Romanian Neurosurgery (2019) XXXIII (2): pp. 166-173
DOI: 10.33962/roneuro-2019-031
www.journals.lapub.co.uk/index.php/roneurosurgery

Secondary (Duret) brainstem haemorrhage may not always represent a fatal event. Review of literature and report of four cases

Martin Hanko[1], Branislav Kolarovszki[1], Kristián Varga[1], René Opšenák[1], Pavol Snopko[1], Radoslav Hanzel[1], Kamil Zeleňák[2]

[1] Clinic of Neurosurgery, University Hospital in Martin, Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, SLOVAKIA
[2] Clinic of Radiology, University Hospital in Martin, Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, SLOVAKIA

Keywords
Duret haemorrhage, intracranial hypertension, prognosis, secondary brainstem haemorrhage

ABSTRACT

Background. Secondary brainstem haemorrhage (eponymously called Duret haemorrhage) is a well-known complication of transtentorial brain herniation or of rapid decompression of intracranial space. It is considered to be a consequence of arterial rupture, venous infarction or ischemia-reperfusion injury and it is regarded as a harbinger of an unfavourable outcome for the patient. Despite this, several case reports describing good outcome after Duret haemorrhage preceded by evacuation of an expansive traumatic intracranial mass lesion, an episode of intracranial hypotension or lumbar drainage have been published.

Case description. We present four cases of patients with secondary brainstem haemorrhage linked to an episode of intracranial hypertension due to various reasons who were treated at our clinic. The first patient suffered a small brainstem haemorrhage that was described on his initial CT scan presumably as a result of massive intracranial expansion caused by an acute subdural haematoma and this Duret haemorrhage markedly expanded after the subdural haematoma was evacuated by means of a decompressive craniectomy. The next two patients developed Duret haemorrhage after the evacuation of intracranial haematomas. The fourth patient presented with posttraumatic cerebral oedema complicated by a subtle Duret haemorrhage displayed on his initial CT scan and this bleeding remained stable even after a bilateral decompressive craniectomy. One patient passed away, one remained in a persistent coma and two survived with a light neurological deficit.

Conclusions. However ominous a newly discovered Duret haemorrhage may be, it alone should not discourage us from the further intensive treatment of our patients as their outcome may considerably vary. The extent of this bleeding, type and severity of underlying brain injury and complete clinical status and history of our patients should all be taken into account when deciding about patients' prognosis.

✉
Corresponding author:
Branislav Kolarovszki

Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, Clinic of Neurosurgery, University Hospital in Martin Kollárova 2, 036 59 Martin, Slovakia

kolarovszki@jfmed.uniba.sk

ISSN online 2344-4959
© Romanian Society of Neurosurgery

First published
June 2019 by
London Academic Publishing
www.lapub.co.uk

INTRODUCTION

Originally described by a French surgeon Henri Duret[6], secondary

brainstem haemorrhages or eponymously Duret haemorrhages are a feared consequence of a transtentorial brain herniation or of a decompression of the intracranial space by means of craniectomy or by removal of an intracranial mass lesions [16,21,22].

They can occur with a delay of only 30 minutes after the initial insult and primarily affect the ventral midline parts of the pons and the mesencephalon, but never the medulla oblongata [7,15,26]. Because such haemorrhages often irreversibly destroy the brainstem reticular formation, functional centres and descending pathways, their prognosis is usually very poor with patient either dying or surviving for a limited amount of time with a severe deficit, often unconscious and ventilator-dependent [15,18,26]. However, case reports of patients suffering Duret haemorrhage and achieving a highly favourable outcome with a gentle or no persistent neurological deficit are known [1,2,3,8,11,16,20]. Currently, the question of whether a diagnosis of Duret haemorrhage should discourage us from an aggressive treatment is debated. In this work we review the available knowledge concerning secondary brainstem haemorrhage with attention paid on its pathogenesis and outcome of patients who suffer it. We also present four cases of our patients with Duret haemorrhages which resulted in various outcome.

