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Review Article

Medicolegal evaluation of pontine hemorrhage at autopsy- Highlights from a case and an overview

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ABSTRACT

One in ten non-traumatic intracerebral hemorrhages (ICH) is located in the pons with chronic arterial hypertension as the leading etiology. In the forensic context, deaths related to a pontine hemorrhage (PH) are usually encountered in situations of drug abuse, excited delirium, trauma, as well as in sudden natural deaths where some hypertensive catastrophe is the usual underlying mechanism. The clinical presentation of PH may be variable, causing a failure in timely diagnosis that, if presents with unexplainable circumstances, may become the subject of medicolegal concern. The present case relates to a middle-aged man with a long history of hypertension and presents during an afternoon with an abrupt onset of deleterious symptoms. The patient was managed conservatively but succumbed to his illness and expires during treatment. A questionable diagnosis and the case circumstances, however, directed the doctors to inform the police. A medicolegal autopsy was therefore carried out that leads to the discovery of a lethal pontine hemorrhage rupturing into the fourth ventricle and involving the adjacent cerebellar tissues as well. Severe atherosclerosis of the basal arteries constituting Circle of Willis and Vertebrobasilar system was seen along with their hallmark effects that became evident during brain sectioning. Pathological stigmata of well established hypertension were found in the heart and kidneys. A clinic pathological correlation of the physical characteristics and topography of the hematoma to its severity was also carried out, based upon the known CT and autopsy findings. The possibility of a drug related or traumatic and secondary brainstem/Duret hemorrhage was ruled out.

1. Introduction

Hypertension is the leading cause of intracerebral hemorrhage (ICH). An ICH related to hypertension is most frequently located deep within the cerebral hemispheres, affecting basal ganglia and thalamus while a few cases present with posterior fossa hemorrhage, involving the pons and/or cerebellum[1]. The frequency of pontine hemorrhage (PH) is the lowest, but they are considered to be the most pernicious form of all ICH, with cause-related mortality approaching nearly 50%.[2] The risk of ICH is further increased by the use of cocaine, amphetamines or any other sympathomimetic drug that increases the blood pressure and heart rate.[3]

The present case relates to a middle-aged man that was a known case of hypertension and on regular medications. On an afternoon, he was found in a moribund state on the bed by his son and was immediately admitted to the hospital but could not be resuscitated despite best of the efforts. A doubtful clinical diagnosis and case circumstances, however, lead the doctors on duty to inform the police and an autopsy was therefore carried out. The autopsy revealed a fresh hemorrhage into the pons that was further rupturing into the fourth ventricle, as well as permeating the adjacent cerebellar tissues. The traumatic, drug related, and secondary causes of the hemorrhage were carefully looked

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into and are discussed herein details.

2. Case Details

2.1. History and clinical data

The case relates to a 56 years old married man, a village resident, and working as laborer in the waterworks Dep't. The pt. was a chronic hypertensive and on regular medications since last about 8 to 10 years. Recently, however, he had volitionally refused taking medication in a feeling of desperation. On an afternoon, his son discovers him shivering on the bed with limbs stiffened and hyperextended, and chief complaints of 'feeling cold' in a slurred speech. During this process, the pt. drops down from the bed and became unconscious after a little while.

After about an hour, he is received in the hospital in an unconscious state along with seizures, respiratory distress, and frothing from the mouth. Upon presentation, the blood pressure (BP), pulse rate (PR), and respiratory rate (RR) measured 220/148 (mm Hg), 96/min., and 24/min., respectively. Injection lasix was administered along with other baseline medications and the pt. was transferred to the intensive care unit and put on mechanical ventilation with a diagnosis of "??CVA with aspiration" and managed conservatively.

Endotracheal intubation was performed and the vitals were re-monitored with BP-250/150, 216/128,. . . , PR-96, 94, 98,. . . /min., RR-24, 22, 24. . . /min., SpO_2 90%, and FiO_2 66%. The random blood sugar level measured 181 mg/dl. Bilateral pupils were NSNR. The pt. was responding only to painful stimuli. Oxygen was administered and i.v. fluids along with the injections of mannitol, antiemetics, anticonvulsants and antibiotics were started. Urine output was normal. Chest auscultation revealed bilateral basal crept while S_1S_2 heart sounds were normal, without any muffles. CT head could not be done as the pt. was on the ventilator. The last blood pressure and PR recorded were 198/120 and 108/min., respectively. The pt. remained in coma throughout the course, along with persistent records of malignant hypertension, and with a poor prognosis explained. Finally, the CPR was commenced that lasted for about 30 min., after which he was declared dead (~ 8 hrs after onset of symptoms). Doubtful of the final diagnosis and case circumstances, however, the doctors felt it necessary to inform the police. The relatives denied of any foul play or mismanagement by hospital, over his death. The 'cause of death' certificate read as "Cardiac arrest. ?? CVA with aspiration with HTN". The dead body was brought for postmortem examination in the next morning.

