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Case Report

Iatrogenic cerebral amyloid angiopathy: Two cases linked to childhood cadaveric dural transplantation for different intracranial pathologies, diagnosed using the simplified Edinburgh computed tomography criteria

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ABSTRACT

Background: Cerebral amyloid angiopathy (CAA) is an age-related condition marked by amyloid-β (Aβ) accumulation in the small cerebral vessels. Iatrogenic cerebral amyloid angiopathy (iCAA) is a distinct form of CAA in younger patients with a history of cranial surgeries involving cadaveric dural transplants. Both iCAA and CAA are linked to recurrent lobar intracerebral hemorrhage (ICH). This article highlights iCAA as a distinct variant, discussing the possibility of using simplified Edinburgh computed tomography (CT) criteria as a possible diagnostic tool for CAA and carefully considering plausible childhood surgery, with the risk of Aβ transmission through dural grafts in all, especially middle-aged patients.

Case Description: We present two cases of iCAA in a 46-year-old female and a 52-year-old male who suffered recurrent spontaneous lobar ICHs. The CAA was diagnosed using the simplified Edinburgh CT criteria, leading to further investigations into the underlying pathology. Based on their age, iCAA was suspected, and only after a meticulous search of the hospital documentation it was discovered that they both underwent cranial surgeries in childhood involving cadaveric dural grafts. The diagnosis of iCAA was established using the proposed diagnostic criteria by Banerjee *et al.* and later confirmed by pathological examination.

Conclusion: Our paper emphasizes the simplified Edinburgh criteria as a potential yet preliminary diagnostic tool for iCAA, while also highlighting the long-term risks of iatrogenic amyloid transmission related to dural grafting following various neurosurgical procedures.

Keywords: Amyloid-ß proteins, Cadaveric, Cerebral amyloid angiopathy, Dural allotransplants, Edinburgh criteria, Iatrogenic, Intracerebral hemorrhage, Lobar, Prion-like transmission, Recurrent, Simplified, Spontaneous

INTRODUCTION

Cerebral amyloid angiopathy (CAA), an age-related disorder, is characterized by the gradual accumulation of Aβ proteins, primarily in the walls of small cerebral blood vessels.[7] A recently recognized distinct subtype of CAA[2,32] is iatrogenic CAA (iCAA), which, in contrast, most commonly manifests in younger patients who have undergone neurosurgical procedures

involving cadaveric or bovine lyophilized dural transplants, typically during childhood.[24,26] This complex condition is associated with prion-like transmission of Aβ proteins through heterogeneous biological materials.[15] The CAA, including iCAA, is characterized by the development of spontaneous, relapsing, multi-lobar intracerebral hemorrhages (ICHs). Radiological confirmation of CAA typically relies on the magnetic resonance imaging (MRI)-based Boston criteria,[12] while the less commonly used computed tomography (CT)-based Edinburgh criteria[27] serve as an alternative. To distinguish iCAA from traditional CAA, it is crucial to establish a reliable history.

According to the literature, suspicion of iCAA should be raised mostly in younger patients presenting with recurrent lobar ICHs. However, iCAA should be suspected in all patients with lobar ICH who have a history of cranial surgery, especially if heterologous dural substitutes were used. Namely several descriptions of older patients with confirmed iCAA have been published recently.[22] Cranial surgery several decades ago should probably raise suspicion of iCAA and not the age of a patient *per se*. At present, the use of technologically advanced, mostly synthetic dural substitutes is strongly recommended to mitigate the future risk of iCAA transmission.[28] Therefore, such cases should be even less frequent in the future.

The aims of this article are twofold: first, to highlight iCAA as a distinct variant of CAA causing recurrent spontaneous lobar ICH, and second, to raise awareness about the potential risk of prion-like Aβ protein transmission through heterologous dural grafts while emphasizing the value of the simplified Edinburgh criteria as a possible alternative to the MRI-based Boston criteria.[7,13,14]

This article is based on the previously published two-case report from a single institution featuring patients diagnosed with iCAA by vascular neurologists and managed by neurosurgeons.[11,24]

MATERIALS AND METHODS

This paper presents two illustrative cases of iCAA[11,24] observed in middle-aged patients who underwent various neurosurgical procedures during childhood decades earlier, with documented use of cadaveric dural grafts. Both patients were diagnosed with spontaneously occurring lobar ICHs and were treated at the Department of Vascular Neurology and/or the Department of Neurosurgery at Ljubljana University Medical Center, Slovenia.

