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Traffic accident with minor cervical lesions but with unexpected medico-legal evolution and consequences: dissection of the internal carotid artery

Monica Sabău[1], Gabriel Mihalache[2,], Camelia Buhas[2]*

Abstract: The authors present the case of a 56 year-old male, without medical history. He was driving his car and had the seatbelt fastened when he suffered a sudden deceleration. Immediately after the accident he felt a left sided laterocervical pain and at the level of the left hemithorax. During the following hours he experienced deglutition disturbances, dysarthria and tongue motility disturbances characteristic to a hypoglossal nerve paralysis. The paraclinical investigations performed subsequently, showed the dissection of the left internal carotid artery in the pharyngeal segment. The neurological treatment and the anticoagulant treatment led to the improvement of the symptoms up to their disappearance. The medico-legal consequence was granting a higher number of medical care days compared to the apparent lack of seriousness of the initial trauma. The conclusion is that minor traumas produced under different circumstances, such as aggressions, traffic accidents etc, can sometimes have an unexpected evolution, leading to severe complications, which have important medico-legal and legal consequences.

Key Words: traffic accident, seatbelt, hypoglossal nerve paralysis, dissection of internal carotid artery.

Some traffic accidents, when the driver and the passengers wear seat belts, result in minor post-traumatic lesions, especially when the deceleration is of low intensity. Traumatic marks such as echymosis can be found on the neck and thoracic area due to seatbelt compression. Also excoriations or possible lesions can be identified on the areas where the body has direct contact with the hard parts of the car's interior. Most often the victims of such traffic accidents do not have internal lesions, but the paraclinical investigations, such as ultrasound, computed tomography (CT), magnetic resonance imaging (MRI) are useful, especially when the patient has subjective complaints of pain in different parts of the body. Shortly after a traffic accident, the patient sometimes presents symptoms of different sufferings. This happens in cases of car accident victims who immediately following the event, state that they have no lesions, they claim feel well etc., and as a result of their statements the accident is reported as a minor collision with no human victims. The seatbelt, in some cases, may be exactly what produces the lesions. We present further such a traffic event, with an unexpected neurological and medico-legal evolution.

CASE PRESENTATION

Fifty-six year-old male, with no significant medical history, while driving his vehicle at night, brakes suddenly at an intersection to avoid a collision with another vehicle. The collision could not be avoided, but it resulted in a low intensity impact. The two drivers decided to settle the accident problem amicably as none

1) University of Oradea, Faculty of Medicine and Pharmacy, Department of Neurosciences and Rehabilitation, Oradea, Romania
2) University of Oradea, Faculty of Medicine and Pharmacy, Department of Morphological Disciplines, Bihor County Forensic Service, Oradea, Bihor, România
** Corresponding author: Assoc. Prof., MD, PhD, University of Oradea, Faculty of Medicine and Pharmacy, Department of Morphological Disciplines, str. Clujului, nr. 50, 410053, Oradea, Bihor, Romania, Tel: +40744566540, E-mail: mygabiart@yahoo.com*

of them showed any obvious lesions, and the material damage was insignificant. The following morning, the 56 years old driver involved in the accident, woke up with deglutition and motility disturbances, tongue edema and dysarthria. He arrives at the hospital, and he is admitted into the emergency room where he is examined by an ENT doctor. The patient does not correlate his condition with the accident, nor does he mention to the doctor that he was involved in a car accident in the hours prior to his arrival at the emergency room. He is diagnosed with "Quincke Edema" and he is treated with antihistamines and analgesics. The symptoms persist and the patient has an unfavorable evolution. He is examined neurologically. A cerebral MRI is recommended and performed. The result shows no pathological changes. The neurologist suspects "Motor Neuron Disease" and recommends an electrophysiological with electromyography examination and a neurography. Even after performing these investigations the patient makes no connection with the suffered trauma, therefore he still does not mention to the attending physician the traffic accident he was involved in. Three days later, that is 11 days after the accident, the patient presents laterocervical pain on the left side, dysarthria, deglutition disturbances, edema and motility disorders at the level of the tongue. The neurological examination reveals tongue edema, tongue paralysis, the tongue being deviated to the right side of the oral cavity and to the left at protrusion. The reinterpretation of the MRI shows the dissection of the left internal carotid artery in the pharyngeal segment, before the internal carotid artery enters the carotid canal. The neurologist brings up the possibility of trauma and only then the patient mentions the traffic accident he was involved in. The paraclinical investigations are repeated, CT scan, an MRI, computed tomography angiography (CTA), brain and cervical magnetic resonance angiography (MRA) are additionally performed, and the suspicion of arterial dissection is confirmed. It is noticed a second degree subadventitial dissection in the pharyngeal segment of the internal carotid artery (ICA) (Figs 1-6). The Doppler examination result of the cervical-cerebral vessels is within normal limits. The patient is started on anticoagulant treatment with Heparin, and vitamin K antagonists. The patient's evolution is favorable. Three weeks from the initial appearance of the symptoms the patient's deglutition disturbance improves, the pain stops, the tongue edema subsides. There is a significant improvement in the tongue motility, but a light hypoglossal paralysis persists on the left side, and a light atrophy installs on the left half of the tongue (Figs 7 and 8).