2.2. Autopsy details

The body was brought for an autopsy after about 16 hours of death. The apparent cause of death, as stated by the police, was "dimag ki nas fatne ke kaaran", i.e., due to rupture of some brain vessel. After a thorough perusal of police papers, hospital documents, and recording the witnesses' statements, the autopsy was commenced.

3. External Examination

It was an average-built body of a middle-aged man, enwrapped in a white sheet of cloth and 162 cm in length. The body was clad in a white kurta-pajama and undergarments which were devoid of any tears, abnormal stains, or odor. Eyes and mouth were closed. Bilateral scleral hemorrhages were present, along with well-established arcus senilis (Figure 1).

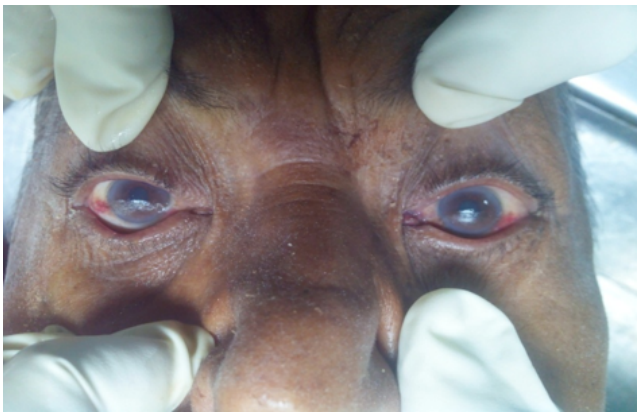

Fig. 1: Bilateral scleral hemorrhages (not *tache noire*) are present

No ocular or facial petechiae were seen. Slight facial congestion was present. All the natural body orifices were healthy. Little yellowish fluid secretions (gastric contents) were coming out of the mouth and nostrils. No suspicious injury marks were seen except for typical external marks of reanimation efforts. Signs of putrefaction were absent. Facial features were well recognizable. Rigor mortis was well developed all over the body. Post mortem hypostasis was livid purplish, bearing prominent vibices over the dorsum of back, root of the neck, and suprasternal regions, and with typical contact pallor.

4. Internal Examination

No evidence of any soft tissue, musculoskeletal or visceral injury was found anywhere during dissection.

4.1. Gross findings

4.1.1. Brain

The brain [Wt. 1320 gm] was congested and softened, especially over the base. As it was removed from the cranial cavity and placed on the dissection table, with the base facing upwards, fresh blood accumulations/hematomas were noticed at different areas, in the vicinity of the brainstem, as follow (Figure 2): (1) apex of the fourth

ventricle and adjacent right flocculus of the cerebellum (clot size ~ 3 x 2.5 x 1 cm), (2) Bilateral foramina of Luschka, in the cerebellopontine angles (left clot ~ 1.2 x 0.8 x 0.3 cm, right clot ~ 0.8 x 0.5 x 0.2 cm).

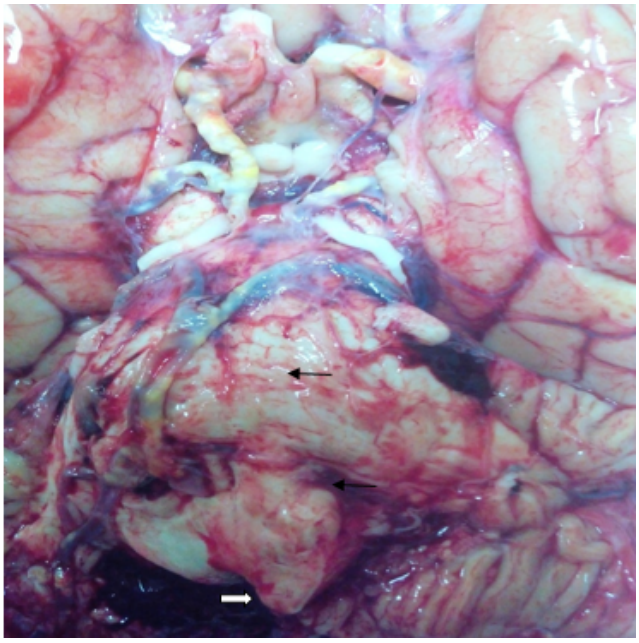

Fig. 2: Blood accumulations (hematoma) are present over (1) Apex of the fourth ventricle and adjacent flocculus of right cerebellum (white arrow), (2) Foramina of Luschka over cerebellopontine angles (black arrows). Severe nodular atherosclerosis of basal arteries is present.

