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Response to the reported cases of "cerebral amyloid angiopathy" in patients with a history of cadaveric dura mater graft

I. Introduction

1) The product

Cadaveric dura mater is a medical device primarily used to replace the dura mater resected during brain surgery (Table 1). It is estimated that over 200,000 patients have received dura mater graft in Japan between 1983 and 1997.[1)] In March 1997, at least 50 cases of Creutzfeldt-Jakob disease (CJD), or transmissible spongiform encephalopathy, were reported in patients who had received cadaveric dura mater graft worldwide. Following the report, a recommendation not to use cadaveric dura mater was issued by the World Health Organization (WHO) expert meeting on medical products for transmissible spongiform encephalopathy. In Japan, an emergency order to discontinue the use of cadaveric dura mater and recall the product was issued.

Table 1. Medical devices containing cadaveric dura mater as a raw material

Brand name	Lyophilized Dura	Tutoplast Dura
Importer/Distributor	Nihon BSS	Sata Corporation
Approval number (approval date)*	48B Import No. 593 (July 23, 1973)	60B Import No. 764 (August 6, 1985)
Annual sales (then)	About 11,000 pieces	About 7,000 to 10,000 pieces
Name of the manufacturer (country) and overseas brand name	B. Braun Melsungen (Germany) LYODURA	Biodynamics International (Germany) Tutoplast Dura

*Approved under the old Pharmaceutical Affairs Act

2) Cerebral amyloid angiopathy (CAA)

CAA is cerebrovascular amyloid deposition seen in the elderly and in patients with Alzheimer's disease (AD). It may be non-hereditary or hereditary and is known to be one of the causes of cerebral hemorrhage and cerebral infarction.[2)] It is usually seen in the

elderly. According to the Boston criteria (Table 2), age 55 or older is one of the diagnostic criteria for possible and probable CAA-related cerebral hemorrhage.[3)] Onset of iatrogenic cerebral amyloid angiopathy (iCAA) after 30 to 40 years of incubation period has been reported in recent years in patients who had head trauma, underwent brain surgery, or received cadaveric dura mater graft in their childhood.[4)] The elements of the diagnostic criteria for iCAA proposed by Gargi Banerjee and colleagues in 2022 (Table 3) include age of onset < 55 years and a medical history suggesting exposure, such as a procedure using cadaveric human central nervous system tissues (i.e., brain, meninx, and pituitary hormone) or a neurosurgical intervention (i.e., brain, spinal cord, and posterior eye segment).[4)5)]

iCAA is now recognized to be caused by the spread of Aβ seeds after the first pathological description of amyloid β (Aβ) propagation in 2015 and the experimental confirmation in 2018.[5)] Although the mechanism of onset is still unknown, iCAA is thought to be possibly induced by Aβ seeds directly spread from the cadaveric central nervous system tissues. Possible preferential deposition of Aβ from the surface layer of the brain in the meninx and in the surface cortical vessel is suggested.[4)] Onset of CAA has also been reported in patients who had not received cadaveric dura mater graft. Possible spread of Aβ from the surgical instruments contaminated with Aβ seeds is suggested in patients who had received brain surgery. In patients with a history of head trauma not treated with neurosurgery, possible Aβ accumulation associated with the failure of the excretory mechanism, i.e., the intramural peri-arterial drainage (IPAD) or glymphatic system, is suggested.[4)]

Table 2. Boston criteria for CAA (revised version)[3)]

Definite CAA
Full postmortem examination demonstrating:
Lobar, cortical, or cortical–subcortical hemorrhage
Severe CAA with vasculopathy
Absence of other diagnostic lesion
Probable CAA with supporting pathology
Clinical data and pathological tissue (evacuated hematoma or cortical biopsy) demonstrating:
Lobar, cortical, or cortical–subcortical hemorrhage (including ICH, CMB, or cSS)
Some degree of CAA in specimen
Absence of other diagnostic lesion
Probable CAA
Clinical data and MRI or CT demonstrating:
Multiple hemorrhages (ICH, CMB) restricted to lobar, cortical, or cortical–subcortical regions (cerebellar hemorrhage allowed), or single lobar, cortical, or cortical–subcortical hemorrhage and cSS (focal or disseminated)
Age ≥55 y
Absence of other cause of hemorrhage*
Possible CAA
Clinical data and MRI or CT demonstrating:
Single lobar, cortical, or cortical–subcortical ICH, CMB, or cSS (focal or disseminated)
Age ≥55 y
Absence of other cause of hemorrhage*