Meanwhile the patient requested a medico-legal certificate. On the occasion of the examination, the only sign of a traumatic lesion was a very pale yellow-greenish linear bruise, located diagonally on the left lateral side of the neck, pointing towards the presternal region, approximately 12 cm long and 4 cm wide. The patient accused pain in this area and presented the entire neurological symptoms described previously. The events chronology and the diagnosis of the post-traumatic complication resulted from the study of the medical documents presented on the occasion of the medico-legal examination. The conclusions of the medico-legal certificate showed that the lesions were produced by a deceleration mechanism, through seat belt compression; the patient received 35 - 40 days of medical care.

DISCUSSIONS

The dissection of the cervical arteries (DCA) is a relatively frequent condition. The rupture of the arterial wall happens frequently at the level of the tunica media and results in bleeding inside the arterial wall. The intramural hematoma spreads transversally to the intima or to the adventitia. The increase in volume of the intramural hematoma causes compression of the vascular lumen [1].

The extension of the intramural hematoma towards the intima can cause its rupture, the appearance of the intimal fold, and of the double lumen which determines the occlusion/stenosis of that vessel. The lesion of the intima leads to the release of the tissue factors which cause platelet activation and activation of the coagulation cascade, making it possible for local thrombi to be formed, that could either cause microemboli downstream or vascular occlusion. All these mechanisms cause vascular cerebral damage and transient ischemic attacks (TIA) or cerebrovascular accidents (CVA). Sometimes the arterial dissection can even begin at the level of the intima [1, 2].

The extension of the hematoma towards the adventitia can cause a large size intramural hematoma which can lead to compressions of the adjacent structures, can determine the formation of some pseudoaneurysms or even the rupture of the vessel and its bleeding in the adjacent tissues, or in case of intracerebral vessels bleeding in the subarachnoid space [1, 2].

The hematoma can also spread longitudinally along the vessel, proximally and/or distally to the initial lesion location, and an extended dissection on variable lengths of the vessel can occur [1-3].

In the case of the Internal Carotid Artery (ICA), the dissection happens most frequently at the level of the pharyngeal segment, where the vessel is not tied to other structures, the artery being mobile [1-3].

The cervical arteries dissection causes are controversial. There is the sudden dissection and the post-trauma dissection [1-3]. Sometimes, the patients present connective tissue abnormalities, which increase the risk of dissection: Marfan syndrome, Ehlers-Danlos syndrome, fibromuscular dysplasia [4, 5]. In familial cases, with recurring dissections, it has been found to

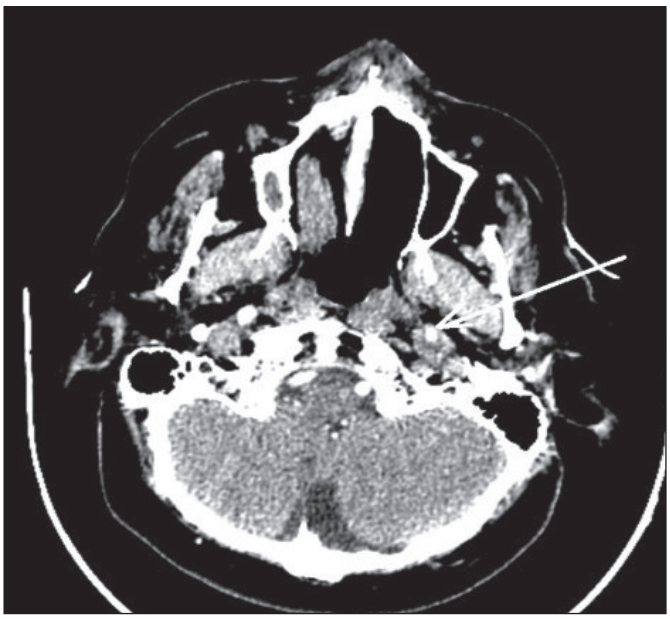

Figure 1. CT without contrast. Emphasizing the increase in size of the ICA due to intramural hematoma and narrowing of the arterial lumen.

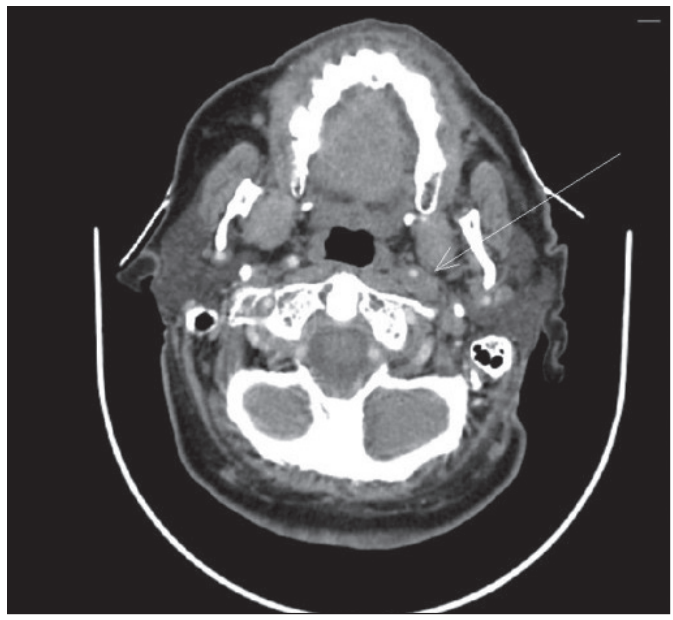

Figure 2. CTA, sequence of acquisition. Narrowing of the arterial lumen and intramural hematoma.

