Case discussion

Supervisor: 
Dr. Najafi

By: 
Maryam Alizadeh

25 October 2014
The patient is 28 y/o woman Married, Lives in Esfahan, House keeper and is Lactating mothers that presented with true vertigo and headache 4 days before admision

- Vertigo: non positional, continuse
- Headache: mild, non tander, without photophobia and phonophobio, non pulsetile
- Diplopia: vertical
- Ataxia: right side lateropulsion
- RT Side weakness: mild
- N/V

- P.m.hx: negative
- D.hx: negative
- P/E: young woman, alert, obey, orient to TTP
- V/S: stable
- Heart: NL S1, S2
- Lunge: Clear
- Abdomen: Soft
- Skin: NL
C.N: NL

RT eye: petosis, miosis,

Horizontal and rotational nystagmus

Fondoscopy: nl

Reflex: 2+

F.T.N: Rt hand dismetry

M.P: , LT: 5/5   RT: 4/5+

Plantar reflex: RT: up   LT: up

Gait: ataxic

Romberg: falling to Rt side

Sensory: decrease of sense at lt leg
ECG: NSR

ECHO: NL  EF: 60%

Cervical Sono doppler:
Lt vertebral arteries had high flow.
High resistant flow in Rt vertebral artery (vertebral Dissecion should be considered).

Lab data: normal
Vasculitis Tests: ?
Strokes in Young Adults

Similarly, stroke is not an uncommon event in young adults (ages 15 to 45 years), accounting for an estimated 3 percent of cerebral infarctions in typical series. Nevertheless, most of the strokes could be accounted for by three categories, more or less equal in size: (1) atherosclerotic thrombotic infarction (usually with a recognized risk factor); (2) cardiogenic embolism (particularly in the past association with rheumatic heart disease, infective and noninfective endocarditis, paradoxical embolism through patent foramen ovale and other cardiac defects, and prosthetic heart valves); and (3) one of several nonatherosclerotic vasculopathies (arterial trauma, dissection of the carotid artery, moyamoya, lupus erythematosus, drug-induced vasculitis).
Hematologically related disorders-use of oral contraceptives (discussed further on), the postpartum state, and other hypercoagulable states-were the probable causes in 15 percent patients. The presence of antiphospholipid or anticardiolipin antibodies (lupus anticoagulant) explains some of these cases. The majority of these patients are women in their thirties without manifest systemic lupus erythematosus. The frequency of inherited deficiencies of naturally occurring anticoagulant factors as a cause of stroke is low.
Overall, in children and young adults with ischemic stroke, the main diagnoses to be considered are carotid and vertebral dissection, drug abuse (mainly cocaine), thrombosis induced by contraceptive estrogens (see below), antiphospholipid antibody syndrome, and cardiac disease including patent foramen ovale (PFO). Migraine might be added to this list, but it is a diagnosis by exclusion in these circumstances and CADASIL, albeit rare, should also be considered if migraine headaches and TIAs precede a stroke.

Inherited prothrombotic states—such as those caused by the various clotting factor deficiencies discussed above, Fabry disease, moyamoya, and Takayasu arteritis.
Vertebral Artery Dissection

Dissection of these arteries may originate in the neck and extend into the intracranial portion of the vessel or remain isolated to either of these segments as noted below. In both instances there is a tendency to form pseudoaneurysms, mostly with the intracranial type, and in the latter there is a risk of rupture through the adventitia leading to a subarachnoid hemorrhage.

Rapid and extreme rotational movement of the neck is the most common identifiable cause of vertebral artery dissection, as in turning the head to back up a car or with chiropractic manipulation.
Extending the neck to have one's hair washed, swinging a golf club, and direct neck trauma have also been precipitants. Forceful coughing may also cause dissection, as it may in the carotid vessels.

There is no clear female predominance (as there may be in carotid dissection) but the previously cited intrinsic weaknesses of the vascular wall from Ehlers-Danlos disease and fibromuscular dysplasia are risk factors.
The dissection most commonly originates in the Cl-C2 segment of the vessel, where it is mobile but tethered as it leaves the transverse foramen of the axis and turns sharply to enter the cranium. The symptoms, mainly vertigo, are fragments of the lateral medullary syndrome, often with additional features referable to the pons or midbrain, particularly diplopia and dysarthria.

The clinical manifestations in our experience have fluctuated over minutes and hours, quite unlike the usual vertebrobasilar TIA.

Dissection of the vertebral artery was a more common association with "RCVS" than was carotid artery dissection.
Suspected if persistent occipitonuchal pain and vertigo or related medullary symptoms arise following one of the known precipitants—such as chiropractic manipulation of the neck, head trauma, or Valsalva straining or coughing activities—but it may otherwise escape detection until the full-blown medullary or cerebellar stroke is established.

The stroke may follow the inciting event by several days or weeks or even longer, obscuring the relationship.
Axial MRI images, particularly the T1-weighted sequences, show a double lumen in the dissected vessel, as described for carotid artery dissection earlier, and skillful ultrasound investigation documents the same. Some patients will be found to have evidence of spontaneous or traumatic dissection of multiple extracranial vessels; this also occurs as a consequence of dissection of the aortic arch from chest trauma.
No generally agreed upon method has been devised to detect the infrequent instance of subarachnoid hemorrhage from dissection. Lumbar puncture is not routinely performed.