CAA indicates cerebral amyloid angiopathy; CMB, cerebral microbleed; cSS, cortical superficial siderosis; CT, computed tomography; ICH, intracerebral hemorrhage; and MRI, magnetic resonance imaging.

*Other causes of hemorrhage (differential diagnosis of lobar hemorrhages): antecedent head trauma, hemorrhagic transformation of an ischemic stroke, arteriovenous malformation, hemorrhagic tumor, warfarin therapy with international normalization ratio >3, and vasculitis.

Table 3. Proposed diagnostic criteria for iCAA[4)]

Proposed diagnostic criteria for iatrogenic cerebral amyloid angiopathy (CAA)
1. Age of onset ⇒ Symptom onset before age of 55 years (ie, below the age threshold for 'probable' or 'possible' CAA within the modified Boston criteria); *strongly suggestive (although note ascertainment bias)* ⇒ *Note: diagnosis cannot be excluded based on age alone, and should be considered in people aged 55 years or above, should they meet the other criteria (detailed below)* 2. History of potential exposure; one or more of the following: ⇒ Procedure or treatment using cadaveric human CNS tissues (ie, brain, meninges, pituitary-derived hormones); *strongly suggestive* ⇒ Relevant neurosurgical procedure (ie, those involving the brain, spinal cord, posterior eye) ⇒ *Note: diagnosis can be considered if history of alternative potential exposure and all other criteria are met* 3. Clinical and radiological features consistent with a diagnosis of CAA: Clinical: ⇒ Evidence of at least one of the following features, either at presentation or during disease course: ⇒ Intracerebral haemorrhage or convexity subarachnoid haemorrhage (single or multiple) ⇒ Transient focal neurological episodes ('amyloid spells') ⇒ Focal seizures (with or without secondary generalisation) ⇒ Cognitive impairment not attributable to another cause (including acute stroke) Radiological; at least one of the following: ⇒ CT: ⇒ Lobar intracerebral haemorrhage ⇒ Convexity subarachnoid haemorrhage ⇒ MRI (blood sensitive sequences; T2*-GRE, SWI) ⇒ Cerebral microbleeds with predominantly lobar distribution, distant from sites of parenchymal intracerebral haemorrhage ⇒ Cortical superficial siderosis (focal or disseminated) on MR blood sensitive sequences 4. Evidence of amyloid-beta (Aβ) accumulation in the CNS: ⇒ Positive amyloid-PET scan (note this is not specific for vascular Aβ deposition) ⇒ Supportive CSF features (reductions of Aβ−42, Aβ−40) ⇒ Brain biopsy demonstrating vascular Aβ deposition, in the absence of significant inflammation ⇒ *Notes raised:* ⇒ *A positive amyloid-PET scan in isolation might not necessarily be specific for Aβ accumulation, depending on the tracer used; correlation with either CSF Aβ measures, brain biopsy findings and/or genetic testing for non-Aβ CAAs (details below) is advised* ⇒ *Presence of significant inflammation might support an alternative diagnosis of CAA-related inflammation or Aβ related angiitis (ABRA)* 5. Exclusion of genetic causes of Aβ CNS disease; this should include:

⇒ Duplications of *APP* (including Trisomy 21, where relevant)
⇒ Mutations of *APP, PSEN1, PSEN2*
⇒ In cases where CNS Aβ deposition has not been confirmed by other means (CSF Aβ measures, brain biopsy), next-generation sequencing for mutations resulting in non-Aβ CAA (*CST3, TTR, GSN, PRNP, ITM2B*) should be considered

In order for a diagnosis of probable iatrogenic CAA to be made during life, criteria 2, 3, 4 and 5 must be met as a minimum. Features in the history which are strongly suggestive of the diagnosis are highlighted.
A diagnosis of possible iatrogenic CAA can be considered if criteria 1, 2 and 3 are met.