Case presentations

Case A

A 50 years old man was found unconscious with a right-sided mydriasis. He had a history of chronic alcoholism and ethylic liver disease. The emergency ambulance was called, he was intubated and sedated, then transported to our hospital. After arrival, his GCS score was 4 (1-1-2), he was anisocoric with bilateral absence of pupillary and corneal reflexes and with a decerebrate response to painful stimuli. CT scan verified an acute left-sided subdural haematoma 24 mm thick, descending transtentorial herniation, enlargement of the right lateral ventricle, midline shift of 21 mm and a small interhemispheric haemorrhage. There was also a subtle Duret haemorrhage located in the pons (Figure 1A). The patient was immediately brought to the operating room and a decompressive craniectomy with an evacuation of the subdural haematoma was performed. He was then kept sedated and ventilated with circulatory support by norepinephrine until a control CT scan was performed 6 hours after the surgery. It revealed a reduction of the midline shift to 3 mm, however remains of the subdural haematoma were present and new bilateral intracerebral haemorrhages occurred together with a bleeding into the third and fourth ventricle. Massive enlargement of the pontine haematoma that was now extending into mesencephalon was described. Perimesencephalic cisterns were bilaterally compressed (Figure 1B). Based on these findings the patient's prognosis was considered highly unfavourable and no other surgical intervention was indicated, we continued in a palliative care. The sedation and mechanical ventilation were discontinued (the patient remained unconscious with GCS 3), oxygen and norepinephrine support was further provided. 14 hours after the surgery our patient died due to failure of cardiorespiratory functions.

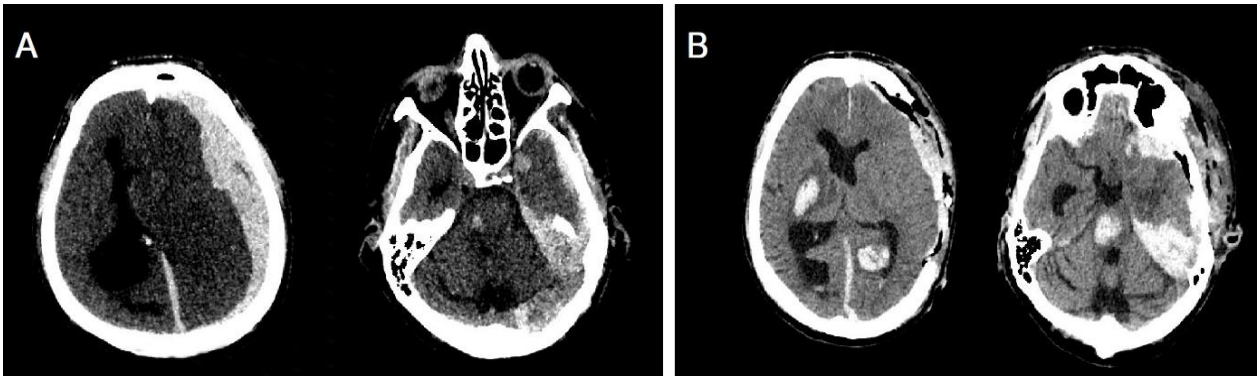

Figure 1. Preoperative CT scan (A) revealing a massive acute subdural haematoma 24 mm thick, smaller interhemispheric acute subdural haematoma, enlargement of the right lateral ventricle, midline shift of 21mm, and a subtle Duret haemorrhage located in the pons. After a decompressive craniectomy (B) the midline shift had resolved however remains of subdural haematomas are apparent. New intracerebral haemorrhages together with progression of pontine bleeding are visible.

Case B

This 64 years old man with a history of arterial hypertension, liver steatosis, lung resection due to tuberculosis and chronic obstructive pulmonary disease underwent a right-sided decompressive craniectomy with an evacuation of an acute posttraumatic haemorrhage into a chronic subdural haematoma. Two months later he was admitted again for a planned cranioplastic surgery usong an autologous bone flap. At that time, he was fully conscious, well-oriented and communicating with a moderate left-sided hemiparesis persistent since the event of his traumatic brain injury. He underwent the cranioplasty without any serious complications and without any changes in his neurological status immediately after surgery. Approximately 8 hours later the patient had an epileptic seizure that terminated after a 5 mg intravenous dose of diazepam, he however remained non-responsive, his right-sided pupil was enlarged. The attending neurosurgeon indicated an immediate CT scan which revealed an epidural haematoma 47 mm thick at the site of reimplanted bone flap, midline shift of 19 mm, compression of lateral ventricles and perimesencephalic cisterns and a transtentorial herniation of the right-sided temporal lobe. The patient was transported to the operating room with pupils already bilaterally mydriatic and non-reactive. Emergent evacuation of the epidural haematoma was performed and the bone flap was returned with drainage tubes placed into the epidural space. The patient remained sedated and ventilated until the next CT scan 8 hours later which revealed a recent haemorrhage into right-sided basal ganglia, all four ventricles and a Duret haemorrhage into the upper pons and mesencephalon. 24 hours later the sedation and mechanical ventilation were discontinued and a tracheostomy was performed. At that time the patient was unconscious with GCS 3, he had irregular pupils with a slight right-sided mydriasis, absent pupillary reflex and present corneal reflex, spontaneously ventilating. He was later transferred to a nursing home and remained in persistent coma with his neurological status unimproved (as reported from his last known clinical check 2 months after the surgery).