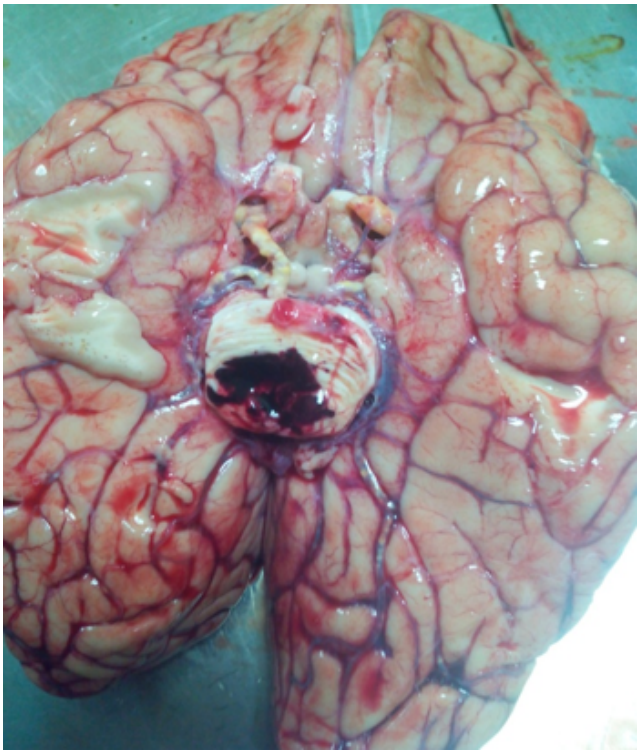

Fig. 3: Fresh hemorrhage over the basal-tegmental region of the rostral pons with rupture into the fourth ventricle. No evidence of herniation or brain swelling is present.

As the rostral pons was sectioned and separated, a ‘large’ area of fresh hemorrhage was seen over its basal-tegmental region, measuring about 23 mm x 14 mm (trans. x long.) and rupturing into the fourth ventricle (Figure 3). Fresh hematoma of approx. 7 mm in thickness and volume ~ 8 ml was adherent to the pons that could be traced further deep into the cerebellar sections. (Figure 4) The hemorrhage was appreciated in multiple sections of the pons. The whole of the fourth ventricle was filled with blood. Cerebral aqueduct was, however, largely unaffected.

Severe atherosclerosis of the arteries constituting the Circle of Willis and Vertebral-basilar system was present, with yellowish nodular hard atheromas giving a "String-of-beads" appearance to the basal vessels. (Figures 2 and 3) Upon cross-sectioning, the arterial lumina at multiple places were critically stenosed, while small to moderate sized variegated thrombi were laminated upon them. Vascular wall thickness was variable at different locations. The supratentorial region of brain did not reveal any space occupying lesion or bleeding. Numerous ischemic and lacunar infarcts were present in the serial sections of brain, chiefly involving the basal ganglia, subcortical white matter, and the pons as well. (Figure 5) Severe atherosclerosis of

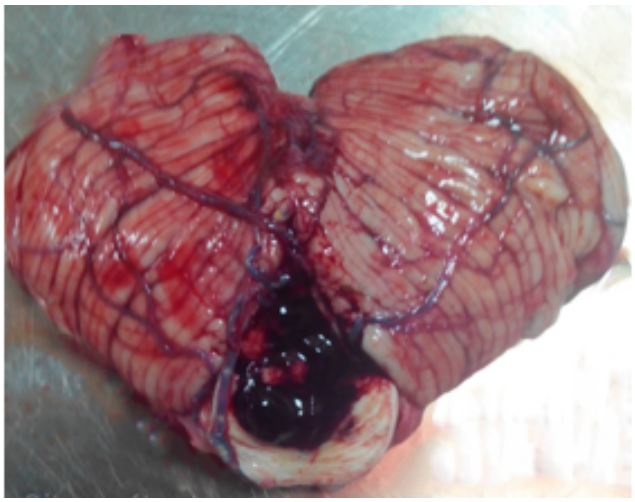

Fig. 4: Thick hematoma over the rostral pons with hemorrhage permeating into the adjoining cerebellar tissue.

vessels was seen in the insular cortex and elsewhere to.