CT is probably adequate for this purpose but it must be acknowledged that it, too, is not often obtained, except in cases of strong suspicion that the dissection has extended into the subarachnoid space, as evidenced by lower cranial-nerve palsies.
Once a stroke has occurred, even though embolic in most cases, prompt reopening of the artery can at times prove beneficial; this is currently performed by endovascular techniques.

Most neurologists take the approach that warfarin, if used, may be discontinued after several months or a year, when angiography or MRA shows the lumen of the carotid artery to be patent, or at least reduced to no more than 50 percent of the normal diameter, and smooth walled.
Despite numerous publications demonstrating the ability of skilled operators to reopen a dissection by endovascular methods, acute intervention has not been studied in a way that allows a judgment regarding its value.

Of both therapeutic and diagnostic value is the relief of pain afforded by corticosteroids in cervical and intracranial dissections, as mentioned earlier. Pseudoaneurysms in the cervical portions of the vessels generally do not require specific treatment;
The study by Mokri and colleagues (1988) reported a complete or excellent recovery in 85 percent of patients with the angiographic signs of cervical artery dissection; mainly, these were patients who had fluctuating ischemic symptoms but without stroke. The outcome in cases complicated by stroke is far less benign. Approximately 25 percent of such patients succumb and most others remain seriously impaired.
If early recanalization of the occluded artery is observed (as determined by ultrasonography), there may also be good functional recovery. Local pseudoaneurysms form in a small proportion of patients and generally do not require surgical repair; they also do not preclude cautious anticoagulation. Subarachnoid hemorrhage from transmural rupture is mostly a complication of vertebral artery dissection.
The lower images show a left vertebral artery dissection (arrows). The T1 hyperintensity that is shown in the left upper and lower images is due to thrombus within the false lumen of the vessel.
Intracranial Arterial Dissection

Dissections of intracranial arteries are less common than extracranial ones and they present in several unusual ways. A number of times we have misinterpreted the arteriographic appearance of a short segment of narrowing of the basilar or proximal middle cerebral arteries, assuming these changes to represent embolism or arteritis when in fact they proved to be dissections of the vessel wall.

In the case of purely intracranial dissection of the middle cerebral or basilar arteries, there is usually no preceding trauma, but a few patients have had minor head injuries, extreme coughing, or other recently Valsalva-producing events (e.g., after childbirth)—or they had used cocaine.
The typical picture is of fluctuating symptoms referable to the affected circulation and severe cranial pain on the side of the occlusion-retroorbital in the case of middle cerebral dissection, occipital in the case of basilar dissection, occipital combined with supraorbital in the case of vertebral dissection. A few patients have had sudden strokes that suggested embolic infarction, and a small number present with subarachnoid hemorrhage.
Treatment of Cervical Artery Dissection

As an overall comment pertaining to dissection, treatment is primarily with anticoagulation for several weeks or months and followed up with some form of arteriography. The choice between aspirin and warfarin has not been clarified as the rate of stroke is low, in the range of 5 percent or less, and remains so with either agent; however, there have not been adequately controlled trials to determine if this is true for large groups of patients.
If the dissection has produced complete occlusion of the vessel, the role of anticoagulation is less clear. Endovascular revascularization has been attempted with mixed results, the main problem being catastrophic and sometimes fatal vessel rupture during angioplasty.
Although there are no data to determine the proper approach to anticoagulation in these circumstances that entail a risk of a subarachnoid hemorrhage, in general we do use heparin and warfarin for a brief period because of the greater concern for embolus, unless there is existing subarachnoid blood on a CT scan or if there is a pseudoaneurysm within the intracranial portion of the dissection.

Some stroke specialists have suggested that a lumbar puncture be performed before initiating anticoagulation but this has not been our practice.
FIGURE 26-7  Right internal carotid dissection in a 12-year-old girl. A, Unenhanced CT shows dense right middle cerebral artery, consistent with thrombosis (arrow). B, CT a day later shows right frontal transcortical infarct (arrows). C, Axial FLAIR MR image on day 8 demonstrates intramural thrombus (arrow) surrounding lumen of internal carotid (arrowhead). D, Coronal oblique MRA shows clot in false lumen (white arrow), slow flow in true lumen (arrowhead), and occlusion of middle cerebral artery (black arrow). E, Diffusion-weighted MRI demonstrates extent of infarct.
Figure 1. Cervical artery dissections. T1 MRI with fat saturation (left) and magnetic resonance angiography (right). The upper images show bilateral internal carotid artery dissections (arrows).
The vertebral arteries are the chief arteries of the medulla; each supplies the lower three-fourths of the pyramid, the medial lemniscus, all or nearly all of the retroolivary (lateral medullary) region, the restiform body, and the posteroinferior part of the cerebellar hemisphere through the posterior inferior cerebellar arteries.