Case C

A 63 years old woman pedestrian was struck by a car and subsequently transported to a peripheral hospital, unconscious with GCS 7 (1-1-5), present pupillary reflex, sedated and intubated. CT scan was performed revealing a skull base fracture, pneumocephalus, traumatic subarachnoid haemorrhage, small subdural haematoma over the right hemisphere and a small haemorrhagic contusion in right temporal lobe. She also suffered numerous smaller lung contusions. 24 hours later she was transferred to our institution due to a progressive oedema of the right hemisphere with an enlargement of the haemorrhagic contusion in the right temporal lobe. Decompressive craniectomy and evacuation of the haemorrhagic contusion was subsequently performed and the patient remained sedated for following 24 hours. A control CT scan revealed two subtle Duret haemorrhages (one located in the pons and one in the mesencephalon) both with a diameter of approximately 1 cm (FIGURE 2). No further surgical intervention was indicated. Two months later she underwent autologous bone flap replantation and a ventriculo-peritoneal shunt insertion due to development of a posttraumatic hydrocephalus. After the surgery she remained conscious with GCS 14 (4-4-6), disoriented with signs of organic psychosyndrome - slightly bradypsychic, occasionally agitated and verbally aggressive. Her only persistent motoric deficit was a right sided ptosis.

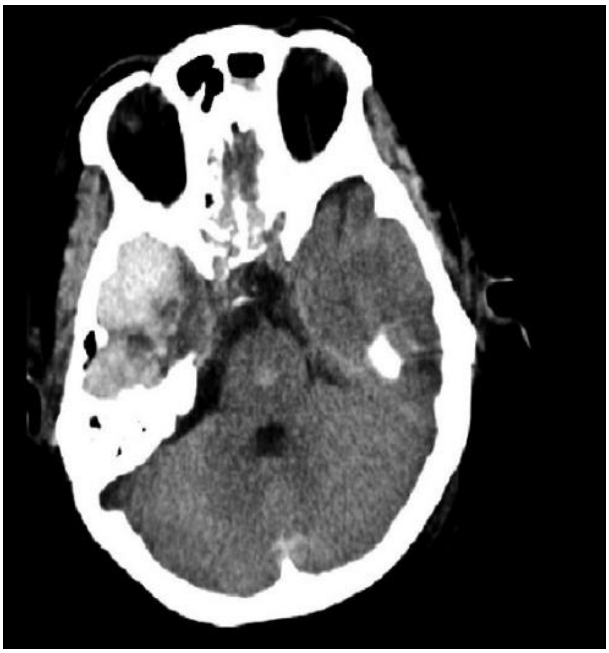

FIGURE 2. Postoperative CT scan verifying basally located remains of haemorrhagic contents and a subtle Duret haemorrhage in the pons.

Case D

A 27 years old patient who suffered a motorcycle accident was immediately sedated and transferred to our clinic due to a CT verified posttraumatic cerebral oedema, subarachnoid haemorrhage, non-expansive right-sided acute subdural haematoma, intracerebral haematomas in both frontal lobes and a small Duret haemorrhage located in his ventral mesencephalon. Prior to sedation he had GCS 8 (1-2-5), he was isocoric with pupillary reflex bilaterally preserved. Due to a presence of non-expansive haemorrhagic intracranial lesions only an intraparenchymal intracranial pressure sensor was initially inserted but 12 hours later the patient developed a refractory intracranial hypertension with pressure values exceeding 40 mmHg and a bilateral decompressive craniectomy was performed. However, because of a CT verified Duret haemorrhage the patient's prognosis was considered unfavourable. The postoperative CT revealed stabilized intracranial findings with an adequate decompressive effect of the bilateral craniectomy with the brainstem haemorrhage being stable and non-expanding. The sedation was discontinued and the patient was later discharged to a peripheral hospital surprisingly achieving a highly favourable outcome: GCS 15 and a slight right sided hemiparesis and facial nerve palsy.

