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CASE REPORT

L. Pötsch · J. Bohl

Traumatic lesion of the extracranial vertebral artery – a note-worthy potentially lethal injury

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Abstract The autopsy findings from routine neuropathological investigations of the cervical spine after any history of trauma emphasized the vulnerability of the extracranial vertebral arteries. In 21 cases with trauma to the head and neck, normal autopsy procedures did not succeed in revealing an obvious cause of death. Traumatic lesions of the spinal cord such as contusion or neurorrhexis were seen in 10 cases. In 15 cases we observed different degrees and stages of traumatic lesions of the extracranial vertebral arteries. Sudden death due to acute brain stem ischemia might be considered as an explanation in some of these cases. Six case reports with traumatic vertebral artery (VA) lesions after severe or minor extracranial trauma are presented. A review of the literature revealed that this vascular injury has only rarely been directly established by gross pathological examination. Possible consequences of blunt trauma to the head and neck include angiorrhexis, subintimal, intramural and perivascular hemorrhage leading to a partial narrowing of the injured vessel. These processes can be complicated by thrombosis. Unilateral VA obliteration may be an occult risk of acute brain stem ischemia when associated with contralateral reduction of the posterior circulation.

Key words Vertebral artery · Traumatic lesion
Craniocervical trauma · Brain stem ischemia
Vertebrobasilar insufficiency

Zusammenfassung Nach einer Traumatisierung erfolgte regelmäßig die Untersuchung der anatomischen Strukturen der Halswirbelsäule unter Einbeziehung neuropathologischer Befunde am zentralen Nervensystem. Die erhöhte Vulnerabilität der extrakraniellen Vertebralarterien ließ sich bestätigen. Bisher liegen 21 Fälle nach Traumatisierung vor, bei denen die vorgeschriebene Öffnung der drei Körperhöhlen bei der Obduktion und die histologische Untersuchung zunächst keine eindeutige Todesursache erbrachte. Die vorgefundenen Verletzungen und mikroskopischen Befunde waren weder geeignet, den Tod unmittelbar herbeizuführen, noch die medizinische Todesursache zu erklären. Bei den erweiterten Untersuchungen an der Halswirbelsäule waren Autopsiebefunde zu erheben, die für den Eintritt des Todes als bedeutsam zu diskutieren sind. In 15 Fällen wurden Verletzungen der extrakraniellen Vertebralarterien festgestellt. Traumatische Läsionen des Rückenmarks und/oder der Nervenwurzeln lagen in 10 Fällen vor. In 11 Fällen wurde nach Ausschluß anderer Todesursachen unter Berücksichtigung individueller anatomischer Gegebenheiten eine akute Hirnstammischämie infolge traumatischer Vertebralarterienverletzung angenommen. Es werden 6 Fälle berichtet mit einer traumatischen Verletzung der extrakraniellen Arteria vertebralis. Sowohl nach schweren HWS-Traumen als auch bei leichten Traumatisierungen kann es zu einer Gefäßläsion kommen, in deren Folge die verletzte Arteria vertebralis obliteriert. Die Thrombosierung einer Arteria vertebralis im extrakraniellen Abschnitt kann weitgehend durch das kontralaterale Gefäß kompensiert werden. Eine unilaterale Thromboocclusion stellt jedoch ein latentes Risiko für eine akute Hirnstammischämie dar, wenn kontralateral eine kritische Reduktion der vertebrobasilären Versorgung auftritt. Zur Abklärung der eigentlichen Todesursache und für die Beurteilung der Kausalität zwischen äußerer Gewalteinwirkung und Todeseintritt werden bei einem Trauma in der Vorgeschichte und beim Fehlen von Befunden, die den Tod zwanglos erklären können, erweiterte Untersuchungen an der Halswirbelsäule als unverzichtbar angesehen.

