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INTRACEREBRAL HEMORRHAGE- AN ANALYTICAL STUDY OF AUTOPSY CASES

Pathology

Manisha Narkar Assistant Professor, B.Y.L Nair hospital

Sweety Vijay Shinde* Associate Professor, B.Y.L Nair hospital *Corresponding Author

Vrushali Balwant Patil Laboratory Technician, B.Y.L Nair hospital

ABSTRACT

Intracerebral hemorrhage is a leading cause of death. Rapid death offers less time for pre-mortem investigational modalities. Detection of haemorrhage patterns, clinical manifestations and gross-histopathology findings is a rich learning experience. Knowledge is essential for treating physicians and budding pathologists.

KEYWORDS

Intracerebral, Hemorrhage, Brainstem, Hypertension, Autopsy

INTRODUCTION

Stroke patients can have underlying cerebral infarction or intracerebral haemorrhages (ICH). Haemorrhagic stroke has incidence of 32—270 per 100,000 population. ICH forms upto 15% of stroke cases. [1-3]

OBJECTIVES:

To analyse ICH cases at autopsy over a two-year study period.

MATERIALS AND METHODS:

A retrospective observational study conducted at pathology department of tertiary care center. Medical autopsies with provisional cause of death due to ICH were included. Autopsies without ICH were excluded. Autopsy records were scrutinised for patient demographic details, clinical presentation, gross and microscopic findings.

RESULTS:

In two-year period total 539 medical autopsies were performed of which 72 cases (13.6%) had CNS related deaths. Cases due to meningitis, infarction, neoplasms were excluded. Total study sample was 42.

Male preponderance (32, 76.2%) was noted. The mean age was 44.5 years (27 - 74) Commonest age group in males was 41-50 years (11,34.3%) and in females was 71-80 years (4,40%). Time period between admission to death was 1-3 days (12, 28.6%), <1 day (27, 64.3%) and brought dead (3,7.1%).

Table 1: Clinical presentation of ICH autopsy cases (N=42)

CLINICAL PRESENTATION	NO. OF CASES	PERCENTAGE
Unconsciousness	21	50.1%
Hemiparesis	9	21.4%
Convulsion	9	21.4%
Altered sensorium	3	7.1%

Co-morbid clinical conditions included isolated hypertension (HT) (12, 28.6%)), renal disease (7, 16.7%), seizure disorder (4,9.5%), ischemic heart disease (3, 7.1%), Gram negative septicemia (3, 7.1%), and one each of rheumatic heart disease and pituitary adenoma. In addition, 9 cases had combination of > 1 lifestyle or medical condition as in **Table 2.**

Table 2: Co-morbid conditions in study cases (N=42)

HYPERTENSION	DIABETES	ALCOHOL USE	TOBACCO USE	NO.OF CASES
√	√	--	------	4
√	--	√	-	1
√	√		√	1
-	√	√		1
-	-	√	√	1
√	-	√	√	1
				9 TOTAL

Radiological findings were available in 13 of 42 (28.5%). They showed lobar hemorrhage LH (5), intraventricular hemorrhage IVH (2) and subarachnoid membrane SAH (1), cerebellar hemorrhage (2) and brainstem hemorrhage (3).

Gross organ findings:

1] LH was commonest type as shown in **Table 3.**

Table 3: Gross Brain findings

Brain Gross findings	Number of cases	%
Lobar hemorrrhage	20	47.6
Brainstem haemorrhage	10	23.8
Ganglio-capsular hemorrhage	6	14.5
Cerebellar hemorrhage	2	4.7
SAH	2	4.7
IVH	2	4.7
TOTAL CASES	42	100

Raised intracranial tension was evidenced by tonsillar herniation. Circle of Willis showed atherosclerotic thickening in 29 /42 cases (69 %). None of the cases had skull fracture or dural sinus thrombosis **(Figure 1 a-e).**

