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Case Report

Multi-septate cavitary lesion of the subcortical gray matter: structural dynamics in a cadaveric specimen

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ABSTRACT

Deep seated cortical lesions possess a distinct capacity to expand extensively within subcortical gray matter while leaving the superficial cortical topography intact, often causing significant clinical latency until terminal mass effect occurs. Routine dissection of a formalin preserved human brain revealed an incidental, massive right-sided subcortical cavitary lesion with the intact overlying cerebral architecture. Despite the external cortical topography, coronal sectioning demonstrated extensive destruction of lentiform nucleus, featuring ablation of putamen and a multi-septate cavitary network. This typical architecture represents secondary liquefactive necrosis from metabolic demands outpacing lenticulostriate vascular supply, morphologically distinguishing it from smooth-walled cavity of a chronic ischemic infarction. The expanding mass obliterated the right lateral ventricle and caused an impending subfalcine herniation. Mechanical force transmitted across the midline caused vertical elongation of contralateral thalamus along with contralateral lateral ventricle. This specimen provides direct gross morphological evidence of silent subcortical tumor expansion, lenticulostriate vessel vulnerability and trans-midline pressure vector, helping in differentiating neoplastic necrosis and ischemic stroke.

Keywords: Thalamic glioma, Lenticulostriate arteries, Subcortical gray matter

INTRODUCTION

The structural study of macroscopic neuroanatomy frequently reveals incidental anomalies and pathologies challenging the structural paradigm of cadaveric brain providing a direct bridge between morphology and clinical reality. The subcortical gray masses are present bilaterally in the white matter of cerebral hemisphere. The lentiform nuclei is one of the gray matters which is wedge shaped and is present between internal capsule medially and external capsule laterally.

The thalamus is another large gray matter that forms the major part of diencephalon. The thalami are situated one in each hemisphere laterally to the third ventricle and medial to internal capsule, connected to each other by interthalamic adhesion.[1]

Although an idealized version of neural architecture is described in literatures, the variations encountered in dissection halls offer a more accurate presentation of anatomical diversity.

Among these incidental findings, a peculiar cavitary lesion within the subcortical gray matter is of interest to both an anatomist and a neurosurgeon. When observed during dissection, these specimens serve as highly effective teaching tools.

This article presents a macroscopic documentation of an incidental, multilobulated subcortical cavity discovered during a routine anatomical examination, establishing a systematic approach to its differential diagnosis based on gross morphological features.

CASE REPORT

During a routine neuroanatomy specimen preparation session, on a formalin fixed human brain in the Department of Anatomy at a tertiary care center in central India, a large subcortical cavitary lesion was discovered during coronal sectioning. Clean, high-resolution images were recorded and marked (Figure1 and 2).

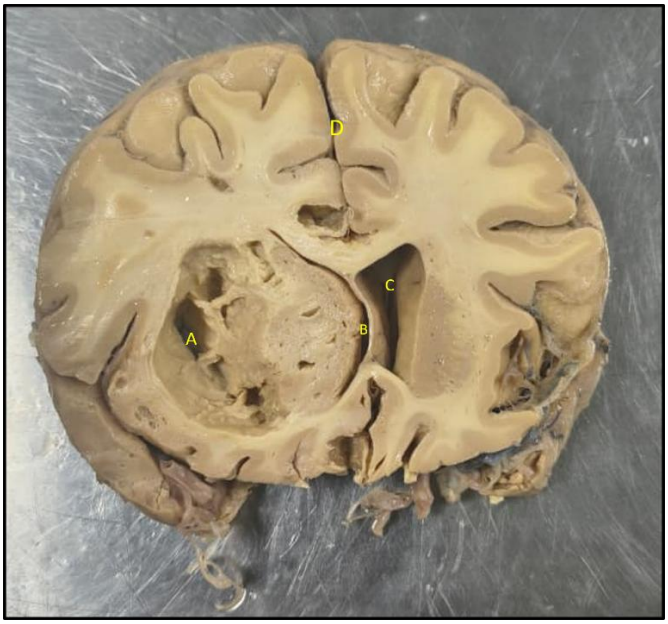


Figure 1: Coronal section of brain showing maximum rearrangement: (A) Thalamic lesion with septation (B) Obliterated right lateral ventricle (C) Left lateral ventricle-narrowed and elongated and (D) Midline shift.



Figure 2: Posterior surface of coronal section of brain at multiple level: (A) Cavitation, (B) Lentiform nuclei, shifted towards midline with putamen destroyed leaving a thin crescent shaped tissue behind and (C) Vertically elongated thalamus of contralateral side.