Schlüsselwörter A. vertebralis · Verletzung · Trauma
Halswirbelsäule · Hirnstammischämie · Vertebrobasiläre
Insuffizienz

L. Pötsch (✉)
Institut für Rechtsmedizin,
Johannes-Gutenberg-Universität Mainz, Am Pulverturm 3,
D-55131 Mainz, Deutschland

J. Bohl
Abteilung Neuropathologie,
Pathologisches Institut Universitätsklinikum,
D-55131 Mainz, Deutschland

Introduction

Traumatic cervical and extracranial vascular injuries are relatively uncommon but not rare. In recent years a significant number of vertebral injuries have been reported after neuroradiological investigations of vascular trauma [8, 11, 13, 15, 17, 18, 21, 30–32, 40, 46, 51, 54, 56, 58, 62, 71, 75, 82, 93]. The exact incidence of extracranial vertebral artery (VA) injury is unknown. Saternus [84, 85] and Saternus and Burscheidt [86] assumed a high incidence of post-traumatic extracranial VA injuries and found VA lesions in 52% of unselected autopsy cases after acceleration trauma. In these studies it was also pointed out that even mild nonphysiological strains on the neck can lead to multiple injuries of the VA. There are only a few autopsy reports of lethal cases that emphasize the significance of intracranial VA lesions, embolism or subarachnoid hemorrhage [10, 19, 48, 50, 59, 62, 63, 70, 80, 84–86, 88, 89, 105]. Pathology findings sometimes showed thrombosis and/or embolism of the basilar artery and/or cerebellar arteries as well as repeated infarcts but unfortunately the starting point of thrombus or emboli formation was not determined since VAs were not examined proximal to their dural entrance in most of the cases. Reviewing the literature revealed that pathological post mortem findings of extracranial VA lesions associated with the cause of death are rare. One of the reasons may be the difficult and time-consuming dissection technique of the extracranial VAs that is required and exceeds all routine autopsy practice. Another probable reason is that the onset of clinical symptoms may be frequently delayed or even absent. In recent years increasing attention has been paid to this problem and dissection of extracranial VAs has led to remarkable results in some cases [10, 59, 62, 63, 69, 80, 88, 89, 93, 104, 105]. Subsequent to the observations by Saternus we examined the VA, cervical spine and spinal cord regularly in unclear causes of death with severe or minor trauma to the head and neck.

Materials and methods

We have investigated 21 cases with known history of trauma in which the normal autopsy procedure had not succeeded in revealing an obvious cause of death. In 15 cases we observed different degrees and stages of traumatic lesions of the extracranial arteries (Table 1). Traumatic lesions of the spinal cord such as contusion or neurorrhexis were seen in 10 cases. In a number of cases unexpected sudden death raised the suspicion of an acute ischemia of the brain stem. By exclusion of other cause of sudden death and the consideration of the individual anatomical structures we concluded acute brain stem ischemia to be the cause of death in 11 of the cases. The importance of detailed neuropathological examination of the cervical spine and the potentially lethal consequences resulting from blunt closed trauma to the extracranial VAs are illustrated by 6 case reports. Only pathology findings are listed. Inconspicious autopsy findings including neuropathological investigation of the brain, macroscopically and microscopically evaluated, are not mentioned. The spinal cord, ganglions, nerve roots, peripheral nervous tissues as well as soft tissues, although macroscopically normal, were routinely investigated. Additional interest was focused on microscopical findings that could be considered as predisposing factors or to cause the VA lesion. The survival times of cases 1–3 are estimated by the extent of autopsy findings known to be signs of vital reactions.

Table 1 Autopsy cases with lesions of the extracranial vertebral arteries ($n = 15$) after history of trauma. Normal autopsy procedure had not succeeded in revealing an obvious cause of death

	Number of cases
Traumatic event	
Traffic accident	10
Accident at work	3
Accident at home	1
Iatrogenic lesion	1
Time interval between trauma and death	
Immediate death < 5 min (estimation)	5
Hours	2
Days	7
More than one month	1
Traumatic lesions of the extracranial VAs	
Total disruption	1
Partial disruption of the vessel wall with intramural hematoma and thrombosis (e.g. dissecting aneurysms)	7
Intimal lesion with thrombosis	2
Adventitial hemorrhages and effusion of blood into the surrounding soft tissue	3
Partial disruption with well organised and partial recanalized thrombosis	1
Formation of fusiform aneurysm with partially obliterating thrombosis	1

Case reports

Case 1: an 18-year-old healthy female was involved in a traffic accident as a pedestrian. Medico-legal aspect: emergency ambulance arrived 20 min after being reported. Could sufficient intensive care or early admission to hospital have prevented death? General pathology findings: polytrauma – multiple fractures of both lower extremities – rib fractures (3–5 right side) – fractures of the pelvic girdle. Neuropathological examination: severe trauma to cervical spine with destruction and hemorrhage in the upper cervical cord – dislocated dens fracture – complete angiorrhexis of A. vertebralis in the C1/C2 region – dissection of A. verteb. dex. with fresh intramural hematomas of the vessel wall extending from C1 to C3 with occlusion by intraluminal clot. Survival time: less than 5 min (estimation). Cause of death: brain stem ischemia due to severe trauma of the upper cervical cord.