Discussion

Henri Duret (1849-1921), a French surgeon and a pioneer of neuroscience focused his aim in neurological research on localising of functional areas in the brain, describing the vascular supply of the central nervous system and investigating pathomechanisms of traumatic brain injuries [27]. In experimental conditions he had described mechanisms of intracranial hypertension by simulating intracranial mass lesions and correctly assumed brainstem (especially medulla oblongata) as a centre of cardiorespiratory functions. He had also noted a disturbance in the cerebral tissue perfusion during periods of increased intracranial pressure and had observed microscopic haemorrhages located in the floor of the fourth ventricle which were associated with episodes of increased intracranial pressure [6,27]. Despite of having inaccurately linked these bleedings to an increased cerebrospinal fluid pressure at the moment of primary brain injury and despite a fact that these haemorrhages might not even be true secondary brainstem bleedings, Duret haemorrhages have remained an eponymous term for secondary brainstem haemorrhagic lesions caused mostly by intracranial hypertension and following the transtentorial herniation [21,27].

Causes of Duret haemorrhage

Duret haemorrhage is typically a result of a descending transtentorial brain herniation. Cases of a brainstem haemorrhage preceded by an occurrence of an intracerebral haematoma [19,21], acute subdural haematoma [1,10,17,20,21], cerebral infarction [19] or diffuse cerebral oedema caused by hyponatremia [11] are known. On the other hand, several cases of the Duret haemorrhage following an episode of intracranial hypotension which can also provoke a descending transtentorial herniation have been reported. Cardinale et al. have published a case of the Duret haemorrhage in a patient after a decompressive craniectomy complicated by an episode of intracranial hypotension and a paradoxical brain herniation presenting as the sinking skin flap syndrome [4]. Similarly, Yuan et al. have described a case of the Duret haemorrhage in a patient with lumbar drainage following an operation of a thalamic tumour [28]. There is also a report of a patient who suffered a brainstem haemorrhage caused by a postoperative lumbar pseudomeningocele and associated intracranial hypotension [3]. Interestingly, even a rapid decompression of the intracranial space by means of evacuation of an expansive intracranial haemorrhage [2,8,13,22] or by means of a decompressive craniectomy [16,17,25] can result in the Duret bleeding.

Incidence

In their autoptic research of haemorrhagic stroke Nedergaard et al. have observed an incidence of the Duret haemorrhage in 45% of intracerebral haemorrhages, 15% of cerebral infarctions and 36% of aneurysm haemorrhages [19]. In a clinico-pathological report of Klintworth, Duret haemorrhage was typically associated with intracranial lesions complicated by a rapidly expanding intracranial haemorrhage or brain oedema or with a surgical decompression of intracranial space even in slowly growing lesions with overall incidence of 16.5%, specifically 31.5% for

intracerebral haemorrhages, 21.5% for subdural haematomas and 11.6% for cerebral infarctions [13]. In a non-missile brain injury, Graham et al. have reported an incidence of the Duret haemorrhage in 51% of autopsies [9]. However, almost 20% of these haemorrhages could only be discovered during microscopic examination [9] and this fact leads us to expect that radiological examination will underestimate the incidence of secondary brainstem haemorrhages in surviving patients.