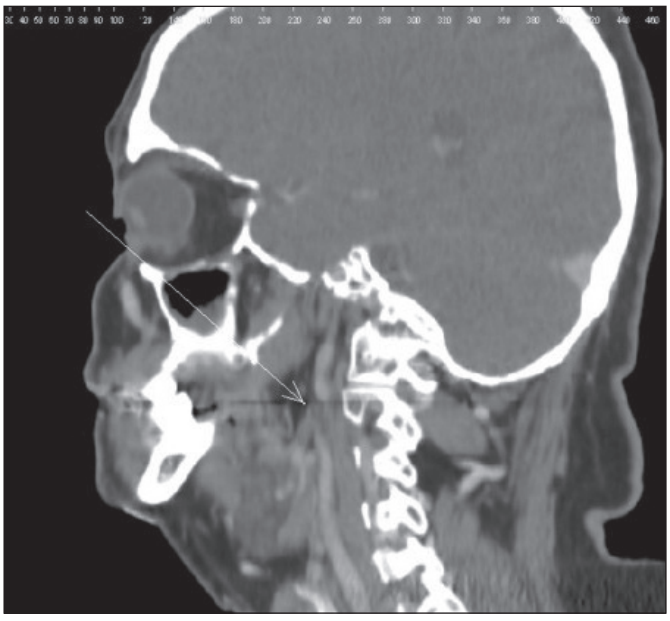

Figure 3. CTA, reconstruction. Emphasizing the stenosis obtained by dissection, with "flute beak" aspect.

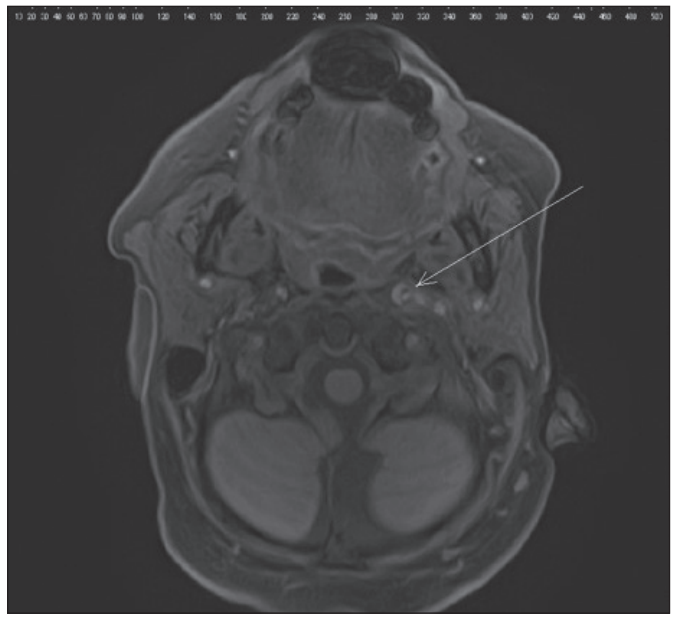

Figure 4. MRI FLAIR secquences. Semilunar aspect of the intramural hematoma.

have α1- antitrypsin deficiency [6, 7]. Other genetic mutations affecting the stability of the vascular wall have been noticed as well [8]. A significant connection between migraines and arterial dissection has been identified, possibly due to alterations of the vascular wall, caused in the case of migraines by repeated episodes of the vascular wall edema [9, 10]. It has also been noticed a connection between the arterial dissections and the presence of some infections [11].

The post-traumatic dissection may appear after minor traumas: stretches, sudden or exaggerated rotations during some home activities or minor accidents [2, 12], after chiropractic maneuvers [13-17], medical maneuvers [18-22], paroxysmal cough [23], vomiting [24], or extended forced positions [25-27]. The traffic accidents are by far the most frequent cause of arterial dissections [28, 32]. Most often the arterial dissections are due to deceleration during accidents [33, 34]. The seatbelt can have a decisive role in the accident's outcome [35]. The diagnosis is sometimes difficult, as the patients have no traumatic signs and the post traumatic neurological signs are not present immediately [36]. Considering the severity of the symptoms and the risks of the patients with such post-traumatic lesions, specific screening procedures were implemented for patients at risk of cervical arteries dissection [37].

The symptoms of arterial dissections include signs correlated directly to the impairment of the vascular

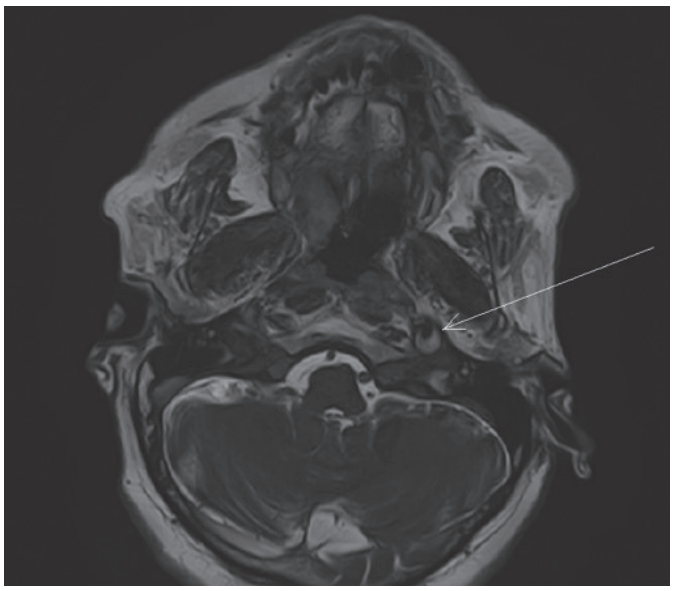

Figure 5. MRI TOF sequences T2. Semilunar aspect of the intramural hematoma at dissection level.