The presence of CAA was evaluated using the simplified Edinburgh criteria [27], while iCAA was assessed based on the proposed diagnostic criteria by Banerjee *et al*.[2]

The simplified Edinburgh criteria categorize brain CT findings into three probability tiers: high probability of CAA, characterized by lobar ICH accompanied by subarachnoid hemorrhage (SAH) and finger-like projections from the ICH; intermediate probability, defined as lobar ICH associated with isolated SAH; and low probability when lobar ICH occurs without accompanying SAH. A high probability of CAA, identified by the presence of all three CT predictors, demonstrates 100% specificity in confirming the association between CAA and lobar ICH.[27] The recently proposed diagnostic criteria for iCAA by Banerjee *et al*.[2] provide a framework for identifying cases as either probable or possible iCAA. The proposed diagnostic criteria for iCAA by Banerjee *et al*. include an onset age before 55 years, a history of potential exposure to cadaveric human central nervous system (CNS) tissues during relevant neurosurgical procedures, clinical and radiological features consistent with CAA, evidence of Aβ accumulation in CNS, and the exclusion of genetic causes of Aβ-related CNS disease.[3]

RESULTS

Case one

A 46-year-old female was admitted to the Department of Neurology at UMC Ljubljana with severe headache, vomiting, and left-sided hemiplegia, followed by rapid onset of stupor.[11] Her medical history included cranial surgery in childhood at the age of 9 for the resection of glioma from the left temporal lobe, followed by duraplasty using a cadaveric allograft. She was neither exposed to cadaveric human growth hormone nor received blood transfusions. Upon admission, she was diagnosed with a large, atypically located spontaneous ICH in the right temporal lobe, accompanied by SAH, as evidenced by CT brain imaging [Figure 1a]. The estimated baseline volume of the ICH, according to the ABC/2 equation[18], was 42 mL. Cerebral CT angiography (CTA) was performed at the time of admission and did not reveal any intracranial vascular pathology. Emergency neurosurgical intervention was performed, first with an attempt at ICH evacuation. During the time of surgery, the ICH was evacuated sufficiently. However, hemostasis proved impossible, and massive brain edema arose. An intraoperative decision was to perform a right-sided frontal-temporal decompressive craniectomy. A postoperative CT brain scan revealed large recurrent scattered ICHs and intraventricular bleeding with massive brain edema and midline shift [Figure 1b]. Several weeks later, follow-up postcontrast CT imaging showed multiple abscesses in the right hemisphere and ICHs with brain edema [Figure 1c]. The abscesses were partially removed during subsequent surgeries.

Based on the simplified Edinburgh CT criteria, and taking into account the substantial baseline volume of the ICH exceeding 40 mL, the probability of CAA was considered

highly likely, with a high risk for CAA-associated ICH. Several weeks later, follow-up CT imaging showed relapsing ICH in the right hemisphere with concomitant brain edema [Figure 1d]. Unfortunately, the patient ultimately succumbed to multiorgan failure 3 months after surgery. A postmortem examination confirmed multiple bilateral ICHs and SAH. Microscopic analysis revealed thickened and congested small leptomeningeal and cortical vessel walls. Immunohistochemical staining demonstrated Aβ deposits in the affected vessels, confirming the diagnosis of CAA and meeting the criteria for probable iCAA as defined by the proposed diagnostic criteria by Banerjee *et al.*[2]

Case two

A 52-year-old male was first admitted to UMC Ljubljana 7 years ago following a spontaneous ICH in the right frontal lobe.[24] [Figure 2a]. The estimated baseline volume of the ICH, according to the ABC/2 equation[18], was 16 mL. At the time of admission, the CTA was negative for intracranial aneurysms or arteriovenous malformations. Over the following years, he experienced three additional consecutive multilobar ICHs, each accompanied by SAH, as confirmed by brain CT imaging [Figures 2b and c]. Despite these recurrent hemorrhagic events, the patient was not selected