Case 2: a 56-year-old man found dead beside a building. He had been seen working on the roof hours before. Medico-legal aspect: violent death due to occupational accident or sudden death by somatic cause? General pathology findings: lacerations to the forehead and face – age-dependent alterations of the internal organs – cardiomyoliposis – moderate coronary atherosclerosis. Neuropathological examination: Extensive atlanto-occipital hemorrhage – incomplete angiorrhexis of the art. vertebr. sin. at C2 level – dissection of intima and media extending to C4 with fresh clots along the narrowed arterial lumen – spinal medulla normal. Survival time: less than 5 min. (estimation). Cause of death: brain stem ischemia.

Case 3: a 26-year-old man found dead at the roadside. Medico-legal aspect: traffic accident with hit and run driver. Eye witness did

not inform police or emergency physician. Could death have been prevented? General pathology findings: polytrauma – fractures of the right knee joint – rib fractures (2–6 both sides) – fracture of the right mandible – basal skull fracture – discrete subarachnoidal hemorrhage basal – contusions of the brain stem. Neuropathological examination: multiple fractures of the cervical vertebrae (C2, C3, C5, C6) – contusions and traumatic lesions of the upper cervical cord – extensive hemorrhage of the meninges – dissection of A. vertebralis (from C1 to C3) bleeding and acute thrombosis. Survival time: less than 5 min (estimation). Cause of death: contusion of the brain stem – brain stem ischemia.

Case 4: a 71-year-old car driver involved in a severe traffic accident. Clinical diagnosis: tetraplegia, dens fracture, dislocated fracture Th2/Th3. Sudden death 7 days after admission. Medico-legal aspect: widow claimed insuffient intensive care treatment. General pathology findings: hemorrhage in the neck muscles and in the upper shoulder girdle region – compression fracture of vertebrae Th1/Th2, dislocation Th2/Th3 – cardiomyoliposis – moderate coronary atherosclerosis – stasis cirrhosis – congestion of the spleen – fibrosis of the pancreas – bilateral bronchopneumonia basal. Neuropathological examination: luxation fracture C1/C2 – spinal medulla revealed signs of contusion with predominantly centrally located hemorrhagic necrosis in the upper cervical cord – dissection of VA sinistra with intramural hematoma in the C1/C2 region extending to C3 – occlusion of both VAs at the level of the atlanto-occipital joint – in VA sinistra thrombus from C4 vertebrae to point of penetration of the dura mater – intradural segments normal – in VA dextra at C2 level an organized thrombus composed of fibrin and neovessels was associated with a recent thrombus formed of fibrin and blood cells. The arterial lumen was narrowed to 20% of normal – at C1 level a thrombus of similar age and a fresh thrombus were present occluding the complete arterial lumen. Survival time: 7 days. Cause of death: acute brain stem ischemia.

Case 5: a 73-year-old man after traffic accident as pedestrian. Clinical diagnosis of fender fracture and fracture of the talocrural joint on the left side. Fissur of os frontale sin. – superficial lacerations – neurological status normal. Sudden death during rapid head movement. Medico-legal aspects: causal relationship between accident and death? General pathology findings: lacerations to the left face – fracture os frontale sinister – age dependent alterations of the organs – extensive general atherosclerosis – moderate coronary atherosclerosis – signs of chronical myocardial insufficiency – chronic pulmonary emphysema. Neuropathological examination: extensive hemorrhage of the upper neck muscles and the atlanto – occipital region – extensive hemorrhage along VA dextra at the level C2 to C4 with angiorrhexis – complete occlusion of the proximal and distal vessel stump by thrombi – hypoplasia of VA sinistra with narrowing by osteophytic spur at C1/C2 level. Survival time: 3 days. Cause of death: brain stem ischemia.