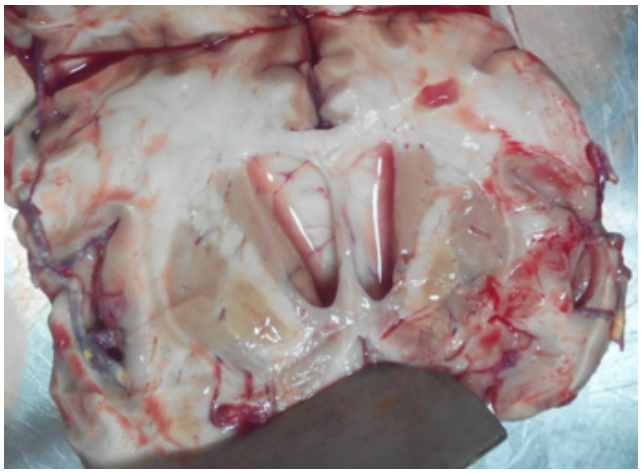

Fig. 5: Ischemic and lacunar infarcts in the basal ganglia and subcortical white matter, indicating a chronic pathological process. Insular vessels show atheromas. Lateral ventricles are dilated.

Midbrain, medulla oblongata, and dentate nuclei were unaffected. No evidence of herniation, midline shift, or brain swelling was seen. No other intracranial bleed or traumatic hemorrhages were found. The vertebrae and spinal cord were not opened.

4.1.2. Heart

[436 gm] measured 10.6 x 7.6 x 7.0 cm. Upon dissection, thickness of LV fee wall, IV septum, and RV free wall measured 1.8 cm, 2.0 cm, and 1.0 cm, respectively, implying significant biventricular hypertrophy. The circumferences of tricuspid, pulmonary, mitral, and aortic valves were (in cm) 10.5, 9, 7, and 6, respectively, depictive of narrowed AV and dilated pulmonary valves, while the circumference of aortic valve was borderline low-normal.

4.1.3. Respiratory system

The left lung [416 gm] was almost glued to the parietal pleura through fibrous adhesions while the right lung [846 gm] was firm and heavy with diffuse tan to grayish areas. The cut surface of the right lung was congested and exuded copious frothy secretions. Petechial hemorrhages were seen over visceral as well as parietal pleurae. Abundant coarse-whitish froth and mucus were present in the respiratory tract up to the carina, sticking to a congested mucosa.

4.1.4. Abdominal organs

Liver [1460 gm], spleen [145 gm], and right kidney [114 gm] showed congested cut surfaces. Left kidney [150 gm] had a slightly granular surface along with fluid filled cysts over the cortex.

4.1.5. Organs of generations (external and internal):

Unremarkable

4.2. Microscopic findings (H & E staining)

4.2.1.

4.2.1.1. Brain. Sections taken from the representative areas of the brain showed congestion and hemorrhage.

4.2.1.2. Heart . Representative micro sections taken from different areas of the heart and stump of the aorta revealed no significant pathology. Both the coronary arteries were traced as far as possible. Micro sections from the right coronary a. revealed complicated atherosclerosis with calcification while the left main coronary a. and its left anterior descending branch showed moderate atherosclerosis.

4.2.1.3. Liver. Marked congestion and mild fatty changes were present.

4.2.1.4. Lungs. Sections from the lungs revealed marked congestion and oedema.

4.2.1.5. Kidneys . Micro sections examined from the pieces of kidneys revealed marked congestion and thickening of the arterial walls (i.e., arteriosclerosis).

4.2.1.6. Spleen . Representative sections revealed marked congestion.

4.3. Toxicology report

No common poison or drug or ethyl alcohol was detected in the viscera, body fluids, and pontine hematoma that were preserved for the chemical and toxicological analysis. The analysis included testing over the following exhibits:

1. Viscera: stomach and portions of small and large intestine with their contents, core portions of liver, spleen, kidneys, and a portion of subcutaneous abdominal fat.
2. Body fluids: peripheral venous blood, bile and vitreous humor.
3. Deep pontine and ventricular hematomas: as such; sodium fluoride was added as a preservative.

4.4. Cause of death

The cause of death was given as primary pontine hemorrhage (PPH), due to hypertension. The manner of death was natural.