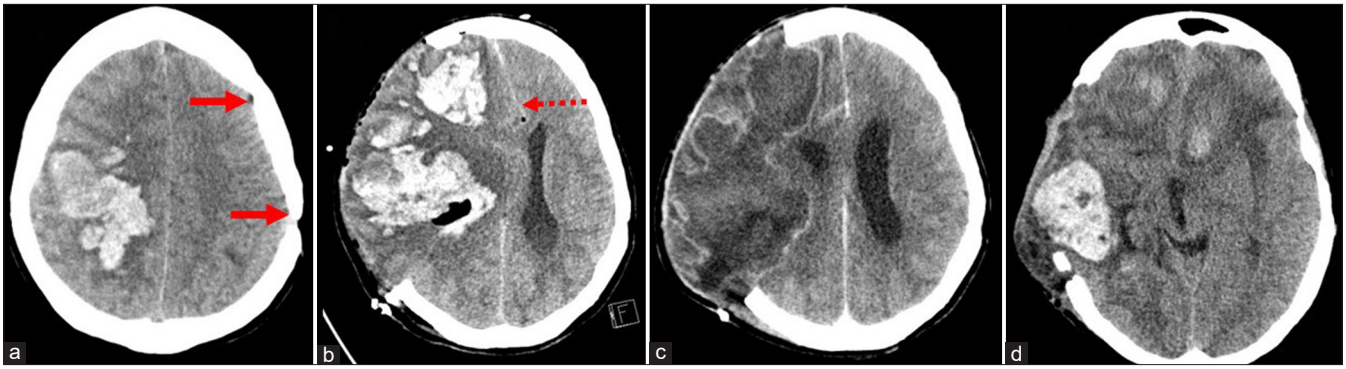

Figure 1: (a) A head computed tomography (CT) scan of the patient at the time of admission. A large intracerebral hemorrhage (ICH) in the right fronto-parietal area can be seen, extending down into the basal nuclei. The right hemisphere is compressed and liquor spaces are obliterated, as well as the right lateral ventricle. In the left temporo-frontal area, the deformed cranial bone can be seen after previous surgery in childhood (arrows). (b) Postoperative CT scan of the same patient. An area of decompressive craniectomy with numerous scattered ICHs is evident. Most of the right hemisphere is filled with an area of irregular hematoma with adjacent edematous brain substance. Pronounced extracranial herniation of virtually the entire right cerebral hemisphere and pronounced left-sided subfalcine herniation is discernible (dotted arrow). (c) The control postcontrast CT scan was taken a few weeks after the initial insult. Extensive scattered new abscesses with surrounding edema in the right cerebral hemisphere can be seen. (d) The last CT scan. A relapsing ICH in the right hemisphere is present, as is edema, and pronounced right to left shift of the brain substance.

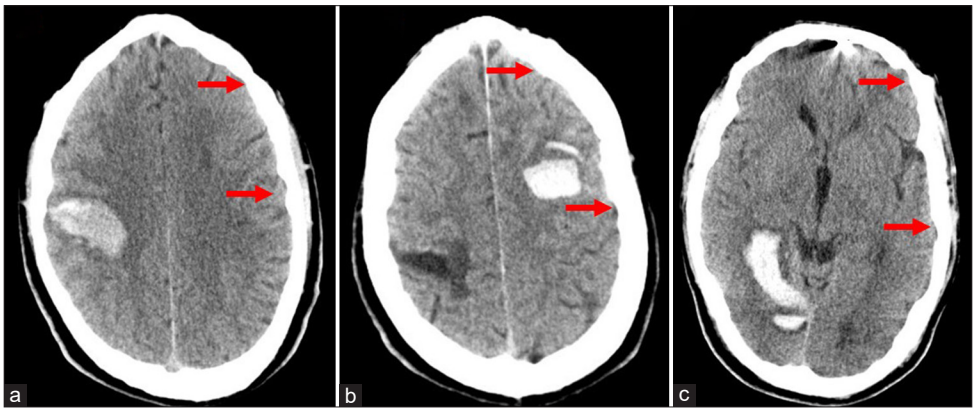

Figure 2: (a) The initial brain computed tomography (CT) showing a large intracerebral hemorrhage (ICH) in the right frontal corticosubcortical area. In the left frontopolar area, an up to 2 cm large area of cortical atrophy and encephalomalacia of the inferior white matter is discernible – a condition after old traumatic brain injury following surgical intervention (arrows). (b) The second bleeding revealed an ICH in the left frontal area. Hypodense area in the right parietal area can be seen – a condition after previous bleeding. (c) The third intracranial bleeding is documented in a CT scan. A large fresh hematoma in the right occipitotemporal area with minimal surrounding edema is present (a-c: arrows).

for neurosurgical intervention. All intracranial bleedings resorbed spontaneously, and no neurological consequences were observed. Amyloid positron emission tomography scans revealed moderate amyloid deposition in the brain's gray matter.