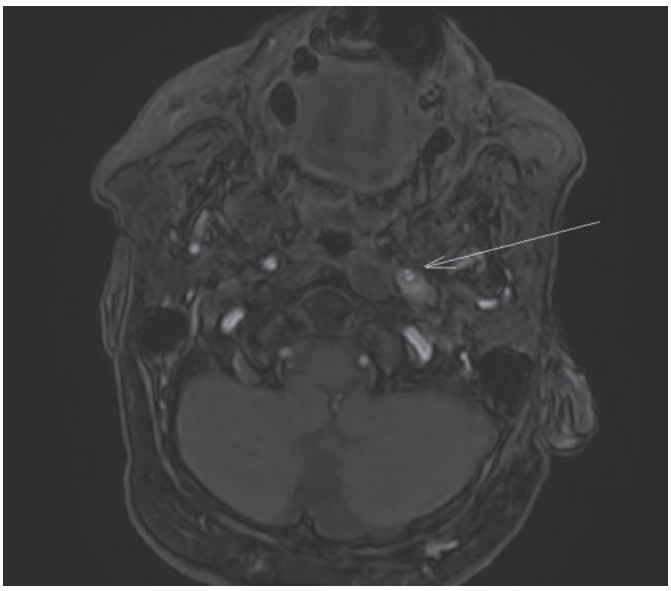

Figure 6. MRI sequences TOF. Intramural hematoma at dissection level and narrowing of arterial lumen.

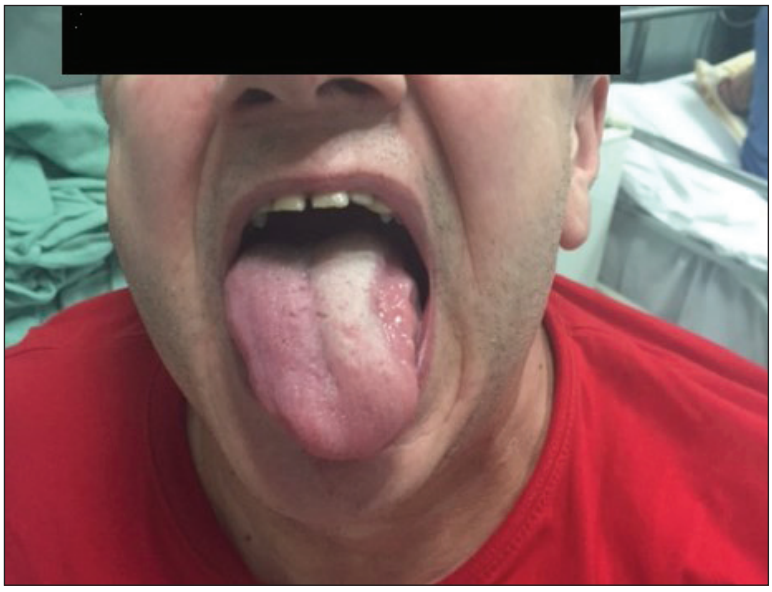

Figure 7. Tongue deviation towards the left with small protrusion and light atrophy of the left half of the tongue.

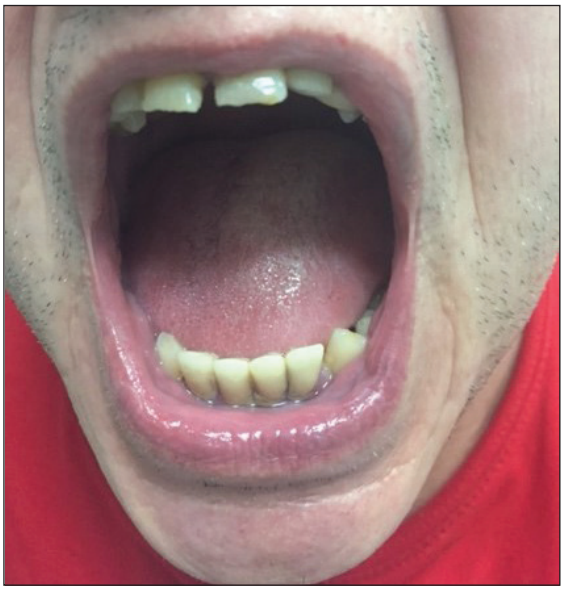

Figure 8. Tongue deviation towards the right inside the oral cavity.

wall (laterocervical pain, facial pain or headache with suggestive characteristics) signs due to the impairment of the vascular lumen (cerebral ischemia manifestation, transient ischemic attacks (TIA) or cerebrovascular accidents (CVA), and possible signs due to the expansion of the vessel with the compression of the adjacent structures (paralysis of the inferior cranial nerves and Claude-Bernard Horner syndrome) [1-3, 38, 39].

The most frequent symptom is the pain, because the cervical vessels have a rich network of nervous fibers sensitive to pain. The pain can be cervical pain, pain located at the level of the face or headache. The headache can be generalized, but usually it begins at the level of the dissection. The pain usually precedes the other clinical symptoms. The time frame before the appearance of other symptoms varies from a few minutes, to hours, days, or weeks [40, 41].