The relative sizes of the vertebral arteries vary considerably, and in approximately 10 percent of cases, one vessel is so small that the other is essentially the only artery of supply to the brainstem.
The posteroinferior cerebellar artery (PICA) is usually a branch of the vertebral artery but can have a common origin and form a loop with the anteroinferior cerebellar artery (AICA) from the basilar artery.
The vertebral arteries are most often occluded by atherothrombosis in their intracranial portion. Because the vertebral arteries have a long extracranial course and pass through the transverse processes of C6 to C1 vertebrae before entering the cranial cavity, one might expect them to be subject to trauma, spondylotic compression, and a variety of other vertebral diseases. With the exception of arterial dissection, in our experience the other causes of vascular occlusion happen only infrequently.
Less often, occlusion of the vertebral artery or one of its medial branches produces an infarct that involves the medullary pyramid, the medial lemniscus, and the emergent hypoglossal fibers; the resultant syndrome consists of a contralateral paralysis of arm and leg (with sparing of the face), contralateral loss of position and vibration sense, and ipsilateral paralysis and later atrophy of the tongue. This is the medial medullary syndrome.
Occlusion of a vertebral artery low in the neck is usually compensated by anastomotic flow to the upper part of the artery via the thyrocervical, deep cervical, and occipital arteries or by reflux from the circle of Willis.
Lateral Medullary Syndrome

Known also as the Wallenberg syndrome, this common stroke is produced by infarction of a wedge of lateral medulla lying posterior to the inferior olivary nucleus. The complete syndrome, comprises (a) symptoms derived from the vestibular nuclei (vertigo, nystagmus, oscillopsia, vomiting); (b) spinothalamic tract (contralateral or, less often, ipsilateral impairment of pain and thermal sense over half the body); (c) descending sympathetic tract (ipsilateral Horner syndrome-miosis, ptosis, decreased sweating); (d) issuing fibers of the ninth and tenth nerves (hoarseness, dysphagia, hiccough, ipsilateral paralysis of the palate and vocal cord, diminished gag reflex);
(e) utricular nucleus (vertical diplopia and illusion of tilting of vision and rotation of the vertical meridian, rarely so severe as to produce upside down vision);
(f) olivocerebellar, spinocerebellar fibers, restiform body and inferior cerebellum (ipsilateral ataxia of limbs, falling or toppling to the ipsilateral side, and the sensation of lateropulsion);
(g) descending tract and nucleus of the fifth nerve (pain, burning, and impaired sensation over ipsilateral half of the face);
(h) nucleus and tractus solitarius (loss of taste); and rarely, (i) cuneate and gracile nuclei (numbness of ipsilateral limbs). Fragmentary syndromes are more frequent, especially at the onset of the stroke.
These subsyndromes may consist of vertigo and ptosis, toppling and vertical diplopia, hoarseness and disequilibrium, or other combinations short of the entire syndrome. While vertigo is the most frequent feature alone, it is not usually an indication of lateral medullary infarction.
The eye signs of lateral medullary infarction are also varied and quite interesting. There is often a fragment of an internuclear ophthalmoplegia or a skew deviation (the globe on the affected side usually being higher). Direction-changing nystagmus (with different head positions) is a useful feature that suggests labyrinthine disease from brainstem forms of nystagmus, but infarction of the vestibular nucleus as part of the lateral medullary syndrome may also produce this sign.
The entire lateral medullary syndrome, one of the most striking in neurology; is almost always caused by infarction, with only a small number of cases being the result of hemorrhage, demyelination, or tumor. Although it has traditionally been attributed to occlusion in the course of the PICA, as mentioned earlier, careful studies have shown that in 8 of 10 cases it is the vertebral artery that is occluded by atherothrombosis; in the remainder, either the posterior inferior cerebellar artery or one of the lateral medullary arteries is occluded. Embolism to the PICA is a less frequent cause. The inferior cerebellum is usually affected, sometimes in isolation if the embolus travels distal to the origin of PICA, causing a cerebellar infarct with attendant vomiting, vertigo, and ataxia often with occipital-nuchal headache.
In recent years, we have had experience with patients who initially have considerable recovery in the first days and weeks, but experience sudden and unexpected death from respiratory or cardiac arrest, even in the absence of cerebellar swelling or basilar artery thrombosis. Cases of this nature have been reviewed by Norrving and Cronqvist. The related and important issue of cerebellar swelling after vertebral artery or PICA occlusion and the need for surgical decompression.
FIGURE 26-4 Bilateral carotid dissection from a motor vehicle accident. A, Left internal carotid artery (white arrow) is markedly narrowed, as is right internal carotid artery (black arrow). B, Sagittal MIP image shows marked narrowing and irregularity of right carotid lumen (arrows). C, Higher slice shows thrombus (white arrow) in right petrous carotid and marked narrowing of left petrous carotid (black arrow).
FIGURE 26-6  Right internal carotid dissection in a 54-year-old man. A, Gadolinium-enhanced MRA. Arrow indicates pseudoaneurysm. B, Axial source image demonstrates true and false (arrow) lumina.
FIGURE 26-5  Left vertebral artery pseudoaneurysm. Coronal MIP image demonstrates outpouching (arrow) from inferior surface of artery at the C1 level.
Thanks for your attention