CNS, central nervous system; CSF, cerebrospinal fluid; GRE, gradient recalled echo; PET, positron emission tomography; SWI, susceptibility weighted images.

3) AD

AD is a progressive neurodegenerative disease in which Aβ and phosphorylated tau accumulate in the brain, leading to cognitive decline. CAA affects the cognitive function independently of AD. Cognitive dysfunction such as a decrease in information processing speed and executive function is reported in 20% to 30% of CAA patients before the onset of cerebral hemorrhage. In a clinicopathological study in the general population, CAA is highly frequent in patients with dementia. The frequency of dementia is particularly high in patients with severe CAA. Although the pathophysiological mechanism of cognitive dysfunction caused by CAA has not been fully elucidated, involvement of neurodegeneration caused by CAA itself following chronic cerebral hypoperfusion, decreased vascular reactivity, microhemorrhage, microinfarction, and white matter lesion is suggested.[6)]

II. Background

In March 2025, the PMDA received a report on a case of cerebral hemorrhage associated with CAA likely caused by human dura mater graft in childhood from a neurosurgeon and related recent study reports.[2)4)5)] A literature review was performed in response.

III. Results of the PMDA review

1) Review of literature on CAA

Case reports on CAA that met one of the proposed diagnostic criteria for iCAA, age of onset < 55 years or a medical history suggesting exposure “A. a procedure or treatment using cadaveric human central nervous system tissues (i.e., brain, meninx, and pituitary

hormone)" or "B. a neurosurgical intervention (i.e., brain, spinal cord, and posterior eye segment)," were reviewed based on the recent study reports (including references) described in 2. Background[2)4)5)] and a report from the Ministry of Health, Labour and Welfare designated research (review of literature on early onset, non-hereditary CAA that developed before the age of 55) described below.

The literature review found 54 CAA cases (including 9 cases in Japan) that met one of the criteria.[2)4)5)7)-34)] Twenty-three of the cases (4 cases in Japan) were of CAA that developed after cadaveric dura mater graft. The age at the onset of the initial symptoms suggestive of CAA was 27 to 71 years, and the time from dura mater graft to onset of the initial symptoms suggestive of CAA was 25 to 49 years (excluding cases lacking the relevant information).[4)5)8)10)13)14)16)22)-28)32)-34)] Twenty-five of the cases (5 cases in Japan) were of patients who underwent neurosurgery with no documented cadaveric dura mater graft. The age at the onset of the initial symptoms suggestive of CAA was 29 to 84 years, and the time after surgery to onset of the initial symptoms suggestive of CAA was 28 to 46 years (excluding cases lacking the relevant information; see Appendix 1 for details).[2)7)9)11)12)15)17)18)20)21)29)34)]

2) Results of the review on early onset, non-hereditary CAA by the MHLW-designated research group

In Japan, the Surveillance Committee of the MHLW "Research Group on Late-onset Viral Infections" was established in 1999 after the report from the UK on variant CJD in human likely to be associated with consumption of beef contaminated with bovine spongiform encephalopathy virus in 1996. In 2010, the Research Committee on Surveillance of Prion Disease and Infection Control (hereinafter referred to as "the Research Committee") appointed by the MHLW started the review research focusing on the surveillance of prion disease and the infection prevention.

Two sets of results of the review research conducted by the Research Committee, including classification of dura mater graft sites (central and peripheral), are presented below.