The cerebrovascular accidents are the most severe category of symptoms subsequent to arterial dissection [1-3]. The cerebral ischemic manifestations can be the result of vascular stenosis/occlusion, but most frequently they are the result of microembolisms at the level of endothelial lesion subsequent to dissection [42, 43]. The extended cortical strokes [44, 45], happen most frequently, but small asymptomatic strokes may also occur [43].

The dissections which spread towards the adventitia frequently cause paralysis of the inferior cranial nerves [46, 50] and the impairment of the pericarotid sympathetic plexus [51].

The paralysis of the hypoglossal nerve in the internal carotid artery dissection (ICAD), although rare, is already well known and described in the literature. [52 - 63] The frequency of secondary hypoglossal nerve paralysis in the case of internal carotid artery dissection

(ICAD) is estimated at 10% of the ICAD cases [46, 47].

The tongue edema is explained by the liquid flowing from the intracellular space to the extracellular space in case of tongue denervation [64].

In the presented case there was a trauma due to traffic accident consisting in a sudden deceleration, possibly aggravated by the patient wearing the seat belt. The lesion of the arterial wall was immediately felt by the patient as a left lateral cervical pain. The expansion of the intramural hematoma led to the appearance of tongue edema and paralysis by morning. In this case the time frame between the minor trauma, practically neglected by the patient and unknown by the medical services at the moment, and the appearance of the symptoms was not long. The appearance of pain, followed by the hypoglossal nerve paralysis and tongue edema is very suggestive for the diagnosis. Unfortunately, many cases are misinterpreted as infections [52, 57, 63, 64] or tumors [57, 61, 63].

The diagnosis was suggested by the clinical aspect, the time connection between the trauma and the appearance of the clinical signs, and sustained by imaging examinations CT, CTA, MRI and MRA. Angiography was not performed as the patient was stable, the evolution was favorable and the diagnostic elements obtained by other methods were sufficient [65-67].

The Doppler examination was within normal parameters, due to the fact that despite the vascular lumen stenosis, the flow was maintained. The lesion area was not visible for examination as it was located exactly at the entrance of the internal carotid artery (ICA) into the carotid canal. The Doppler examination brings important diagnostic elements when in the case of a sub-intimal dissection that causes significant modifications of the vascular flow and possibly intimal flap, or arterial occlusion [68-70].

Anticoagulant treatment with heparin was administered, followed by vitamin K antagonists, with the patient's INR under observation. The results with aspirin and anticoagulants seem to be similar [71-73].

The evolution continued to be favourable, with the complete restoration of the arterial wall, but it could have been possible for complications to appear: cerebral ischemia manifestation by spreading of the lesions at the level of the intima or the appearance of a pseudoaneurysm on the segment where [74] the intramural hematoma was initially formed. The patient was kept under observation up to full recovery.

The lesions caused by post-cervical contusions are relatively frequent in the medico-legal pathology [75-78]. The ICA dissection following a minor traffic accident, such as the presented one, is a rare case. It should be emphasized once again that the arterial lesions are frequently unrecognized in the initial phase due to the diversity of the clinical signs and the different degrees of severity, from a simple pain, up to lesions of the cranial nerves, as in the previously mentioned case, or up to severe vascular accidents in other cases described in the medical literature. The sudden deceleration causes a brutal flexion followed by a brutal extension of the ICA. The presence of the seat belt increases the risk of some contusional lesions at the level of the neck. The trauma lesions are absent or minimal: a minor contusion at the level of the neck. Sometimes, the actual collision between vehicles may not occur, and the deceleration due to the sudden brake remains the sole traumatic agent. The presented case is very rare; this explains the failure to diagnose the patient on the initial examinations. This particular case and its diagnostics investigations are found in the medical literature. The persistence of the symptoms, imaging examinations, lack of previous vascular lesions, and presence of a contusion type of cervical trauma due to sudden deceleration associated with wearing a seat belt, and the chronological criteria makes it possible to identify the cause-effect mechanism between the traffic accident and the appearance of the arterial lesions.

CONCLUSIONS

1. A traffic accident of low intensity may cause internal lesions which manifest as a complex neurological picture. The correct anamnesis can direct the clinician to correlate the minor trauma the patient has suffered with the subsequent symptoms.

2. The highly accurate paraclinical investigations (especially the MRI with contrast agent and the CT scan) establishes the diagnosis of the lesion, in this particular case the dissection of the internal carotid artery, which caused the neurological condition.

3. The external traumatic lesions due to traffic accidents can be minimal or even unobservable. This does not exclude the presence of lesions to the internal organs, vessels or nerves.

4. The interdisciplinary cooperation between the forensic physician and neurologist in this case led to issuing a medico-legal document that offered an objective answer, in accordance with the medical facts, to the legal matter of the case. Essentially, a connection was established between the traffic accident, the lesions suffered by the patient, and the neurological complications of these lesions.

Conflict of interest. The authors declare that there is no conflict of interest.

References

1. Caplan R.L: Dissections of brain-supplying arteries. Nature Clinical Practice. 2008;4(1):34-42.
2. Blum A.C, Yaghi S. Cervical Artery Dissection: A Review of the Epidemiology, Pathophysiology, Treatment, and Outcome. Arch Neurosci. 2015; 2(4):2-7.