Case 6: a 87-year-old man involved in a car accident as driver. Clinical diagnosis: superficial laceration to the forehead – vertebral arch fractures of C2. Since admission to hospital he became progressively more disoriented – in absence of neurological symptoms interpretation as organic brain syndrome – compression fracture Th4 – fracture metacarpale sin. Sudden death during positioning for CT-investigation. Medico-legal aspects: causal relationship to external event (motor accident) or somatic cause? General pathology findings: extensive hemorrhage in the neck muscles and shoulder girdle – compression fracture Th4 – rib fracture (2. left) – massive general atherosclerosis – heavy coronary atherosclerosis – mature myocardial scar – no signs of actue myocardial infarcation – signs of chronic myocardial insuffiency – recent bilateral bronchopneumonia basal. Neuropathological examination: extensive hemorrhage at the C1 to C2 level – lateral and posterior arch fracture of C2 vertebrae next to VA sinistra – recent angiorrhexis of all vessel layers, intramural hemorrhage – occlusion over a distance of 2 cm by recent thrombus – recent venous obstruction by thrombi in the neighbouring with punctiform reactive inflammatory infiltrates – stigmata of vertebrae degeneracy with extensive spondylosis of C1/C2 vertebrae. Survival time: 2 days. Cause of death: brain stem ischemia.