5. Discussion

Etiologically, about half of all ICH seem to be caused by hypertension.[4] The remaining non-hypertensive ICHs are caused by cerebral amyloid angiopathy, vascular

malformations, neoplasms, drugs, thrombocytopenia, DIC, and hemophilia.[1] Hypertensive cerebral hemorrhages have their favored anatomic sites. Common sites are putamen and thalamus, lobar white matter, cerebellum, and pons. In an autopsy series, with the ICH-related deaths, 42% of the cases involved striatum; 16%, pons; 15%, thalamus; 12%,. cerebellum; 10%, cerebral white matter; and 5%, other sites[5] Among all kinds of intracerebral hemorrhages, PPH is known to have the worst prognosis. Clinically, the onset of PH is usually sudden, following a chain of signs and symptoms that are often characterized by an early coma with tetraplegia, decerebrate posture, respiratory disturbance, hyperthermia (rarely hypothermia), and pin-point pupils.[6]

5.1. Relation of physical parameters to mortality

A pt. suffering from massive PH usually dies within the first 48 hrs.[6] Small tegmental, dorsal, and lateral localizations may have better chances of survival.[7] Previous radiological studies have attempted to measure the size of the pontine hematoma to correlate it with the prognosis and pt.'s survival. Measurement of both transverse and longitudinal diameters revealed that prognosis was more related to the transverse diameter. Tanabe et al. found that among survivors, the maximum transverse diameter was 31.5 mm or less, whereas most fatal cases had hematomas exceeding that size.[7] Masiyama et al found that the prognosis of cases with hematoma of the maximum transverse diameter of 20 mm or less was often favorable, whereas it was mostly poor when the hematoma exceeded 21 mm in size.[8]

In autopsy cases, it is frequent to encounter a 'large ICH' causing the death of an individual. 'Large' is a relative term, but some attempts have been made to quantify the volume. For example, a hemorrhage greater than 3.0 cm in diameter in the cerebral hemisphere, 2.0 cm in the cerebellum, and 1.0 cm in the brainstem, has been considered large.[9] Large central hematomas often begin at the junction between the basis pontis and pontine tegmentum and may extend to the rostral midbrain and the fourth ventricle. A volume of 5 ml or greater has been found to be associated with a poor prognosis.[10] Depending upon the axial CT-scan features, PPH has been divided into four types: basal-tegmental, bilateral tegmental, massive, and small unilateral tegmental.[11] Massive hematomas occupy the basis pontis as well as the tegmentum bilaterally. In the bilateral tegmental type, a hematoma is present in the bilateral tegmentum only. Basal-tegmental variety involves the junction between basis pontis and tegmentum bilaterally, while in small unilateral tegmental, the hematoma is located exclusively in the unilateral tegmentum. Case survival rate was found to be the best with small unilateral tegmental and least with the massive type. The authors of the study concluded that small hematomas located in the unilateral tegmentum had the best prognosis.[11]

Dziewas and colleagues classified PH locations into three categories, following its trans-axial location: (a) large paramedian, (b) unilateral basal-tegmental and, (c) lateral tegmental.[12] The large paramedian PH reflected a poor prognosis, whereas the lateral tegmental type was associated with a favorable outcome. The transverse hematoma diameter threshold value related to the outcome was found to be 20 mm,[12] similar to the findings of Masiyama et al.[8] The size of the PH in the present case, measuring about 23 mm (trans.) x 14 mm (long.), could be related to a poor outcome that was probably further worsened by its location (basal-tegmental; along with fourth ventricular rupture), free-clot thickness (~ 7 mm), volume (~ 8 ml), and involvement of multiple pons sections.[8,13]

5.2. Autopsy and histological findings in PH

In a hypertensive PH, the pons at autopsy is usually grossly distended by the blood, palpably soft, and consists of soft, friable, bloody tissue. Hemorrhages such as these not uncommonly, destroy the pontine tissue completely. Features typical of an acute intraparenchymal hemorrhage are noted that are usually located centrally within the pons, on account of the larger paramedian perforators of basilar a. which are the common sites of rupture.

The hematoma more frequently extends in a rostrocaudal direction, along the traversing long tracts rather than laterally into the middle cerebellar peduncles. Usually, the hematoma does not extend beyond the pontomedullary junction inferiorly and the inferior midbrain superiorly but frequently ruptures into the 4th ventricle.[14] The precise point of vascular rupture in a hypertensive ICH, however, is virtually never identified.[1] Histology of the hematoma is usually not rewarding. In a few cases, however, changes of arteriosclerosis and fibrinoid necrosis (viz. malignant hypertension) in the vessels, situated in the immediate vicinity of hemorrhage or in the opposite grossly unaffected cerebral parenchyma, may be found.