Gross examination of the intact brain revealed a midline displacement and hemispheric asymmetry, with the right hemisphere visibly bigger compared to left. The external surface of the specimen was structurally normal with no indication of any abnormality. The topography of the cortex was intact with all the sulci and gyri showing normal architecture, no wasting was seen.

The cavity was discovered when series of systematic coronal sections were made through cerebrum. A well demarcated single cavitary lesion was revealed, which was entirely in the subcortical gray matter of right hemisphere. The tumor showed extensive focal tissue loss precisely within the area of lentiform nucleus, globus pallidus was unidentifiable while a very thin crescent shaped putamen was present sharing its border with the posterior limb of internal capsule. The extensive ablation strongly indicates an ischemic occlusion or rupture of the ascending lenticulostriate branches of middle cerebral artery. The architecture of the cavity is what makes it visually compelling. The cavity presents as a stark punched out void showing sponge like network of fine tissue strands

and septations traversing the vault posteriorly, giving it spongy appearance. This meshwork corresponds to chronic, multifocal cystic degeneration and liquefactive necrosis. This distinct architecture is a classic macroscopic signature of a rapidly proliferating high grade aggressive tumor which outgrew its blood supply. This multiseptated appearance favors with the uniform, smooth walled, fluid filled cavity typically seen in chronic macrovascular ischemic infarction. The surrounding neural tissue shows remarkable transition zones maintaining the normal gray and white matter of brain. The surrounding parenchyma shows no softening or discoloration lateral to the mass.

The expanded mass on right hemisphere has exerted severe pressure causing compression and obliteration of the lumen of the right lateral ventricle leading to a significant midline shift forcing the right cingulate gyrus to squeeze under the rigid falx cerebri, capturing a classic view of impending subfalcine herniation. The massive pressure extended midline, deformation was seen in the left hemisphere as well. The left thalamus was compressed leading to its narrowing and vertical elongation reaching down towards midbrain junction. The left lateral ventricle was crushed into a long, narrow vertical slit parallel to the skewed midline structures.

DISCUSSION

This incidental finding of a massive, right sided thalamic glioma highlights the unique capacity of deep central nervous system tumors expansion within the subcortical area without disruption of the superficial architecture of the brain. Deep midline lesions can reach advanced stages of disease without causing cortical wasting or surface distortion, leaving the external topography of cerebrum intact.[2,3] This grossly normal exterior hides the internal structural reorganization completely masking any external footprint until the terminal stages of expansion.[4]

The striking features of the specimen, the stark, punched out cavity with multi-septate network represents the classical manifestation of secondary liquefactive necrosis within a malignant tumor.[5] As primary thalamic mass proliferates rapidly, the increasing volume and metabolic demands inevitable outpaces the vascular supply of the area, triggering chronic hypoxia, multifocal cystic degeneration and liquefactive necrosis, ultimately coalescing into a large cavitation.

The fine septations traversing the cavity represents the stromal elements that survives the initial parenchymal breakdown.[5,6] Liu et al also documented that thalamic glioma in adults frequently exhibit central cystic necrosis and local tissue breakdown.[6] Esquenazi et al reported that 93% cases had characteristic radiological feature of high-grade tumor with necrosis and three specific cases presented with large cystic mass.[7]

This destruction is related to the vascular anatomy of subcortical gray matter of the brain which is supplied by lenticulostriate arteries, perforating branches from middle cerebral artery.[8] The continuous tumor expansion leads progressive rise in pressure which compressed these tiny vessels causing secondary ischemia of neural tissue. This microvascular failure explains both the multi-septate appearance of the tumor and destruction of adjacent structures demonstrating that the tumor did not displace the basal ganglia but rather compromised their blood supply.[2] The cysts indicate that the growth of tumor outpaced its blood supply causing the breakdown and necrosis of the cysts. This typical appearance differentiates it from a chronic stroke where a uniform, clean and empty cavity is seen after the dead tissue is cleared.[9]

The massive tumor expansion completely flattened and obliterated the right lateral ventricle, obstructing the cerebrospinal fluid pathway, which was also reported to be the cause of hydrocephalus when seen radiologically in 44.5% cases of adult midline glioma.[10] This progressive volume expansion led to a midline shift, pushing the central structures across the median plane and into the left hemisphere, squeezing the right cingulate gyrus beneath the inferior margin of falx cerebri leading to a textbook presentation of an impending subfalcine herniation with contralateral ventricular dilatation due to compression of foramen Monro.[11]

The mechanical forces caused structural deformation in the contralateral hemisphere demonstrated by changes in left lateral ventricle into a long, narrow vertical slit running parallel to midline structures, emphasizing how a unilateral mass can alter the arrangement of ventricles and subcortical gray matter of opposite, unaffected hemisphere.