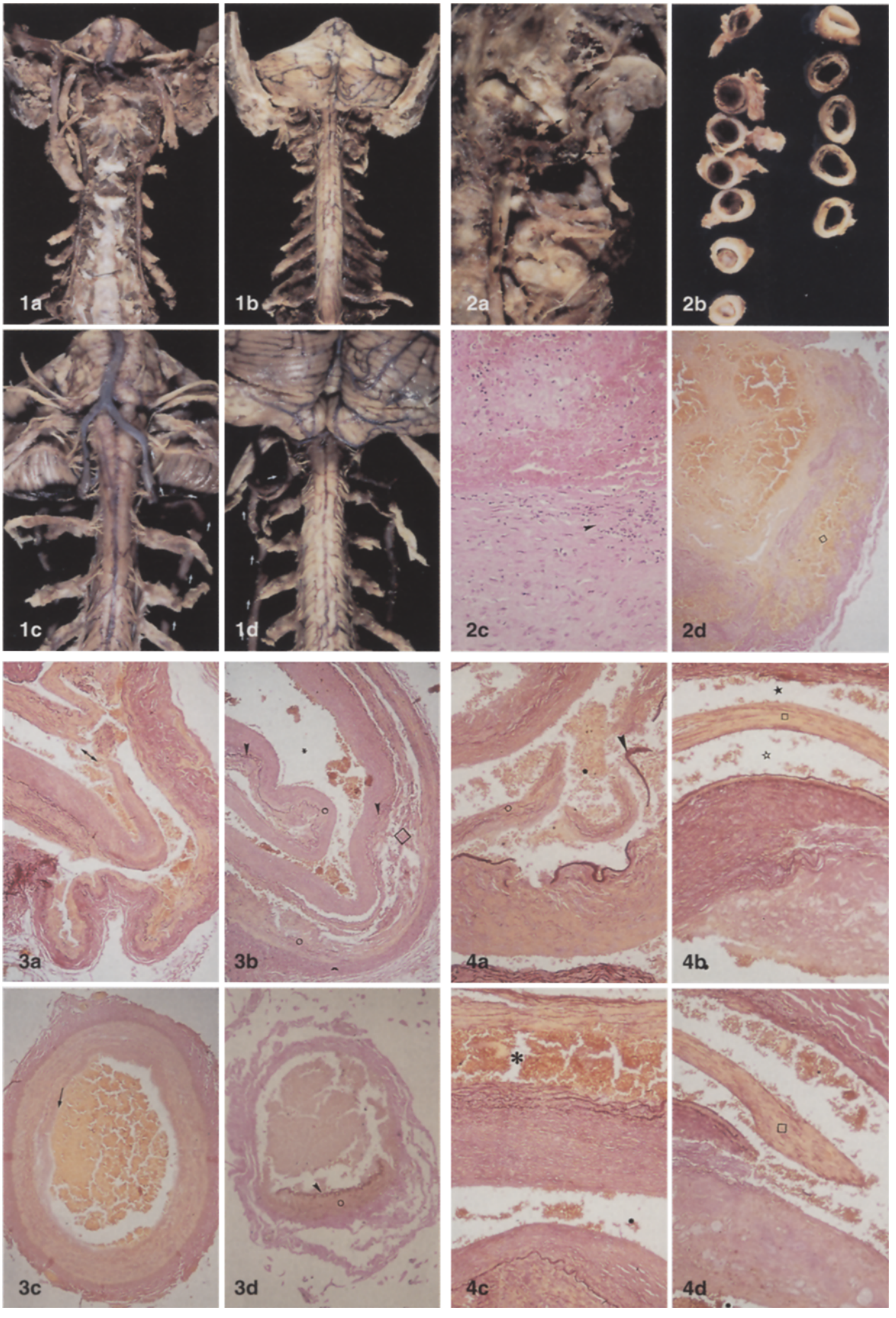
Dissection technique

In our opinion trauma to the head and neck always requires detailed morphological investigations of all possible sequelae. The cerebrum was separated from the cerebellum which was then removed with the fossa cerebri posterior and cervical spine en bloc and fixed in formaldehyde. Contrary to the proposal of Rabl et al. [80] this procedure allows investigation of the extracranial, transcranial and intracranial sections of the VA. In addition reliable and accurate examination of all other anatomical structures including cranial nerves, nerve roots, ganglions, meniges, spinal canal and spinal cord as well as blood vessels, muscles, fascial bands and bony structures can be performed. Moreover dissection artefacts caused for example by post mortem hemorrhage are prevented.

After removal of the soft tissues from the spine, the VA and cervical nerve roots were exposed from the anterior side. The skull base was removed ca 2 cm from the foramen magnum and the atlanto-occipital joint was inspected. The arch of the atlas was opened, the VAs were displayed and lamellated into 2 mm disks for paraffin and/or epoxy resin embedding. The spinal processes of the vertebra were removed and the nerves demonstrated from the posterior aspects. The spinal canal was inspected. The spinal cord and the cerebellum were removed. The dura was carefully slit at the back and front. After the cervical cord and the cerebellum were dissected the vertebral column was sawn longitudinally to demonstrate the vertebrae and intervertebral disks (Fig. 1 a–d).

Discussion

Extracranial VA dissections are often classified as either "traumatic" in cases potentially related to minor trauma or "spontaneous" when no apparent trauma is documented [3, 9, 11, 17, 18, 27, 29, 35, 56, 58, 67, 71, 82]. In our opinion extracranial VA injuries can be divided into acute (examples given by cases 1–3) and late groups (cases 4–6). The acute group includes immediate life threatening vascular injury. The exact survival time in cases 1–3 is not known. The time interval between trauma and death was estimated by the extent of autopsy findings such as hemorrhage, shock equivalents of the internal organs etc. Death was assumed to have occurred at once or in less than 5 minutes after traumatization. Suprisingly the examination of the VAs and surrounding tissues suggested that the local intravasal clotting processes seemed to start immediately in the region of the traumatic arterial lesion. As additional signs of traumatization and lesions of the VAs were present these clots were not considered to be an artefact due to post mortem blood coagulation or fixation procedure. In injured arteries, in cases of traumatic events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during life there were at least erythrocytes and platelets penetrating the vessel wall. The thrombosis in cases 4–6 could be established by light microscopy.

The late group is characterized by fatal sequelae with the onset of symptoms following lesion and can be delayed for hours up to days or sudden death resulting in certain head movements when a unilateral proximal VA occlusion was asymptomatic due to former adequate contralateral vertebral blood flow. Special attention is drawn to the late group because detailed knowledge of the symptoms may need propagation.

