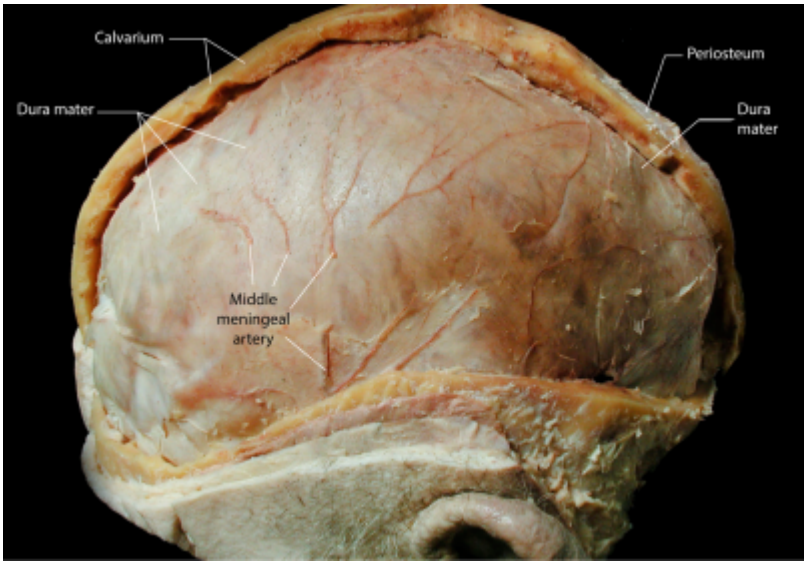
Cadaveric dura



Cadaveric dura mater graft-associated CJD (dCJD) accounts for a common form of iatrogenic CJD.

There is strong evidence that cadaveric dura mater represents a completely unnecessary risk of CJD to the public. The disease in question is fatal, the risks cannot be reliably eliminated and there are clear alternatives. Yet the FDA has not opted to ban the material, even though two other countries, Japan and Britain, have done so without evident problems, and the WHO recommends this course of action. Ironically, the U.S. still permits the import of dura mater sourced from the very countries (Japan and Britain) where the material is no longer used. We hope that the recent attention to BSE will induce the FDA to finally take action to adequately protect the public [1)]

Case series

Banerjee et al., describe in detail the clinical and neuroimaging findings in three patients with early-onset symptomatic amyloid-β cerebral amyloid angiopathy following childhood exposure to cadaveric dura (by neurosurgical grafting in two patients, and tumor embolization in a third). The observations provide further in vivo evidence that cerebral amyloid angiopathy might be caused by the transmission of amyloid-β seeds (prions) present in cadaveric dura, and has diagnostic relevance for younger patients presenting with suspected cerebral amyloid angiopathy [2)].

A report summarizes the epidemiologic features of 154 cases of dCJD identified in Japan during 1975-2017; these cases account for >60% of dCJD cases reported worldwide. The unusually high prevalence of dCJD in Japan was first reported in 1997. In 2008, a single brand of graft (Lyodura [B. Braun Melsungen AG, Melsungen, Germany]), frequently used as a patch in neurosurgical procedures, was identified as the probable vehicle of transmission. No international recall of the implicated Lyodura occurred, the product had a relatively long shelf life, and the grafts were used frequently in Japanese patients with non-life-threatening conditions.

Since 2008, additional cases have been ascertained, reflecting the identification of previously missed cases and the occurrence of new cases with longer latency periods (interval from exposure to symptom onset) for dCJD (up to 30 years), underscoring the importance of maintaining surveillance for dCJD [3].

A nationwide survey and information documented 57 patients with Creutzfeldt-Jakob disease (CJD) who had received dura mater grafts during the period between January 1979 and September 1999. At least 54 of these 57 patients received the same brand of dura mater graft from the same processor. Mean age at disease onset in the 57 patients with dural grafts was younger (51.9 years) than that in patients with sporadic CJD (63 years) ($p < 0.0001$). Initial symptoms were cerebellar ataxia, disorientation, and visual or oculomotor disturbance [4].

Case reports

A 31-year-old woman 56 months after she received a cadaveric dura mater graft after the removal of a giant pituitary neuroendocrine tumor. Creutzfeldt-Jakob disease was confirmed by a brain autopsy and the existence of an abnormal isoform of prion protein, verified by both immunohistochemical and Western blot analysis. Moreover, prion protein gene analysis was shown in this case to possess a wild-type genotype [5].

References

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