The arteriosclerosis in hypertension may be of benign/hyaline or hyperplastic/malignant variety. In malignant hypertension, a hyperplastic type of arteriosclerosis usually affects the blood vessels, which is characterized by a pattern of thickening commonly likened to an "onion skin" due to concentric laminations of the smooth muscle cells, causing narrowing of the vessel's lumen. The condition frequently affects blood vessels of the kidneys, though it can occur anywhere. Lacunar infarcts (état lacunaire) and similar perivascular infarcts in the subcortical white matter (état criblé), are common gross and microscopic findings in hypertensive ICH.[15] True saccular microaneurysms, commonly known as Charcot-Bouchard aneurysms, may be seen histologically[16]

5.3. The impact of atherosclerosis and hypertensive heart disease

Atherosclerosis (ATH) is by far the leading systemic vasculopathy to result in occlusive-ischemic infarcts and stroke, especially in older patients. The basic underlying pathogenesis of cerebral ATH is probably no different from that in any other artery, and the risk factors that have been recognized, including smoking, hyperlipidemia, diabetes, obesity, and hypertension, probably apply as well to brain vessels, like any other.[17] However, ATH is not a uniform process, and there is often great disparity between the degrees of ATH found in the aorta, peripheral vessels, coronary arteries, and cerebral vessels.[15] This has been attributed to the different anatomic and hemodynamic peculiarities of intracranial arteries, such as lack of an external elastic lamina as well as a distinct metabolism.[18] Cerebral ATH is not usually as severe as in systemic vessels but rarely may be far more severe than anywhere else. Intracranial ATH is most severe in the major branches of the Circle of Willis and Vertebral-basilar system.[19] Vascular findings in cerebral ATH may show variations as follow[15]

1. Arteries with normal diameter and lumens except for focal plaques, if present. The person(s) rarely suffers any significant stroke.
2. Arteries of inherently small and delicate caliber, with very little ATH required to compromise the circulation. Plaques are nodular and turn the vessels literally into bead-like strings (viz. the present case fig. 2). Such cases usually depict severe atheromatous disease in other vessels too and may suffer numerous major strokes over many years.
3. Arteries are much larger than normal, often calcified, having rather flat, ulcerated, or excavated intimal plaques (i.e., a calcified–dilated form). An S-shaped deformity of the basilar artery may be discovered (atherosclerotic/cirsoid aneurysm). Lacunar infarcts and TIAs are commonly seen.

Although hypertension is well known as a significant risk factor for the development of atherosclerosis and is also often found in association with sudden death due to atherosclerotic coronary artery disease, one must also recognize that hypertension can be the sole factor in causing the death of an individual. In particular, hypertensive LVH can be associated with sudden arrhythmic deaths, or with dissecting aortic aneurysms and spontaneous/non-traumatic intracerebral hemorrhage.[20]

Concentric left ventricular hypertrophy with cardiomegaly is generally a reflection of underlying hypertension and it is not surprising that hypertensive LVH can be the sole anatomic finding in some sudden deaths related to hypertension. Benign or malignant nephrosclerosis of the kidneys and vascular hypertrophy and sclerosis in the brain, if present, will support the diagnosis of hypertension as the likely cause of the hemorrhage.[1]

5.4. Medicolegal Aspects of PH

5.4.1. Drug abuse and intracerebral hemorrhage

Pontine hemorrhage has been a frequent finding in drug abuse-related deaths, especially with the use of cocaine and methamphetamines.[3] It is well-known that cocaine abuse can precipitate ICH or an aneurysmal (viz. Berry's aneurysm) rupture, causing a massive basal subarachnoid hemorrhage that often occurs in the setting of hypertensive cardiovascular disease. Such cases are not uncommon in deaths related to excited delirium where an extensive evaluation of the circumstances and autopsy findings may be required before concluding the cause and manner of death.[21]

ICHs related to hypertensive pathology are usually easy to distinguish because they mostly involve a 'blow out of the basal ganglia in an individual with a large heart and/or with a history of hypertension. In such cases, if it is anticipated that cocaine, methamphetamine, or some other sympathomimetic drug(s) may have contributed to a hypertensive hemorrhage, thereby possibly indicating an 'accidental' manner of death, the hematoma can be sent to toxicology; especially if the brain has been dissected in a fresh state.[22]

In one study, 23 of 26 people, who were abusing cocaine when they sustained an ICH, had autopsy findings of hypertensive cardiovascular disease.[3] Methamphetamine abuse-related deaths from fatal pontine hemorrhage have been reported.[23]

The sympathomimetic effects of drugs of abuse cause an elevation of the pulse rate and blood pressure, hyperthermia, arrhythmia, and uncontrolled hypertension, which occasionally result in a fatal outcome.[24] Coronary thrombosis and spasms, as well as intractable angina, are other known deleterious effects of these drugs. The drugs may be snorted, injected intravenously, taken orally, or smoked, resulting in intense euphoria and addictive potential.

The toxicological analysis of the hematoma in the present case, however, did not reveal any of such drug(s) or ethyl alcohol.