At the age of 7, the patient sustained a skull fracture and brain contusion following a sledding accident, necessitating neurosurgical intervention through osteoplastic craniotomy. The procedure was completed by alloplastic dural grafting since the dura and the underlying brain substance were also severed. He was not exposed to cadaveric human growth hormone, but he did receive blood transfusions. The patient recovered well after this injury and did not experience any neurological consequences.

Based on the simplified Edinburgh CT criteria, regarding the smaller volume of the ICH, the diagnosis of CAA was considered likely, and according to the diagnostic criteria proposed by Banerjee *et al*., it was assessed as possible iCAA.[2]

DISCUSSION

Our paper presents two cases of iCAA in middle-aged patients who underwent neurosurgical procedures in childhood for different conditions. In both cases, cadaveric dura was used during surgery as a dural substitute for brain covering. In the first patent, the initial cause of the surgery was a low-grade glioma, while in the second, the allograft dura was implanted for the treatment of traumatic brain injury. Decades later, both patients developed early-onset CAA. The preliminary diagnosis in both cases was made using the simplified Edinburgh CT criteria, based solely on brain CT imaging, with the definitive diagnosis of CAA in Case 1 confirmed postmortem through autopsy. The Edinburgh criteria seem to have good sensitivity (over 80%) with an ICH volume of ≥40 mL, whereas in ICH volumes <15 mL, the sensitivity is below 50%.[29] Therefore, we applied these criteria with caution, especially in case two, with initially a small-sized lobar ICH.

The proposed diagnostic criteria by Banerjee *et al*. provided a reliable framework for classifying the condition as either probable or possible, with the first case classified as probable and the second one as possible.[2]

Our paper highlights two key points: first, cadaveric dural grafts were used in childhood for different pathologies, and second, both patients later developed iCAA, underscoring the potential long-term risks associated with dural transplantation. Importantly, the initial diagnosis in these cases was made using the simplified Edinburgh CT criteria, which, although less commonly cited than the MRI-based Boston criteria, may become a possible diagnostic tool. However, the CT-based Edinburgh criteria reliability is significantly dependent upon the ICH volume. Therefore, they should be applied with caution in small-sized lobar ICHs.[21,29]

Prion diseases are transmissible neurodegenerative disorders characterized by the accumulation of abnormal prion proteins in the brain.[25] Creutzfeldt-Jakob disease, the most common human prion disorder, is a type of transmissible spongiform encephalopathy with an estimated annual incidence of 1–1.5 cases per million.[4] First described in 1922 by German neurologists Creutzfeldt and Jakob,[19] this disease can occur sporadically, be inherited, or result from acquired causes. The iatrogenic form of CJD (iCJD) results from unintended surgical exposure to prions. The primary sources of iatrogenic prion transmission were initially identified in the 1960s and include cadaveric dura grafting and contaminated human growth hormone.[5] Other occasional sources include corneal transplants,[9] contaminated neurosurgical instruments,[4,6,10] and blood transfusions.[8,23,31] Several outbreaks of iCJD associated with dura grafts have been documented, with incubation periods ranging from 1 to 14 years.[10] Although these outbreaks peaked in the 1990s, they have steadily declined since then.[5]

An age-related prion-like disorder, CAA is characterized by the gradual accumulation of Aβ proteins in the walls of small cerebral vessels. Advancing age is the primary risk factor for its development.[7] The simplified Edinburgh criteria for CAA diagnosis involve brain CT findings, including spontaneous lobar ICH, SAH, and finger-like projections from ICH.[27] Indeed, the application of these CT-based criteria should take into account the volume of ICH since with small-volume ICHs, their sensitivity is below 50%.[29] A recently recognized variant, iCAA, is associated with early-onset spontaneous ICH, distinct from traditional CAA.[3] Our first case met the simplified Edinburgh criteria for CAA, indicating a high risk for CAA-associated ICH. However, our second case, due to its reasonably small ICH volume, could be regarded as a borderline regarding the low sensitivity of the CT-based Edinburgh criteria for ICH volumes below 15 mL. Sharing a similar pathophysiological mechanism with CJD, iCAA involves Aβ protein transmission that facilitates amyloid deposition.[2]