3. Baumagartner RW, Bogousslavsky J. Clinical manifestations of carotid dissection. Front Neurol Neurosci. 2005;20:70-76. Review.
4. Schievink WI, Wijdicks EF, Michels VV, Vockley J, Godfrey M. Heritable connective tissue disorders in cervical artery dissections: a prospective study. Neurology. 1998;50(4):1166-1169.
5. Brandt T, Hausser I, Orberk E, Grau A, Hartschuh W, Anton-Lamprecht I, Hacke W. Ultrastructural connective tissue abnormalities in patients with spontaneous cervicocerebral artery dissections. Ann Neurol. 1998;44(2):281-285.
6. Schievink WI, Prakash UB, Piepgras DG, Mokri B. Alpha 1-antitrypsin deficiency in intracranial aneurysms and cervical artery dissections. Lancet. 1994;343(8895):452-453.
7. Martin JJ, Hausser I, Lyrer P, Busse O, Schwarz R, Schneider R, Brandt T, Kloss M, Schwaninger M, Engelter S, Grond-Ginsbach C. Familial cervical artery dissections: clinical, morphologic, and genetic studies. Stroke. 2006;37(12):2924-2929. Epub 2006 Oct 19.
8. Pezzini A, Del Zotto E, Archetti S, Negrini R, Bani P, Albertini A, Grassi M, Assanelli D, Gasparotti R, Vignolo LA, Magoni M, Padovani A. Plasma Homocysteine Concentration, C677T MTHFR Genotype, and 844ins68bp CBS Genotype in Young Adults With Spontaneous Cervical Artery Dissection and Atherothrombotic Stroke. Stroke. 2002;33(3):664-669.
9. D'Anglejan-Chatillon J, Ribeiro V, Mas JL, Youl BD, Bousser MG. Migraine—a risk factor for dissection of cervical arteries. Headache. 1989;29(9):560–561.
10. Tzourio C, Benslamia L, Guillon B, Aïdi S, Bertrand M, Berthet K, Bousser MG. Migraine and the risk of cervical artery dissection: a case–control study. Neurology. 2002;59(3):435-437.
11. Guillon B, Berthet K, Benslamia L, Bertrand M, Bousser MG, Tzourio C. Infection and the risk of spontaneous cervical artery dissection: a case-control study. Stroke 2003;34:e79 - 81.
12. Mokri B. Traumatic and spontaneous extracranial internal carotid artery dissections. J Neurol 1990;237:356-361.
13. Lansley MJ. Cervicocephalic artery dissections and chiroprac - tic manipulations. Lancet 1993;341:1214.
14. Lee KP, Carlini WG, McCormick GF, Albers GW. Neurologic complications following chiropractic manipulation: A survey of California neurologists. Neurology 1995;45:1213-1215.
15. Hufnagel A, Hammers A, Schonle PW, Bohm KD, Leonhardt G. Stroke following chiropractic manipulation of the cervical spine. J Neurol 1999;246:683-688.
16. Rothwell DM, Bondy SJ, Williams JI. Chiropractic manipulation and stroke: a population-based case-control study. Stroke 2001;32:1054-1060.
17. Haneline M, Lewkovich G. Identification of internal carotid artery dissection in chiropractic practice. J Can Chiropr Assoc 2004;48(3): 206-210.
18. Tettenborn B, Caplan LR, Sloan MA, Estol CJ, Pessin MS, DeWitt LD, Haley C, Price TR. Postoperative brainstem and cerebellar infarcts. Neurology. 1993;43:471-477.
19. Gould DB, Cuningham K. Internal Carotid Artery Dissection After Remote Surgery. Iatrogenic Complication of Anesthesia. Stroke. 1994;25:1276-1278.
20. Ricchetti A, Becker M, Dulguerov P. Internal carotid artery dissection following rigid esophagoscopy. Arch Otolaryngol Head Neck Surg. 1999;125(7):805-807.
21. Testai DF, Gorelick BP. An Unusual Cause of Vertebral Artery Dissection. Stroke Res Treat. 2010;2010. pii: 915484.
22. Ringrose T, Thompson W. An unusual case of postoperative nausea, vomiting and neck pain. Crit Care Resusc. 1999;1(3):288-290.