5.4.2. Differentiation between primary and secondary brain stem hemorrhages

Apart from the primary brainstem hemorrhage, there is a separate hemorrhage of secondary nature in the upper brain stem that results from herniation, often referred to as a 'Duret hemorrhage', although this term is probably a misnomer.[25] This hemorrhage most likely occurs when unilateral rapidly developing supratentorial mass lesion(s) leads to brain stem herniation. The hemorrhage is usually intraparenchymal but may develop

very rapidly (as short as 30 min.), and is often associated with other signs of intracranial hypertension such as subfalcine, parahippocampal-uncal/transtentorial, and cerebellar tonsillar herniation, with/without associated brain swelling[26].

The usual plane of a Duret hemorrhage is vertical and in the midline of the midbrain and rostral pons, either in continuum or patchy. In some secondary brainstem hemorrhages, there may be two or more linear hemorrhages in the midbrain. Some authors have likened them to 'crow's feet'.[27] Being in a paramedian location, these hemorrhages carry a poor prognosis.[12] Duret hemorrhages can be very massive, especially in association with trauma-related ICH.[28]

Forensically, it is further important to differentiate between a hypertensive and trauma-related primary ICH. In such situations, it is important to note the location of ICH, as well as to notice the additional signs of trauma over brain tissue. A non-hypertensive ICH does not usually occur in the locations where the hypertensive ICH is seen (lateral ganglionic region, basis pontis, dentate nucleus of cerebellum).[15]

Traumatic hematomas mostly underlie the subcortical U-area of white matter and are generally smaller than hypertensive bleeds, but maybe multiple. However, large contusions into the striatum, even rupturing into the lateral ventricle(s) (a central 'brain rupture'), may occur from excessive rotational forces acting upon the head[1,29]; however, a typical 'blow-out' intracerebral hematoma is unlikely.

In apparently traumatic hemorrhages, there may be associated hemorrhagic contusions over the cortex or flame-like hemorrhages over gyral crests, gliding contusions over parasagittal frontal lobes/superior parietal lobules, and/or other evidence of inner brain trauma, such as streak or punctate hemorrhages about the cerebral aqueduct or in the white matter, which should be distinctive.[30] The presence of a significant scalp injury and/or skull fracture along with meningeal hemorrhage(s) may be other trauma-related evidentiary findings.

Trauma-related primary PHs are often located in the dorsolateral quadrant of rostral tegmentum, appearing as either single or multiple focal patches of contusions (fig. 6), where they usually act as independent markers of diffuse traumatic axonal injuries[30,31]. Such hemorrhages are usually incompatible with life.[31]

The speckled and petechiae-type hemorrhages of traumatic axonal injuries are similarly distributed in the dorsolateral midbrain and/or rostral pontine tegmentum (Figure 7).[30] The possibility of a delayed traumatic ICH (Bollinger's traumatische Spät-Apoplexie) should be kept in mind[32], depending upon the case history, circumstances, and complete autopsy findings.

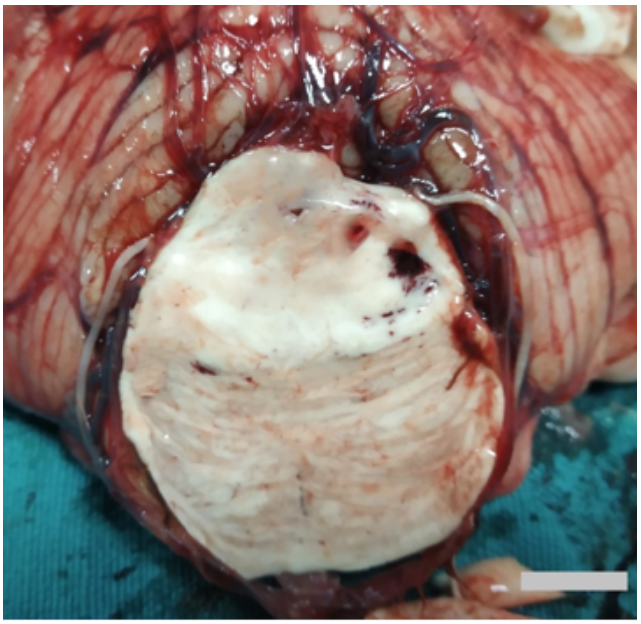

Fig. 6: An isolated patchy contusion and multiple streak-like traumatic hemorrhages in the right lateral pontine tegmentum. an upward herniated and swollen rim of peripontine cerebellar tissue can also be noticed. [Survival period ~ 1 hr., death due to fall from height]