Thirty-three cases of early-onset (age 27 to 53), non-hereditary CAA reported in the literature by the end of September 2022[5)12)18)34)] were reviewed in the 2022 collaborative research “Onset of mechanism of early-onset, non-hereditary cerebral amyloid angiopathy (CAA)” to elucidate the mechanism of onset of early-onset, non-hereditary

CAA (see Figure 1 for the details of the cases). Twenty-six patients had central nervous system diseases (mainly traumatic brain injury) in childhood. Of them, 21 patients underwent brain surgery, and 7 of the 21 patients received cadaveric dura mater grafts. Of 7 patients who had no history of central nervous system disease in childhood, 5 patients underwent embolization with cadaveric dura mater for non-central nervous lesions such as hemangioma (n = 4) or received dura mater graft during heart surgery (n = 1). The time from brain surgery/dura mater use (at age 0 to 17) to onset of CAA (at age 37 to 51) was 25 to 47 years in 26 patients with a history of brain surgery or dura mater graft.[35)]

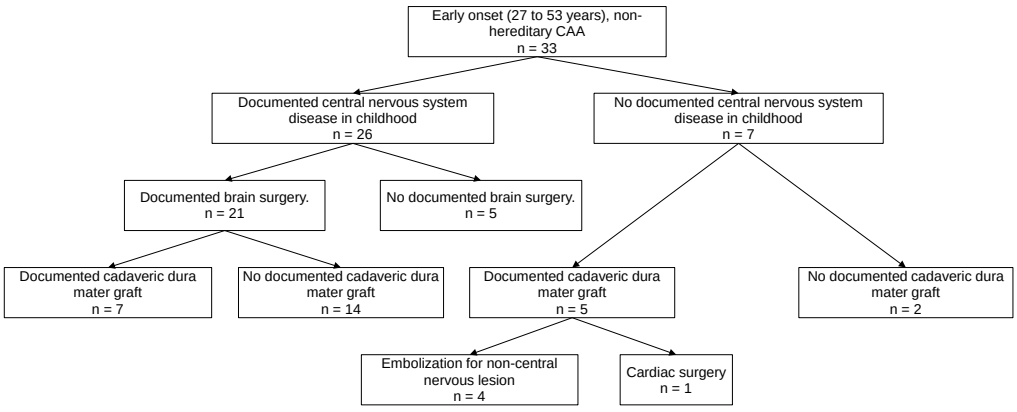

Figure 1. Breakdown of cases reported in Reference 35 (Figure created by the PMDA)

Sixteen cases of non-hereditary CAA that developed before age 55 in patients who had received cadaveric dura mater graft during medical procedures in childhood (more than half of them had received graft between 1972 and 1987) reported in the literature by the end of December 2023[8)10)11)13)14)16)20)-25)] were reviewed by focusing on the association with the dura mater graft site in the 2023 collaborative research to elucidate the mechanism of Aβ spread in human. The dura mater graft sites included the central nervous system in 11 patients (brain surgery for head injury in 7 patients, tumor in 2 patients, and others in 2 patients) and the periphery in 5 patients (embolization for hemangioma in 4 patients, surgery for transposition of the great vessels in 1 patient). The initial symptoms of CAA in 11 patients who received dura mater graft in the central nervous system included lobar intracerebral hemorrhage (ICH) in 10 patients and convulsive seizure in 1 patient. Of 5 patients who received dura mater graft in the

periphery, 3 patients had ICH, 1 patient had convulsive seizure, and 1 patient had transient local neurological symptoms as the initial symptoms of CAA. There was no significant difference in age at which the patients received dura mater graft during medical procedures, age of CAA onset, or time from dura mater use to CAA onset between the patient groups. Comparison of the dura mater graft site in the central nervous system and the site of initial onset of ICH found the sites were consistent or overlapped in 4 patients, inconsistent in 6 patients, and unknown in 1 patient. There was no clear association between the dura mater graft site and the site of CAA-related ICH.[36)]

3) AD-type pathological findings

The literature review found 2 cases of AD-type pathological findings (cortical parenchymal Aβ and tau neurofibrillary tangles), which is rare in patients under 50 years, among patients who had CAA 30 to 40 years after undergoing neurosurgical procedures using cadaveric dura mater in early childhood.[5)22)]

There has been no report on CAA in patients who received cadaveric dura mater graft among the medical device adverse event reports or the study reports from the manufacturers made in accordance with the Pharmaceuticals and Medical Devices Act since there is no marketing authorization holder manufacturing cadaveric dura mater at the moment. There has been no report from healthcare professionals other than those described in 2. Background.