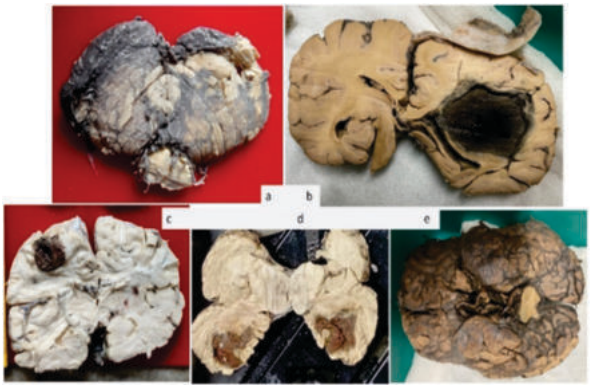

Figure 1 a- subarachnoid hemorrhage, b- gangliocapsular hemorrhage, c- lobar hematoma, d- cerebellar hemorrhage, e- atherosclerotic Circle of Willis

2] Kidney showed capsular adhesion and scarring with cortical thinning in 9 hypertensive cases, while 5 cases with endstage renal disease had bilaterally shrunken and scarred kidneys. One case each showed hydronephrosis and chronic pyelonephritis.

3] Cardiomegaly with left ventricular hypertrophy was noted in 11 cases, while 3 cases showed cardiomegaly, left ventricular hypertrophy and fibrous streaks in myocardium suggestive of chronic ischemia. One case had mitral stenosis with mitral regurgitation following rheumatic heart disease.

4] Hepatomegaly with fatty liver changes were noted in 6 cases of alcoholism.

MICROSCOPIC FINDINGS (**Figure 2 a-f**) corresponded to gross findings. HT related changes were evidenced by hyalinised arterioles with calcification. Hemorrhagic infarcts had reactive gemistocytes and gitter cells with surrounding ischemic red neurons.

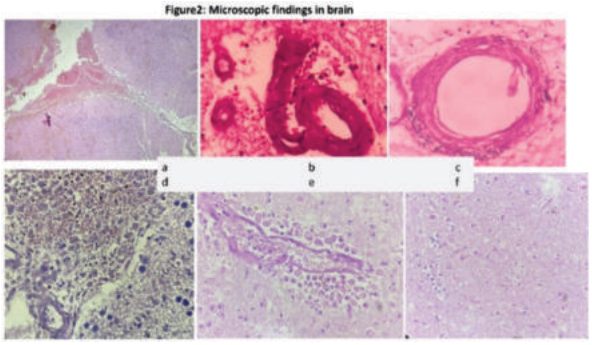

Figure 2: Microscopic images. a – subarachnoid hemorrhage, b- hyalinised vessels, c- vessels with calcification, d- hemorrhage with gemistocytes, e- perivascular gitter cells, f- red neurons

DISCUSSION-

Morillo et al reported cerebrovascular diseases to encompass 25.5% of all autopsies. Vilela et al reported that 15% of CNS stroke were following ICH. They reported 50% deaths occurring within 2 days after presentation with the incidence of ICH doubling every decade after 35 years.[1,3]

Faduyile reported 1.8:1 ration of M:F for sudden death cases ,as did Mbackwen. We noted a similar male preponderance. [4,5] Faduyile found highest HT related deaths in 5 th decade for male and 6th decade for female, like our findings. [4] Their mean age 52 years was higher than our study, perhaps attributable to earlier surveillance check-up in Indian patients.

Morillo etal reported ICH 50%, SAH 20%, disseminated petechiae 19% & IVH 3% with associated herniation in 74.1%cases. They found HT bleed in 33%, atherosclerosis 19% & arteriovenous malformation AVM 13%. [3] Fischben et al also reported commonest causes of HT, cerebral amyloid angiopathy (CAA), aneurysm & AVM. [6] Faduyile reported ICH +IVH in 12.4% hypertensive sudden death,35% brain stem hemorrhage & 2.5% SAH. Mbackwen also found hypertensive ICH as leading cause of death. [4,5] Barnaure found isolated IVH in premature infants while due to AVM, aneurysm of posterior inferior cerebellar artery, meningioma & trauma in adults.[7]

Vilela et al reported HT associated microscopic changes of lipohyalinosis, leucoaraiosis, lacunar infarcts & microbleeds. Their findings were in basal ganglia 65%, thalamus 26%, pons 5-10%cerebellum 6% & lobe 1-2%. [1] In contrast we found predominance of hemorrhage in lobar location. Although CAA is a common cause of lobar bleed, we did not find CAA.