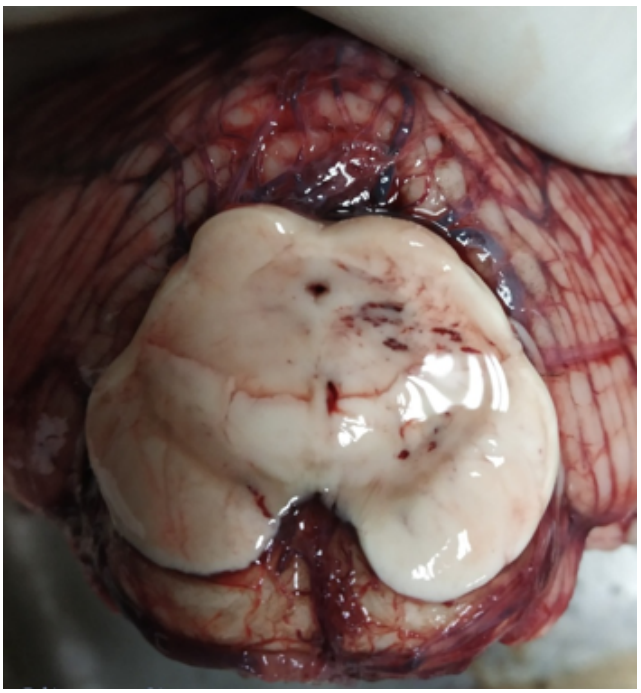

Fig. 7: Multiple petechial to streak-like traumatic hemorrhages in the midbrain's dorsolateral segment, depictive of sheared axons from trauma. Dot-like and punctate hemorrhages are also present in the right substantia nigra. [Acute death in a road-side accident]

6. Special Remarks

Hypertensive intracerebral hemorrhage is more common in blacks in comparison to whites and is further commoner in Asian males.[1] The diagnosis of hypertension may have been made in life, but sometimes individuals may be simply found dead at the scene with ICH, without any prior history of hypertension. Hypertension is a known risk factor for arteriosclerosis, and causes both atherosclerosis of medium and large arteries and arteriolosclerosis of the arterioles. Elevated blood pressure causes damage to the endothelium and vascular wall through both mechanical and humoral factors.[33] Clinically, two scoring systems, i.e., ICH score and PPH score, have been developed to predict a 30-days survivability in cases with PPH.[34,35] The ICH score grades the patient's prognosis on the basis of additive score of five factors: GCS value, age, ICH volume, ventricular blood, and infratentorial bleeding[34], while the PPH score primarily takes three independent factors, i.e., GCS score, pupillary reflex, and plasma glucose levels.[35,36] A GCS score of 6 or less, the absence of pupillary light reflex, and plasma glucose of 180 mg/dL or greater have been found to be independent mortality predictors of PPH. After assigning one point to each of these factors, the PPH score indicated 30-day mortality rates of 7.7%, 33.3%, 78.9%, and 100% for patients with 0, 1, 2, and 3 points, respectively.[35]

Other statistically significant factors associated with poor prognosis have been tachycardia (bpm ≥ 100/min.), hyperthermia (body temperature ≥ 39^0C), an extension of pontine hematoma to the midbrain and/or thalamus, associated ventricular bleed, maximum diameter of hematoma ≥ 20 mm, and volume of hematoma ≥ 30 ml.[35] Before the advent of CT scanning, even when it was possible to make a diagnosis of a brainstem lesion, it was frequently not possible to diagnose a pontine lesion. The diagnosis of PH usually had to await necropsy.[8] With CT, however, it is possible to discover small hematomas which previously could not have been diagnosed, and it is now known that the prognosis of pontine lesions is not always poor.

7. Conclusion

Autopsies are crucial in highlighting vascular mechanisms that are of importance in stroke's pathogenesis. The stroke may have been ischemic or hemorrhagic or a combination of both. Spontaneous hemorrhage within the central nervous system, attributable to hypertension, although arises primarily within the basal ganglia, can also originate from the pons and cerebellar hemispheres. From a medicolegal point of view, postmortem toxicology should be performed to rule out the presence of drugs that might cause an acute elevation of blood pressure (e.g., cocaine or methamphetamine), but there is more likely to be a clinical history of hypertension and/or autopsy evidence of concentric LVH. The possibility of associated head trauma, as well as the nature of the brain stem bleed, i.e., primary versus secondary, must be differentiated at autopsy.

In the absence of any relevant clinical history of hypertension or cardiovascular and/or end-organ pathologies at autopsy, it is still reasonable to attribute spontaneous hemorrhage in one of the three common sites in the brain to hypertension; as hypertension is often clinically silent and may not always produce classical concentric LVH and hypertensive nephrosclerosis.

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None.

9. Conflict of Interest

None.

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