Distinguishing iCAA from sporadic CAA in this paper was grounded on a documented history of the disease. The manuscript's findings are specific to iCAA because our patients of relatively younger age have undergone cranial surgeries during their childhood and met the simplified Edinburgh criteria (with the second case being "borderline"

due to a small baseline ICH volume), which may correlate well with the proposed diagnostic criteria by Banerjee *et al*. However, it is important to note that the proposed criteria by Banerjee *et al*. have not been validated.[2] Prions and Aβ proteins are highly resistant to standard decontamination and sterilization methods, posing a significant challenge in controlling iatrogenic transmission.[20] As a result, stringent preventive protocols are crucial to minimizing the risk of iCJD and iCAA transmission.

Historically, neurosurgeons and suppliers of surgical materials prioritized achieving an adequate seal for dural defects, often overlooking the long-term risks of iatrogenic transmission. Dural grafting was commonly performed using autografts or cadaveric allografts, including human and bovine lyophilized transplants. Years ago, when our two patients underwent surgical treatment, scanning for prion particles was not available in Slovenia, and awareness of this type of transmission was low.

From the neurosurgical point of view, there is uncertainty surrounding the risk of iatrogenic transmission of CJD and the effectiveness of strategies to mitigate the iCAA risk, which remains rare in standard clinical practice. An overview of commercial cadaveric dural substitutes shows a gradual decline in their use in neurosurgical practices over the past 50 years,[5] with several artificial materials being researched to address their limitations. In recent years, the use of cadaveric dura is in decline. In Slovenia, it is not used at all during neurosurgical procedures. Instead, polymeric, bioabsorbable, and bioinert dural substitutes have been introduced as alternatives, which are safer, more easily obtainable, and reduce the potential risk of iatrogenic transmission.[28] In both of our cases, cadaveric dural grafting was documented, which may be the cause of iCAA in the presented patients.

In the early 1990s, while assessing optimal dural closure techniques in patients with war-related brain injuries, we observed the benefits of autologous dural grafts over cadaveric transplants, such as faster healing and reduced cortical scarring, potentially lowering the risk of posttraumatic seizures.[30] However, we did not foresee that cadaveric dural grafting could result in iCAA as a delayed consequence years later.

The ideal dural substitute should effectively restore the integrity of a dural defect while demonstrating biomechanical properties comparable to those of autologous transplants. It should facilitate autologous healing and support structural and functional repair, promoting healthy tissue growth to prevent cerebrospinal fluid leaks and inhibit cortical scarring.[17] To satisfy the criteria of a perfect dural substitute, natural, synthetic, and composite polymers have been meticulously investigated recently.[16,17] However, cranial dural repair appears to be linked to mild to moderate complication rates regardless of the type of dural substitute used.[1]

The limitations of this study include its retrospective design, reliance on previously published cases, and the single-center nature with a small sample size. In addition, in the second case, a biopsy was not performed to obtain a definitive CAA diagnosis. Given this consideration, the conclusions drawn must be interpreted with caution.

To deepen our understanding of the clinical and pathological features of iCAA, further research involving larger, multi-center studies is essential, although this may be problematic due to the avoidance of cadaveric transplants (dural grafts) in neurosurgery, particularly in recent years as awareness of CJD transmission has increased. At present, the use of modern and safer synthetic or organic allogeneic dural substitutes is becoming increasingly common.

CONCLUSION

iCAA, a distinct form of CAA, is characterized by earlier symptom onset in younger patients. This emerging condition should be considered when prion-like disease transmission is suspected following various neurosurgical procedures. The simplified Edinburgh CT criteria can be used to evaluate the diagnosis of CAA, while the proposed diagnostic criteria by Banerjee *et al*. may offer an accurate, though not validated, method for identifying iCAA.

It is important to raise awareness in patients with a history of exposure to prion-contaminated materials, such as cadaveric or bovine lyophilized dural grafts. Although the risk of transmission has decreased with advanced techniques and polymeric dural substitutes, caution remains essential. Implementing preventive measures is vital to reducing the risk of iCJD and iCAA transmission in neurosurgical patients.

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