Lammie et al noted associated micro atheroma & berry aneurysm in basal ganglia & pons following HT stroke. [8] We found florid atherosclerosis but no berry aneurysm, AVM, or CAA. At autopsy it is difficult to inspect vessel branches if the field is obscured by haemorrhage. Formalin fixation often hardens hematoma, thus preventing minute dissection to detect aneurysms. It is advisable to gently wash the hematoma at autopsy to facilitate scrutiny of vessel anomalies.

Lammie et al noted emotional stress, extreme cold weather & vasospasm to cause hypertensive bleed. Suzuki etal reported diabetic microangiopthy leading to hemorrhage in brain stem 29%, cerebral 63%, gangliocapsular 60% cases. They reported plaque formation and calcification in carotid artery.[8,9]

Weisberg etal reported alcohol related IVH & SAH. Patients presented as seizures or comatose state due to metabolic encephalopathy. Abnormal coagulation profile due to alcoholic liver damage led to the hemorrhage. [10] Morillo etal found HT cardiomyopathy in 86.2% & nephrosclerosis in 29 .3%. Faduyile etal noted heart failure in 80% cases of hypertensive haemorrhage, while Mbakwem noted in 40.9%. [3-5]

Vilela et al reported that magnetic resonance with angiography is essential for early detection of ICH. Spot sign is vital as it suggests growth of hematoma. Indications for surgical evacuation of hematoma include expansion, herniation, and hydrocephalus. [1] Mbakwen studied HT deaths and found 33% died within one hour of admission, like our finding. [5] Radiology was not possible in 71.5% of our cases due to rapid onset death.

CONCLUSION:

Advanced age, male gender, underlying hypertension, and renal disease found the highest association with ICH. Lobar and brainstem hemorrhages were commonest locations. Majority cases (71.4%) resulted in death < one hour post admission. This highlights the importance of early diagnosis and early surgical intervention in prevention of mortality.

REFERENCES:

1. Pedro Vilela P, Wiesmann M. Nontraumatic Intracranial Hemorrhage In: J. Hodler et al, eds. Diseases of the Brain, Head and Neck, Spine. IDKD Springer Series; 2020–2023
2. Lammie A. Hypertensive Cerebral Small Vessel Disease and Stroke. Brain Pathol 2002; 12:358-370
3. Arismendi-Morillo GJ, Fernandez-Abreu MC, Barrios GV, Molina- Victoria OM, Cardozo JJ. Cerebrovascular disease: its relationship with cardiovascular and renal pathology in 134 clinical autopsies. Rev Neurol 2004; 39:410-4
4. Faduyile FA, Soyemi SS, Sanni DA, Wright KO. Hypertension and Sudden Unexpected Deaths: An Autopsy Study of Four Hundred and Seventy-Seven Brought-in-Dead in a Tertiary Health Center. Niger Med J. 2019; 60:13-16.
5. Mbakwem AC, Oke DA, Ajuluchukwu JN, Abdulkareem FB, Ale O, Odunlami K. Trends in acute emergency room hypertension related deaths: An autopsy study. Niger J Clin Pract. 2009; 12:15–9
6. Fischbein NJ, Wijman CA. Nontraumatic intracranial haemorrhage. Neuroimaging Clin N Am. 2010; 20:469-92.
7. Barnaure I, Liberato AC, Gonzalez RG, Romero JM. Isolated intraventricular haemorrhage in adults. Br J Radiol. 2017; 90:20160779.
8. Lammie GA, Lindley R, Keir S, Wiggam MI. Stress-related primary intracerebral hemorrhage: autopsy clues to underlying mechanism. Stroke 2000; 31:1426-8
9. Suzuki K, Goto Y. Diabetic macroangiopathy in the elderly. Nihon Ronen Igakkai Zasshi 1993; 30:272-6.
10. Weisberg LA. Alcoholic intracerebral haemorrhage. Stroke 1988; 19:1565-9.