*

Headache: EVIDERA

This is a prospective observational study to evaluate the tolerability and outcomes of prophylactic therapies in migraine headaches. *Principal investigator: Alan Shatzel, DO* ■

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