23. Skorowronski MD, Buxton AJ, Hestrin M, Keyes R, Lynch K, Halperin SA. Carotid artery Dissection as Possible Severe Complication of Pertusis in an Adult: Case report and Review. Clin Infect Dis. 2003;36(1):e1-4. Epub 2002 Dec 16.
24. Kumar DS, Kumar V, Kaye W. Bilateral internal carotid artery dissection from vomiting. Am J Emerg Med. 1998;16(7):669-670.
25. Machado DM, Gomez RS, Gomez RS. Vertebrobasilar ischemia after a dental procedure. J Oral and Maxillofac Surg. 1999;57(12):1463-1465.
26. Shnobha N, Bhatia R, Barber PA. Dental procedures and stroke: a case of vertebral artery dissection. J Can Dent Assoc 2010;76:a82
27. Mourad JJ , Girerd X, Safar M. Carotid artery dissection after prolonged telephone call. N Engl J Med. 1997;336(7):516.
28. Haneline M, Triano J. Cervical Artery Dissection. A comparison of Highly Dynamic Mechanisms: manipulation *versus* Motor vehicle Collision. J Manipulative Physiol Ther. 2005;28(1):57-63. Review.
29. Janjua KJ, Goswami V, Sagar G. Whiplash injury associated with acute bilateral internal carotid arterial dissection. J Trauma 1996;40: 456-458.
30. Beaudry M, Spence JD. Motor vehicle accidents: the most common cause of traumatic vertebrobasilar ischemia. Can J NeurolSci 2003;30(4): 320-325.
31. Yang ST, Huang YC, Chuang CC, Hsu PW. Traumatic internal carotid artery dissection. J ClinNeurosci 2006;13:123-128.
32. Uhrenholt L, Freeman MD, Webb AL, Pedersen M, BoelLW. Fatal subarachnoid hemorrhage associated with internal carotid artery dissection resulting from whiplash trauma. Forensic Sci Med Pathol. 2015;11(4):564-569.
33. Patel NN, Wang SC. Carotid and vertebral artery dissection due to deceleration injury in a motor vehicle crash. J Trauma. 2001;51(4):818.
34. Srivastava A, Bradley M, Kelly M. Bilateral Carotid Artery Dissection after High Impact Road Traffic Accident. J Radiol Case Rep. 2008; 2(5): 23–28.
35. DiPerna CA, Rowe VL, Terramani TT, Salim A, Hood DB, Velmahos GC, Weaver FA. Clinical importance of the seat belt sign in blunt trauma to the neck. Am Surg, 68 (2002), pp. 441–445
36. Carrillo EH, Osborne DL, Spain DA, Miller FB, Senler SO, Richardson JD. Blunt carotid artery injuries: difficulties with the diagnosis prior to neurologic event. J Trauma. 1999;46(6):1120-1125.
37. Carillo Kerwin AJ, Bynoe RP, Murray J, Hudson ER, Close TP, Gifford RR, Carson KW, Smith LP, Bell RM. Liberalised screening for blunt carotid and vertebral artery injuries is justified. J Trauma, 51 (2001), pp. 308–314.
38. Biousse V, D'Anglejan-Chatillon J, Touboul PJ, Amarenco P, Bousser MG. Time course of symptoms in extracranial carotid artery dissections: a series of 80 patients. Stroke 1995; 26(2): 235–239.
39. De Bray JM, Baumgartner RW. History of spontaneous dissection of the cervical carotid artery. Arch Neurol 2005;62:1168-1170.
40. Silbert PL, Mokri B, Schievink WI. Headache and neck pain in spontaneous internal carotid and vertebral artery dissections. Neurology 1995; 45(8): 1517–1522.
41. Campos CR, Calderaro M, Scaff M, Conforto AB. Primary headaches and painful spontaneous cervical artery dissection. J Headache Pain 2007;8:180-184.
42. Baumgartner RW, Arnold M, Baumgartner I, Mosso M, Gönner F, Studer A, Schroth G, Schuknecht B, Sturzenegger M. Carotid dissection with and without ischemic events: local symptoms and cerebral artery findings. Neurology 2001;57:827-883.