◀ **Fig. 1** Dissecting technique of the cranio-cervical region. At autopsy the posterior skull base and the cervical vertebral column are removed as one complete specimen and are primarily fixed in formalin. In order to accelerate or to ameliorate fixation a partial laminectomy could be performed. (**a**) Ventral aspect of the partially prepared specimen showing the vertebral arteries, the carotid arteries on the right and two of the caudal cranial nerves. (**b**) The posterior view of the specimen showing details of the atlanto-occipital junction and the cranio-cervical region. (**c**) After removal of all bony structures the vertebral arteries can be investigated in continuity (→), without any artificial lesions. The nerve roots of the caudal cranial nerves and of the cervical spinal nerves are preserved. (Ventral view). (**d**) By this cautious dissecting technique even small lesions of the roots, spinal ganglia or spinal nerves can be detected. (Dorsal aspect)

Fig. 2 Dissected specimen of the cervical spine (case report 6). (**a**) Ventral view of the cranial cervical vertebral column; detail of the left upper vertebral artery. There are some acute traumatic lesions of the artery, corresponding to a fresh fracture of the lateral part of the vertebral arch behind the VA. (**b**) Transverse sections of the vertebral artery. The artery is totally occluded by a relatively fresh thrombosis. Some sections show dissection of the arterial wall with small intramural hemorrhages. (**c**) Histological specimen of the left vertebral artery with an occluding fresh thrombosis. There is a beginning cellular reaction (▼) in the slightly thickened intima. (HE-staining; magnification 150 ×). (**d**) Left vertebral artery with an extended dissecting aneurysm (□). The lumen is occluded by a fresh thrombosis. The intima with the lamina elastica interna is torn at several sites. The blood flow has divided the tunica media into 2 portions. (HE-staining; magnification 60 ×)

Fig. 3 Histological appearance of vertebral arteries with traumatic lesions in case 2. (Van Gieson staining). (**a**) The vessel wall is divided into 2 portions by an extended intramural hemorrhage (dissecting aneurysm). Note the circumscribed interruption of the intima (↕). (Magnification 60 ×). (**b**) At some levels the inner portion of the tunica media with lamina elastica interna and thickened intima is invaginated into the blood vessel lumen. Thus there are inverted portions of the inner vessel wall in the center: Media (○), elastica interna (▼); intima, lumen, intima, elastica interna, media, dissecting aneurysm (□), media, adventitia (❩). (Magnification 60 ×). (**c**) Fresh coagulation thrombosis in the vertebral artery. The blood clot (↓)is already attached to the vessel wall. (Magnification 60 ×). (**d**) Vertebral artery with a total disruption of the tunica media (○) and internal elastic membrane (▼). The disrupted portion has been invaginated and is not seen in this plane. The lumen is occluded with a fresh thrombosis. (Magnification 60 ×)

Fig. 4 Vertebral artery with traumatic dissecting aneurysm (case 2). Details at higher magnification (150 ×). (**a**) Fragments of the intima (○) and elastic membrane (▼) are isolated in the vessel lumen (✷). (**b**) The tunica media (□) is surrounded by an interior (☆) and exterior (★) dissecting aneurysm. Vessel lumen (✷). (**c**) The thickened intima is separated from the tunica media by a fresh blood clot (❊). Vessel lumen (✷). (**d**) The entrance of the dissecting aneurysm divided by a portion of the tunica media (□)

Our autopsy findings confirm clinical observations that manifestations of vertebrobasilar insufficiency vary in onset and course. Recent reports have defined a wide spectrum of clinical manifestations. The neurological symptoms may develop progressively or discontinuously. The most common syndrome is headache with/or without neck pains, followed by ischemic symptoms in the vertebrobasilar distribution after a delay of hours or days. Thromboocclusion of the extra cranial VA may cause vertigo, disturbance of vision, repetitive transient ischemic episodes interpreted as organic brain syndrome or infarctions due to embolism leading to Wallenbergs syndrome. The pathophysiology of labyrinth-like syndromes has been clarified from the results of magnetic resonance imaging and arteriograms showing dissection of the VA after closed head injury [12, 15, 26, 27, 31, 51, 54, 60, 61, 75, 77, 81, 92, 94, 101]. Unilateral occlusion of the extracranial VA may also be asymptomatic because of the presence of the contralateral VA and with proximal obliteration of the collateral supply to the upper cervical VA from external carotid and thyreocervical vessels. In clinical follow-up studies under angiography, recanalization occurred in 2 out of 3 vessels and the VA either proved to be or returned to normal [12, 46, 58, 71].