Pathogenesis

Unlike primary brainstem haemorrhage which could be a result of a spontaneous arterial rupture, direct traumatic injury to the brainstem or of a rupture of vascular malformation, secondary brainstem haemorrhage typically presents with a delay and is mostly a consequence of the descending transtentorial herniation or follows a rapid decompression of the intracranial space [16,21]. There are several main explanations for pathogenesis of the Duret haemorrhage. The theory of an arterial origin of the Duret haemorrhage assumes that a descending transtentorial herniation causes a displacement and angulation of the brainstem with an elongation and straining of the perforating arteries extending from the basilar artery which is fixed in its upper end by the posterior communicating arteries and the posterior cerebral arteries. This tension results in either vasospasm or rupture of the perforating arteries and causes the Duret haemorrhage [12,15,21,26]. The very appearance of the Duret haemorrhage is highly dependent on the volume and rate of expansion of the underlying intracranial mass lesions and also on variations of systemic blood pressure and blood inflow into the perforating arteries during the episodes of transtentorial herniation. Slowly expanding intracranial mass lesions do not usually cause the Duret haemorrhage [7,12]. An extremely rapid descending transtentorial herniation causing acute brainstem ischaemia results in early death of the patient without an episode of the secondary brainstem bleeding (which is prevented by the disturbed blood flow in perforating arteries) [12]. Based on experimental research on animal model, Klintworth has postulated that the Duret haemorrhage arises during two possible scenarios: The transtentorial herniation can extensively displace the perforating branches of the basilar artery. If sufficient blood flow in these vessels is retained, they are prone to rupture and cause secondary brainstem haemorrhage [12]. Evacuation of the intracranial mass lesion causing transtentorial herniation and perforating vessel damage (mechanic or ischaemic) can also provoke Duret haemorrhage in a situation when the blood flow into these damaged arteries is restored causing them to bleed even more extensively than during transtentorial herniation only, especially in situation of elevated systemic blood pressure [12,14,15,24]. It is possible that all types of blood vessels in the ventral brainstem are damaged in a manner of an ischaemia-reperfusion injury and are later prone to cause a massive secondary brainstem haemorrhage [14]. The theory of reperfusion injury is supported by a report of Sim et al. who have observed an enhancement of ventral pontomesencephalic area on CT angiography in a patient with a transtentorial herniation due to bilateral chronic subdural haematoma and acute tentorial subdural haematoma. After a burr-hole drainage of chronic haematomas was performed, a massive Duret haemorrhage in the area of previous parenchymal enhancement occurred. The contrast extravasation probably demonstrated damaged perforating arteries [24]. Arterial origin of Duret haemorrhage is also supposed when based on a report by Chew et al. who have performed a post-mortem CT angiography on a patient who died due to a middle cerebral artery aneurysm rupture associated with a transtentorial herniation and a secondary brainstem haemorrhage. Multiple linear enhancing foci corresponding to ruptured paramedian perforating arteries found in autopsy were detected in the central pons [5]. Theory of a venous infarction arises from an observation of venous congestion in the brainstem in patients with a supratentorial mass lesions, perivenous localisation of several Duret haemorrhages and a fact that the veins are thin-walled vessels easily compressed by an external pressure, specifically at the site of the tentorial notch. Compression can cause the veins to thrombose and result in venous brainstem haemorrhages [12,15,21,23]. This explanation has been however questioned because secondary brainstem haemorrhages often extend anatomical regions of the venous drainage and the evacuation of intracranial mass lesions can rather provoke Duret haemorrhage than prevent it [12]. Observations of a brainstem ischaemia with no secondary

haemorrhage in patients with the transtentorial herniation also challenges this theory of pathogenesis [26].

Favourable outcome of patients suffering a Duret haemorrhage

Despite being considered a predictor of infaust prognosis, several reports describing a favourable outcome of patients with the Duret haemorrhage caused by various types of intracranial pathologies have been published. Dramatic recovery has been reported by Ishizaka et al.: A patient with an idiopathic subdural haematoma and preoperatively described Duret haemorrhage underwent evacuation of the subdural haematoma and despite of a severe preoperative neurological deficit his status resolved to a persistent oculomotor palsy only [10]. Similarly, Nguyen et al. have described a patient with an acute subdural haematoma and a Duret haemorrhage achieving a favourable outcome (dysarthria and no motor deficit) after an evacuation of a subdural bleeding [20]. Stiver et al. have also reported a favourable outcome of a young woman who developed a Duret haemorrhage after an evacuation of an epidural haematoma by means of decompressive craniectomy [25] and another case of favourable outcome after an evacuation an epidural haematoma in a patient with a Duret haemorrhage has been published by Fujimoto et al. [8]. A report by Park et al. describes a patient who suffered a Duret haemorrhage after a burr-hole drainage of bilateral chronic subdural haematomas and survived with his only persistent deficit being a gentle gait disturbance [22]. Lonjaret et al. have published a case report of patient with a Duret haemorrhage triggered by a decompressive craniectomy for an acute subdural haematoma resulting in a favourable outcome with no neurological deficit [16]. Kamijo et al. have reported a favourable outcome (gentle hemiparesis) in a patient with a severe hyponatremia causing a transtentorial herniation and a Duret haemorrhage [11]. A favourable outcome with persistent internuclear ophtalmoplegia only in a patient with a Duret haemorrhage due to an intracranial hypotension has been published by Bonow et al. [3]. In paediatric patients, Beier and Dirks have reported a favourable outcome in two patients with Duret haemorrhage preceded by a traumatic brain injury [1]. When summarized, most of patients surviving with favourable outcome have suffered a brainstem haemorrhage of a limited extent only [1,2,3,8,20], however exceptions exist [11,16,22]. Younger age of these patients could be a factor predicting a favourable outcome [1,11,16,20]. A typically reported persisting neurological deficit in patients with the favourable outcome is often a cranial nerve weakness [1,3,8,10,11,16], or various subtle forms of the organic psychosyndrome and a cognitive deficit [1,25].