43. Cosottini M, Michelassi MC, Puglioli M, Lazzarotti G, Orlandi G, Marconi F, Parenti G, Bartolozzi C. Silent cerebral ischemia detected with diffusion-weighted imaging in patients treated with protected and unprotected carotid artery stenting. Stroke 2005;36:2389-2393.
44. Weiller C, Müllges W, Ringelstein EB, Buell U, Reiche W. Patterns of brain infarctions in internal carotid artery dissections. Neurosurg Rev. 1991;14:111–113.
45. Steinke W, Schwartz A, Hennerici M. Topography of cerebral infarction associated with carotid artery dissection. J Neurol. 1996;243:323–328.
46. Mokri B, Silbert PL, Schievink WI, Piepgras DG. Cranial nerve palsy in spontaneous dissection of the extracranial internalcarotid artery. Neurology 1996; 46: 356–359.
47. Sturzenegger M, Huber P. Cranial nerve palsies in spontaneous carotidartery dissection. J NeurolNeurosurg Psychiatry 1993;56:1191-1199.
48. Panisset M, Eidelman BH. Multiple cranial neuropathy as a feature of internal carotid artery dissection. Stroke 1990;21:141-147.
49. Vieira VL, Pereira DC, Ribeiro VT, Leite AB, Emerique I. Spontaneous internal carotid artery dissection with paralysis of lower cranial nerves: case report [Portuguese]. ArqNeuropsiquiatr 2006;64:1047-1049.
50. Heckmann JG, Tomandl B, Duhm C, Stefan H, Neundörfer B. Collet-Sicard syndrome due to coiling and dissection of the internal carotid artery. Cerebrovasc Dis 2000;10:487-488.
51. de Bray JM, Baumgartner R, Guillon B, Pautot V, Dziewas R, Ringelstein EB, Sturzenegger M, Garnier P, Ducrocq X, Saudeau D, Neau JP, Larrue V, Vuillier F, Boulliat J, Verret JM, Verny C, Dubas F. Isolated Horner's syndrome may herald stroke. Cerebrovasc Dis 2005;19:274-275.
52. Freilinger T, Heuck A, Strupp M, Jund R. Hypoglossal nerve palsy due to internal carotid artery dissection. Vasc Med 2010; 15: 435.
53. Urseakar MA, Singhal, BS, Konin, BL. Hypoglossal nerve palsy du to spontaneous dissection of the internal carotid artery. ClinRadiol 2000;978-979.
54. Shahab R, Savy LE, Croft CB, Hung T. Isolated hipoglossal nerve palsy due to internal carotid artery dissection. J LaryngolOtol 2001;115:587-589.
55. Lieschke GJ, Davis S, Tress BM, Ebeling P. Spontaneous internal carotid artery dissection presenting as hypoglossal nerve palsy. Stroke 1988;19:1151-1155.
56. Marin LF, Bichetti DB, Felicio AC, dos santos WAC, Oliveira FF, Morita ME, Avelar WM, Braga-Neto P, dos Santos Lima EC, Martins RJ: Hippoglosal nerve palsy as the sole manifestation of internal carotid artery dissection. Arqneuropsiquiatri 2009; 67(1):107-108.
57. Olzowy B, Lorenz S, Guerkov R. Bilateral and unilateral internal carotid artery dissection causing isolated hypoglossal nerve palsy: a case report and review of the literature. Eur Arch Otorhinolaryngol 2006; 263: 390–393.
58. Boukobza M, Ast G, Reizine D, Merland JJ. Internal carotid artery dissection causes hypoglossal nerve palsy: CT, MRI and angiographic findings. J Neuroimaging. 1998;8(4):244-6.
59. Lindsay FW, Mullin D, Keefe MA. Subacute hypoglossal nerve paresis with internal carotid artery dissection. Laryngoscope 2009;113(9):153-1533.
60. Verdalle P, Herve S, Kossowski M, Felten D, Courtois A, Garcia D. Spontanous dissection of the internal carotid artery in its extracranial portion, revealed by a hypoglossal paralysis: report of four cases. Ann OtolRhinolLaryngol 2001; 110:794–798.
61. Ahmad A, Baghomian A, Travis P, Doran M. Internal Carotid Artery dissection presenting as Isolated Hippoglosal nerve palsy. Acute Madicine 2009;8(1):22-25.
62. Spitzer C, Mull M, Töpper R. Isolated hypoglossal nerve palsy caused by carotid artery dissection—the necessity of MRI for diagnosis. J Neurol. 2001;248(10):909-910.
63. Riancho J, Infacnte J, Mateo JI, Berciano J. Unilateral isolated hypoglossal nerve palsy associated with intwrnal carotid artery dissection.J Neurol Neurosurg Psychiatry. 2013;84(6):706.
64. Nair GD, Borja JM. Tongue: Tongue swelling as a sign of internalcarotid artery dissection. Neurology and Clinical Neuroscience 2015;3: 246–247.
65. Chen CJ, Tseng YC, Lee TH, Hsu HL, See LC. Multisection CT angiography compared with catheter angiography in diagnosing vertebral artery dissection. AJNR Am J Neuroradiol 2004; 25(5): 769–774.
66. Taschner CA, Leclerc X, Lucas C, Pruvo JP. Computed tomography angiography for the evaluation of carotid artery dissections. In Bawngartner RW, Bogousslavsky J, Caso V, Paciaroni M (eds): Handbook on cerebral artery dissection. Front NeurolNeurosci. Basel: Karger 2005;20:119-128.
67. Paciaroni M, Caso V, Agnelli G. Magnetic resonance imaging, magnetic resonance and catheter angiography for diagnosis of cervical artery dissection. In Bawngartner RW, Bogousslavsky J, Caso V, Paciaroni M (eds): Handbook on cerebral artery dissection. Front NeurolNeurosci Basel: Karger 2005;20:102-118.
68. Benninger DH, Caso V, Baumgartner RW. Ultrasound assessment of cervical artery dissection. In Baumgartner RW, Bogousslavsky J, Caso V, Paciaroni M (eds): Handbook on cerebral artery dissection. Front NeurolNeurosci. Basel: Karger 2005;20:87-101.
69. Sturzenegger M, Mattle HP, Rivoir A, Baumgartner RW. Ultrasound findings in carotid artery dissection: analysis of 43 patients. Neurology 1995; 45 (4): 691–698.
70. Benninger DH, Caso V, Baumgartner RW. Ultrasound assessment of cervical artery dissection. Front Neurol Neurosci 2005; 20: 87–101.
71. Baumgartner RW. Stroke prevention and treatment in patients with spontaneous carotid dissection. ActaNeurochir 2008(Suppl);103:S47-S50.
72. Beletsky V, Nadareishvili Z, Lynch J, Shuaib A, Woolfenden A, Norris JW. Cervical arterial dissection: time for a therapeutic trial? Stroke 2003;34:2856-2860.
73. The CADISS trial investigators* Antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS): a randomised trial. Lancet Neurol2015; 14: 361–67.
74. Touzé E, Gauvrit JY, Meder JF, Mas JL. Prognosis in cervical artery dissection. In: Bawngartner RW, Bogousslavsky J, Caso V, Paciaroni M (eds): Handbook on cerebral artery dissection. Front NeurolNeurosci Basel: Karger 2005;20:129-139.
75. Marjaei A. Post Traumatic dissection of vertebral artery: A case report. International Journal of Medical Toxicology and Forensic Medicine 2011(1):30-32.
76. Perotti S, Bin P. Vertebral injury and third-party liability: the wrongful attack to whipalsh injury. Prevention and research 2013;2(1):37-39.
77. Dermengiu D. Forensic pathology. Romanian Medical Life Publishing: București; 2002.
78. Mihalache G, Buhas C. Compendium of forensic medicine for general practitioners and dentists. Publisher University of Oradea. 2012.