IV. Overseas situation

No information on current or draft safety measures against iCAA onset associated with cadaveric dura mater was found among the public information provided by the overseas regulatory authorities.

V. Expert discussion

An Expert Discussion was held on May 2, 2025 by requesting attendance of the expert committee members listed below (Table 4) based on the literature review of the PMDA. Opinions of the expert committee members on the awareness of the neurology-related societies about CAA/iCAA (whether or not it has been discussed/studied), the causal

relationship with cadaveric dura mater, and the need for alert (target patients and contents) were asked. The following are the details of the Expert Discussion:

- The diagnostic criteria for iCAA proposed in 2022[4)5)] is merely a proposal made by some researchers. No consensus has been reached at the societies where the expert committee members are affiliated. Although the expert committee members are aware of the recent increase in case reports on iCAA onset in patients with a history of cadaveric dura mater graft, it has not been widely discussed or recognized at scientific conferences or symposia.
- Factors other than cadaveric dura mater graft, such as spread of Aβ seeds during surgery and failure of Aβ excretory mechanism, may be attributable to the onset of iCAA based on the information on the cases currently available from the literature review by the PMDA and the collaborative research of the research group supported by the Health and Labour Sciences Research Grants. The study report that categorized CAA as non-hereditary does not describe what kind of genetic testing was performed, and the possibility of CAA being hereditary cannot be completely ruled out. These circumstances do not provide evidence to evaluate the causal relationship.
- The report on the spread of Aβ seeds after blood transfusion does not discuss the association with blood transfusion, providing no evidence.
- About the relationship between CAA and AD, AD is known to accompany CAA, and post-CAA vascular dementia is also known. On the other hand, AD caused by CAA has not been discussed at the moment since the onset of AD is largely affected by tau protein as well as Aβ. The risk of AD following CAA is not very important because CAA itself is already serious.
- In the current situation where no sufficient evidence is available to evaluate the causal relationship, it is difficult to discuss the need for alert. How to investigate the causal relationship may include, for example, a surveillance using the database of the Japan Neurosurgical Society (Japan Neurosurgical Database, JND) and an epidemiological study rather than literature review in the collaborative research of the study group designated by the MHLW.
- There was an opinion that the framework for the database use would need to allow access of internists who may diagnose CAA as a challenge for conducting a surveillance because the JND is a surgical database. A new research project needs to be launched,

and the JND needs to be modified to allow for surveillance on CAA in advance if the JND is used.

- While patients suspected of CJD must be notified according to the "Act on the Prevention of Infectious Diseases and Medical Care for Patients with Infectious Diseases" (Act No. 114, 1998), CAA is not subject to notification. Consideration should also be given to thorough sharing of the objective and necessity of the surveillance with the relevant societies so that the physicians affiliated with the societies will be able to report and register cases of CAA appropriately.

Table 4. List of expert committee members: Expert Discussion

Name	Affiliation	Affiliated societies
Nobuhito Saito	Faculty of Medicine, The University of Tokyo Professor, Department of Neurosurgery	President, Japan Neurosurgical Society Board member, Japan Stroke Society
Shun Shimohama	Jiseikai Nerima Takanodai Hospital Extraordinary Director	Auditor, Japanese Society of Neurology
Kazunori Toyoda	National Cerebral and Cardiovascular Center Vice Director	Board member, Japan Stroke Society Board member, Japanese Society of Neurology
Shinichi Yoshimura	Hyogo Medical University Chief Professor, Neurosurgery Course	Councilor, Japan Stroke Society Board member, The Japanese Society for Neuroendovascular Therapy

VI. Summary

The Expert Discussion concluded that the literature review conducted by the PMDA provided no sufficient evidence to support a causal relationship between cadaveric dura mater graft and CAA based on the currently available information. The causality therefore cannot be determined. The following is the PMDA's opinion based on the Expert Discussion:

- An epidemiological study on the usage history of cadaveric dura mater in CAA

patients should be conducted to accumulate evidence and evaluate the causal relationship.