These findings closely align with clinical observation in adult neuro-oncology. Liu et al provided direct data from 26 adult patients of unilateral thalamic glioma, documenting that the high-grade thalamic lesions are highly prone to central tissue breakdown, liquefactive necrosis and cystic cavities due to aggressive growth pattern.[5] Menon et al established the force of transmission across midline by structural deformation and vertical elongation of contralateral thalamus which was seen in our case as well.[3] Furthermore, Waqar et al discussed the diagnostic challenges of deep midline lesions, as these lesions are present deep within the subcortical area, patients usually present with very few classical cortical signs until the mass becomes terminal.[12]

CONCLUSION

This gross anatomical specimen provides an invaluable demonstration of the structural mechanics of deep subcortical space occupying lesion, chronic liquefactive necrosis and overall effect of intracranial mass. The regional loss of lentiform nucleus highlights the vulnerability of perforating lenticulostriate branches to compression and encasement. Furthermore, development of multi-septate cavitation along with findings in

contralateral hemisphere such as thalamic elongation, ventricular slit formation and subfalcine displacement provides an insight into intracranial pressure vector, reminding of the extensive, silent structural reorganization possible within the deep gray matter of the brain. A limitation of this report is gross anatomical evaluation without comprehensive molecular study which would have aided in further characterization of adult deep midline tumor.

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REFERENCES

1. Splittgerber R. Snell's clinical neuroanatomy. 8th ed. Philadelphia: Wolters Kluwer; 2019.
2. Zhang P, Wang X, Ji N, Xie J, Han J, Ren X, et al. Clinical, radiological, and pathological features of 33 adult unilateral thalamic gliomas. World J Surg Oncol. 2016;14:78.
3. Cao L, Li C, Zhang Y, Gui S. Surgical resection of unilateral thalamic tumors in adults: approaches and outcomes. BMC Neurol. 2015;15:229.
4. Menon G, Nair S, Sudhir J, Rao BRM, Krishnakumar K. Bilateral thalamic lesions. Br J Neurosurg. 2010;24(5):566-71.
5. Louis DN, Perry A, Reifenberger G, von Deimling A, Figarella-Branger D, Cavenee WK, et al. The 2016 World Health Organization classification of tumors of the central nervous system: a summary. Acta Neuropathol. 2016;131(6):803-20.
6. Liu Y, Zhang Y, Hua W, Li Z, Wu B, Lu W. Clinical and molecular characteristics of thalamic gliomas: retrospective report of 26 cases. World Neurosurg. 2019;126.
7. Esquenazi Y, Moussazadeh N, Link TW, Hovinga KE, Reiner AS, DiStefano NM, et al. Thalamic glioblastoma: clinical presentation, management strategies, and outcomes. Neurosurgery. 2018;83(1):76-85.
8. Djulejić V, Marinković S, Maliković A, Jovanović I, Djordjević D, Cetković M, et al. Morphometric analysis, region of supply and microanatomy of the lenticulostriate arteries and their clinical significance. J Clin Neurosci. 2012;19(10):1416-21.
9. Bladin PF, Berkovic SF. Striatocapsular infarction: large infarcts in the lenticulostriate arterial territory. Neurology. 1984;34(11):1423-30.
10. Dono A, Takayasu T, Ballester LY, Esquenazi Y. Adult diffuse midline gliomas: clinical, radiological, and genetic characteristics. J Clin Neurosci. 2020;82(Pt A):1-8.
11. Riveros Gilardi B, Muñoz López JI, Hernández Villegas AC, Garay Mora JA, Rico Rodríguez OC, Chávez Appendini R, et al. Types of cerebral herniation and their imaging features. Radiographics. 2019;39(6):1598-610.
12. Waqar M, Hanif S, Rathi N, Das K, Zakaria R, Brodbelt AR, et al. Diagnostic challenges, management and outcomes of midline low-grade gliomas. J Neurooncol. 2014;120(2):389-98.
13. Fukami S, Nakajima N, Okada H, Akimoto J, Miki T, Fukuhara H, et al. Pathologic findings and clinical course of midline paraventricular gliomas diagnosed using a neuroendoscope. World Neurosurg. 2018;114.

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