Analysis of our case series

The first three patients (A,B,C) described in our work represent cases of Duret haemorrhage provoked or worsened by a rapid decompression of intracranial space combined with a decompressive craniectomy in 2 of 3 patients. The explanation of this phenomenon could be based on the theory of reperfusion into the damaged pontine perforating vessels as initially postulated by Klintworth [12,13,14,24]. Alleviation of a significant pressure on the brainstem had most likely increased the blood inflow into mechanically and metabolically injured vessels in the pontomesencephalic region and resulted in the secondary brainstem haemorrhage (or its significant expansion in one case). The patient D probably suffered a Duret haemorrhage due to a massive cerebral oedema with a mass effect that displaced the brainstem and distorted the mesencephalic perforating arteries thus causing them to rupture because of a combination of metabolic and mechanic damage [12,15,21,26]. It is possible that due to patient's younger age and absence of complicating diagnoses the perforators remained relatively stable even after the alleviation of the intracranial hypertension by means of decompressive craniectomy and the secondary brainstem haemorrhage did not expand. Two of our four patients had achieved a good outcome and this was probably caused by a smaller extent of the pontine haemorrhage and a younger age of patient D. Chronic liver disease present in two patients with unfavourable outcome could play a role as a potential negative prognostic factor as it can negatively affect the function of the blood coagulating system, however a definite conclusion can only be made when relating to a larger group of patients.

Conclusions

Massive elevation of intracranial pressure, decompression of the intracranial space by means of

craniectomy or intracranial bleeding evacuation can make the patient susceptible to the secondary brainstem (Duret) haemorrhage. Such bleeding, typically localised in the midbrain and pons has been considered a significant predictor of poor prognosis. Despite of this, outcome of patients suffering the Duret haemorrhage can considerably vary as presented in our report. When deciding about the prognosis of a patient with the Duret haemorrhage, attention should be paid to the type and severity of the underlying brain injury, patient's clinical status and history and at last but not least to the extent of the very brainstem bleeding.