VA lesion can be the result of blunt or penetrating tauma. Penetrating vascular injuries to the neck are infrequent but can not be overlooked. Among the various types of blunt trauma resulting in lesions of the VA are severe craniocervical trauma due to motor accidents, falls, blows and strangulation [1–3, 8–10, 21, 23, 32, 33, 41, 42, 44, 47, 51, 57, 58, 66, 81, 86, 89, 108). Involvement of VA in spinal injuries, especially in fractures and dislocations of the cervical vertebrae has been documented by many authors [3, 19, 34, 47, 90]. The aetiology of VA lesions due to severe head trauma and the pathomechanisms of the injury closely linked with the problem of stability and instability of fractures in the cervical spine, is obviously understood.

The fact that the vessel walls may also be injured by minor trauma was established by cumulative reports of patients who had undergone chiropractic manipulation of the neck and had sustained various degrees of brain stem ischemia, sometimes fatal [6, 20, 24, 34, 35, 49, 55, 60, 65, 67, 68, 79, 87, 92, 96]. Vertebrobasilar insufficiency due to cervical hyperextension and/or rotation as a consequence of positioning during surgical operations, complicated births, after cervical disk surgery or reanimation procedures have been reported [3, 5, 70, 72, 74, 95, 107]. Vessel lesions were even associated with daily activities like sport or ceiling painting [14, 33, 36, 54, 56, 74, 83, 91, 103] as well as sudden head rotations [48, 94].

The exact aetiology of VA lesions after minor trauma is unknown. The typical result of "minor head injuries" is a dissection of the VA between C1 and C2. Possible mechanisms of injury at the atlanto-axial level are thought to include stretching, tearing or occlusion of the relatively fixed ipsilateral upper cervical VA during forced extension and/or contra lateral rotation of the extended cervical spine. Nearly all lesions which were known to be traumatic in nature occurred homolaterally with delayed onset

of symptoms. Post-traumatic bilateral lesions were reported by Six et al. [98] and Heilbrun and Ratcheson [42]. The association of VA lesion with migraine, oral contraceptive use and chronic hypertension and atherosclerosis are suggested by observational data [10]. Young women seem to predominate [18] but these findings have yet to be established by adequate case control methods. VA lesions have regularly been associated with fibromuscular dysplasia and intimal abnormalities of uncertain significance [4, 10, 29, 34, 40, 58, 78]. Many aspects of extracranial VA lesions are still unexplained.

It is tempting for clinicians to speculate that these lesions are analogous to saccular aneurysms; a congenital arterial defect manifested with ictus and sometimes precipitated by specific activities. But it is also tempting to speculate that VA dissections result from the late onset of previous traumatic vessel lesions. Recently Pollanen et al. [78] hypothesised that minimal lesions due to mechanical disruption of smooth muscle cells in the arterial vessel wall are followed by liberation of catabolic enzymes with subsequent degradation of the arterial media. In post-traumatic VA ruptures they observed discontinuous medial lesions not associated with the site of the rupture. They concluded by experiments that these lesions of the tunica media are due to "in situ trauma release" of heat labile enzymes.

Former blunt trauma to the head and neck is only occasionally mentioned. Quint and Spickler [75] reported a patient with "spontaneous" vertebral artery dissection, who denied recent trauma or neck manipulation, although she reported having been involved in a motor vehicle accident 1 year previously. Sometimes a connection between trauma and VA lesion is not even seen as in the report of Frauchinger and Reutter Bernays [27] where a 21-year-old girl developed neck pains and brain stem symptomatology after appendectomy (operation positioning/intubation) due to VA dissection which was postulated to be spontaneous by the authors. In most clinical cases the pathogenesis remains unclear.

We find it difficult to classify with certainty whether a lesion is spontaneous or traumatic without detailed morphological and microscopical investigations. Multi-vessel involvement and bilateral dissection in the absence of any history of trauma raises the possibility that an underlying arteriopathy or vasculitis is responsible [10, 52, 104, 106]. But only regular dissection of VAs, cervical spine and cord after severe and minor head trauma will bring further information and basic data for testimony.

The extracranial VAs are partially protected but relatively restricted by the deeper discontinuous bony canals formed by the transverse processes of the cervical spine. Although any segment can be affected there is evidence that the vessel wall is most vulnerable to trauma at characteristic sites:

- at the C1-C2 level
- at bony fixations at the transverse foramina at C6-C2
- at the cranio-cervical junction especially the atlanto-occipital joint.