- Who to be alerted and how to alert them should be considered if evidence for the evaluation of causal relationship becomes available.

References

1) Nakamura Y, et al. Relative risk of Creutzfeldt-Jakob disease with cadaveric dura transplantation in Japan. Neurology 53 218-220, 1999
2) Taketani S, et al.: A case of recurrent subcortical hemorrhage in iatrogenic cerebral amyloid angiopathy after surgery for head injury in childhood and review of literature on a case in Japan. Current Practical Neurosurgery vol. 35 no. 1 e20250501f, 2025
3) Greenberg SM, et al. Diagnosis of Cerebral Amyloid Angiopathy: Evolution of the Boston Criteria. Stroke 49: 491-497, 2018
4) Furutsuka K, et al.: A case of premature cerebral amyloid angiopathy considered to be associated with dura mater graft. Clinical Neurology 64: 736-741, 2024
5) Banerjee G, et al. Iatrogenic cerebral amyloid angiopathy: an emerging clinical phenomenon. J Neurol Neurosurg Psychiatry 93: 693-700, 2022
6) Ii Y, et al.: Cerebral amyloid angiopathy. Neurological Therapeutics 39: 187-191, 2022
7) Ehling R et al. Recurrent intracerebral haemorrhage after coitus: a case report of sporadic cerebral amyloid angiopathy in a younger patient. Eur J Neurol 19(3):e29-31, 2012
8) Herve D et al. Fatal Abeta cerebral amyloid angiopathy 4 decades after a dural graft at the age of 2 years. Acta neuropathologica 135(5):801-03, 2018
9) Jaunmuktane Z et al. Evidence of amyloid-beta cerebral amyloid angiopathy transmission through neurosurgery. Acta neuropathologica 135(5):671-79, 2018
10) Banerjee G et al. Early onset cerebral amyloid angiopathy following childhood exposure to cadaveric dura. Annals of neurology 85(2):284-90, 2019
11) Giaccone G et al. Iatrogenic early onset cerebral amyloid angiopathy 30 years after cerebral trauma with neurosurgery: vascular amyloid deposits are made up of both Abeta40 and Abeta42. Acta neuropathologica communications 7(1):70, 2019
12) Hamaguchi T et al. Cerebral hemorrhagic stroke associated with cerebral amyloid angiopathy in young adults about 3 decades after neurosurgeries in their infancy. Journal of the neurological sciences 399:3-5, 2019