Abbreviations

CT: computed tomography
GCS : Glasgow Coma Scale

References

1. Beier AD, Dirks PB. Pediatric brainstem hemorrhages after traumatic brain injury. J Neurosurg Pediatrics. 2014; 14(4), 421-4.
2. Bermudéz G, Areitio E. Duret haemorrhage with full recovery. Acta Neurochir (Wien). 2015; 157(8), 1337-8.
3. Bonow R, Bales JW, Morton RP, Levitt MR, Zhang F. Reversible coma and Duret haemorrhage after intracranial hypotension from remote lumbar spine surgery: case report. J Neurosurg Spine. 2016; 24(3): 389-93.
4. Cardinale M, Meaudre E, Cunqi PQ, Nguyen C, Esnault P, Joubert C. Duret hemorrhage complicating a sinking skin flap syndrome. Br J Neurosurg. 2017; Sep 8,1-2.
5. Chew KL, Baber Y, Iles L, O'Donnell C. Duret hemorrhage: demonstration of ruptured paramedian pontine branches of the basilar artery of minimally invasive, whole body post-mortem CT angiography. Forensic Sci Med Pathol. 2012; 8(4), 436-40.
6. Duret H. Études Expérimentales et Cliniques sur les Traumatismes Craniocérébraux. Paris: Thése; 1878.
7. Fields WS, Halpert B. Pontine Hemorrhage in Intracranial Hypertension. Am J Pathol. 1953; 29(4), 677-87.
8. Fujimoto Y, Aquiar PH, Freitar AB, de Andrade AF, Marino Júnior R. Recovery from Duret Hemorrhage: A Rare Complication after Craniotomy. Neurol Med Chir (Tokyo). 2000; 40(10), 508-10.
9. Graham DI, Lawrence AE, Adams JH, Doyle D, McLellan DR. Brain damage in non-missile head injury secondary to high intracranial pressure. Neuropathol Appl Neurobiol. 1987; 13(3), 209-17.
10. Ishizaka S, Shimizu T, Ryu N. Dramatic recovery after severe descending transtentorial herniation-induced Duret haemorrhage: A case report and review of literature. Brain Inj. 2014; 28(3), 374-7.
11. Kamijo Y, Kishita R, Hamanaka S, Soma K. Duret haemorrhage is not always suggestive of poor prognosis: a case of acute severe hyponatremia. Am J Emerg Med. 2005; 23(7), 908-10.
12. Klintworth GK. The pathogenesis of Secondary Brainstem Hemorrhages as Studied in an Experimental Model. Am J Pathol. 1965; 47(4), 525-36.
13. Klintworth GK. Evaluation of the role of neurosurgical procedures in the pathogenesis of secondary brain-stem hemorrhages. J Neurol Neurosurg Psychiatry. 1966; 29(5), 423-5.
14. Klintworth GK. Paratentorial Groowing of Human Brains with Particulatr Reference to Transtentorial Herniation and the Pathogenesis of Secondary Brain-stem Hemorrhages. Am J Pathol. 1968; 53(3) 391-408.
15. Leestma JE. Forensic Neuropathology. Boca Raton: Taylor&Francis; 2014.
16. Lonjaret L, Ros M, Boetto S, Fourcade O, Geeraerts T. Brainstem haemorrhage following decompressive craniectomy. J Clin Neurosci. 2012; 19(9), 1293-5.
17. Martins WA, Teixeira AB, Frigeri TM, Paglioli E. Spontaneous subdural hematoma associated to Duret haemorrhage. Interdisciplinary Neurosurgery. 2015; 2(1), 13-15.
18. Moore AS, Mittal MK, Rabinstein AA. Duret Hemorrhage Following Subdural Hematoma Evacuation. JAMA Neurol. 2013; 70(4), 518.
19. Nedergaard M, Klinken L, Paulson OB. Secondary Brain Stem Hemorrhage in Stroke. Stroke. 1983; 14(4), 501-5.
20. Nguyen HS, Doan MB, Gelsomino MJ, Shabani S, Mueller WM. Good outcomes in a patient with a Duret haemorrhage from an acute subdural hematoma. Int Med Case Rep J. 2016; 9, 15-18.
21. Parizel PM, Makkat S, Jorens PG, Ozsarlak O, Cras P, Van Goethem JW, et al. Brainstem hemorrhage in descending transtentorial herniation (Duret haemorrhage). Intensive Care Med. 2002; 28(1), 85-8.
22. Park KJ, Kang SH, Lee HK, Chung YG. Brain Stem Hemorrhage Following Burr Hole Drainage for Chronic Subdural Hematoma. Neurol Med Chir (Tokyo). 2009; 49(12), 594-7.
23. Scheinker IM. Transtentorial herniation of the brain stem. A characteristic clinicopathologic syndrome; pathogenesis of hemorrhages in the brain stem. Arch NeurPsych. 1945; 53(4), 289-298.
24. Sim KB, Na DG, Park JK. Precedence of Parenchymal Enhancement on CT to a Fatal Duret Hemorrhage. J Korean Neurosurg Soc. 2013; 53(6), 380-2.
25. Stiver SI, Gean AD, Manley GT. Survival with good outcome after cerebral herniation and Duret hemorrhage caused by traumatic brain injury. J Neurosurg. 2009; 110(6): 1242-6.

26. Tomliston BE, Brain-stem lesions after head injury. J Clin Pathol Suppl (R Coll Pathol). 1970; 4, 154-65.
27. Walusinski O, Cou.rivaud P. Henri Duret (1849-1921): A Surgeon and Forgotten Neurologist. Eur Neurol. 2014; 72(3-4), 193-202.
28. Yuan X, Zhao H, Zhao C, Zhao H, Xiaho H, Sun G, et al. Duret Hemorrhage after Lumbar Drainage. J Craniofac Surg. 2014; 25(4), 1551-2.