These areas constitute loci of minor resistance [25, 43, 84, 86]. Possible consequences of indirect trauma include pseudo-aneurysms, arteriovenous fistulas as well as subintimal, vascular, intramural and perivascular hemorrhage accompanied by narrowing of the injured vessel lumen. These processes can result in transient or permanent occlusion, either due to an extensive thrombosis, which is occasionally associated with recurrent thrombo-embolisms, or propagation of the thrombi [2, 3, 9, 10, 12, 14, 20, 27, 28, 30, 32, 35, 36, 45, 46, 59, 64, 74, 89, 90, 95, 98, 99, 100].

A process that influences vertebrobasilar circulation and/or starts hemodynamic thrombosis without morphological lesion of the vessel wall is the initiation of vasospasms by the peripheral autonomous nervous system. This reaction to mechanical irritations can develop immediately and last for variable periods of times. This mechanism may partly explain the obliteration of the contralateral VA in case 4 where no VA lesion could be morphologically verified. In general our results are in accordance with the findings of Saternus [84, 85] and Saternus and Burscheidt [86] who emphasized that post-traumatic lesions of the proximal vessels are underestimated. These observations presume that injury to the extracranial VA and resulting consequences may often be overlooked at autopsy. In delayed death there might not even be a suspicion of brain stem ischemia by microscopical examination because morphological alterations always need time to develop and are absent due to the short time interval between the ischemic event of acute vertebrobasilar insufficiency and death caused by apnoea.

Manifestations of acute anoxic or ischemic cell damage in the brain stem that may occur in minimal time intervals prior to death cannot be distinguished from post mortem autolytic processes and therefore can not be proven. Without examination of the VAs the cause of death may be defined by general pathology findings e.g. pneumopathy, acute heart failure accompanied by coronary atherosclerosis or cardiomyoliposis in these cases. Therefore the recognition of unusual but not always uncomplicated extracranial lesions of the VA must be of special interest to forensic pathologists as it may present a life-threatening complication.

Traumatic VA lesion may be the only but most important neuropathological autopsy finding. The present hypothesis is that individual aggravating factors might explain the fatal outcome when normal autopsy procedures including microscopical evaluation does not reveal an obvious cause of death. The consideration of possible acute brain stem ischemia as a cause of death in cases after trauma to head and neck is based on the experience that the incidence of post traumatic extracranial VA injuries seems to be more frequent than supposed and the knowledge of the most common variations of anatomical structures in reality. For better understanding the following facts are reminded briefly: the cerebellum, the lower brain stem and parts of the upper brain stem as well as parts of the occipital and temporal lobes are mainly supplied by the vertebral arteries whereas the rostral brain stem and

the cerebral hemispheres are predominantly supplied by the internal carotid arteries. At the base of the brain arborisations of these arteries form the circle of Willis. A circumscribed ischemic attack in the supply areas of internal carotid arteries is very often not dangerous to life, but an abrupt ischemia in the vertebro-basilar system might be serious and might be the cause of sudden and unexpected death. It must be stressed that the assembly of arterial blood vessels of the circle of Willis shows wide anatomical variations in shape, size and structure. It might not be sufficient to prevent acute brain stem ischemia in a number of cases when a critical reduction in the vertebrobasilar blood supply occurs.

We presume that vertebral artery thrombo-occlusion associated with contralateral critical reduction of the posterior circulation perfusion due to underlying artery anomalies, compression by osteophytic protrusion from cervical spondylosis, involvement of the contra lateral vessel in fractures and dislocations of the cervical vertebrae as well as certain movements or positions of head and neck present an apparent risk of acute brain stem ischemia. Experiments conducted on human cadavers as well as in vivo arteriograms indicated that circulation through one vertebral artery was influenced by certain head positions and was dramatically reduced when the head was hyperextended and tilted to the opposite side [7, 16, 22, 37, 43, 53, 102, 105]. These head positions associated with VA occlusion, atresia, hypoplasia, compression by osteophytic spur, dislocation of cervical vertebrae structures or even narrowing by cervical fascial bands [7, 22, 28, 30, 38, 39, 43, 53, 60, 73, 76, 93, 97, 99, 100, 102] may be occult risk factors leading to sudden death due to acute brain stem ischemia.

Therefore in any fatality involving minor or severe craniocervical trauma, dissection of the vertebral spine and vertebral arteries must be performed to exclude or prove causal relationship between the cause of death and an external event.

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