13) Caroppo P et al. Cerebral amyloid angiopathy in a 51-yearold patient with embolization by dura mater extract and surgery for nasopharyngeal angiofibroma at age 17. Amyloid 28(2):142-43, 2021
14) Raposo N et al. Amyloid-β transmission through cardiac surgery using cadaveric dura mater patch. Journal of neurology, neurosurgery, and psychiatry 91(4):440-41, 2020
15) Tachiyama K et al. Infant critical head injury could be a remote cause of middle-aged cerebral amyloid angiopathy. Interdisciplinary Neurosurgery 22, 2020
16) Yoshiki K et al. Follow-up study of a patient with early onset cerebral amyloid angiopathy following childhood cadaveric dural graft. Acta Neurochir (Wien) 163(5):1451-55, 2021
17) Michiels L et al. The Role of Amyloid PET in Diagnosing Possible Transmissible Cerebral Amyloid Angiopathy in Young Adults with a History of Neurosurgery: A Case Series. Cerebrovascular diseases (Basel,Switzerland) 50(3):356-60, 2021
18) Oblak JP et al. Preceding head trauma in four cases of sporadic cerebral amyloid angiopathy - case report series. Stroke Cerebrovasc Dis 31:106260, 2022
19) Purrcker JC et al. Cerebral amyloid angiopathy--an underdiagnosed entity in younger adults with lobar intracerebral hemorrhage? Amyloid 20(1):45-7, 2013
20) Kellie JF et al. Amyloid-β (Aβ)-Related Cerebral Amyloid Angiopathy Causing Lobar Hemorrhage Decades After Childhood Neurosurgery. Stroke 53:e369-e374, 2022
21) Jaunmuktane Z et al. Alzheimer's disease neuropathological change three decades after iatrogenic amyloid-β transmission. Acta Neuropathol 142:211-215, 2021
22) Milani R et al. Spontaneous intracerebral haemorrhage associated with early-onset cerebral amyloid angiopathy and Alzheimer's disease neuropathological changes five decades after cadaveric dura mater graft. Acta Neuropathol Commun 11:30, 2023
23) Paku S, et al.: A case of subcortical hemorrhage due to premature cerebral amyloid angiopathy after head trauma surgery using cadaveric dura mater in childhood. Japanese Journal of Stroke 45:161-166, 2023.
24) Storti B et al. Iatrogenic cerebral amyloid angiopathy: An illustrative case of a newly introduced disease. Eur J Neurol 30:3397-3399, 2023
25) Fandier-Hofler S et al. Intracerebral haemorrhage caused by Iatrogenic cerebral amyloid angiopathy in a patient with a history of neurosurgery 35 years earlier. Lancet 402:411, 2023
26) Carla Vera-C´aceres, MD et al. Iatrogenic cerebral amyloid angiopathy: Two case reports to explore clinical heterogeneity and pathological patterns. J Stroke Cerebrovasc Dis

Jan;34(1): 107969, 2025

27) Hatakeyama Y et al. A case of cerebral amyloid angiopathy with ipsilateral tau and contralateral amyloid PET uptake related to cadaveric dura mater implanted in childhood. Eur J Nucl Med Mol Imaging 2024

28) Larysa Panteleienko et al. Iatrogenic cerebral amyloid angiopathy in older adults. Eur J Neurol 31(6):e16278, 2024

29) Nakayama Y et al. Cerebral amyloid angiopathy in a young man with a history of traumatic brain injury: a case report and review of the literature. Acta Neurochir (Wien) 159(1):15-18, 2017

30) Wong MP et al. Cerebral amyloid angiopathy and spontaneous intracerebral haemorrhage. Report of a sporadic case in a young Chinese. Clin Neurol Neurosurg 93(2):133-6, 1991

31) Campbell DM et al. Intracerebral hemorrhage caused by cerebral amyloid angiopathy in a 53-year-old man. J Neurol 255(4):597-8, 2008

32) Muller C. Case report of iatrogenic cerebral amyloid angiopathy after exposure to Lyodura: an Australian perspective. Front Neurosci 17:1185267, 2023

33) Sharma R et al. WHEN THE ANSWER IS HIDING IN THE MEDICAL RECORDS: IATROGENIC CEREBRAL AMYLOID ANGIOPATHY. 10.1136/bmjno-2024-ANZAN.125

34) Yamada M et al. Acquired cerebral amyloid angiopathy: an emerging concept. Prog Mol Biol Transl Sci 168:85-95, 2019

35) Yamada M, et al.: Mechanism of onset of early-onset, non-hereditary cerebral amyloid angiopathy (CAA). Research and surveillance of prion diseases and prevention of infections supported by Health and Labour Administration Promotion Research Grants (Intractable Disease Policy Research Project). FY 2022 Summary and Collaborative Research Report P91-94

36) Yamada M, et al.: Dutch-type cerebral amyloid angiopathy (dCAA): relationship between the site of cadaveric dura mater graft and time of onset and clinical presentation of dCAA. Research on surveillance of prion diseases and prevention of infections supported by Health and Labour Administration Promotion Research Grants (Intractable Disease Policy Research Project). FY 2023 Summary and Collaborative Research Report P87-90