

# Molecular imaging of dural microcalcification: Detection of active mineralization with $^{18}\text{F}$ -NaF PET/CT

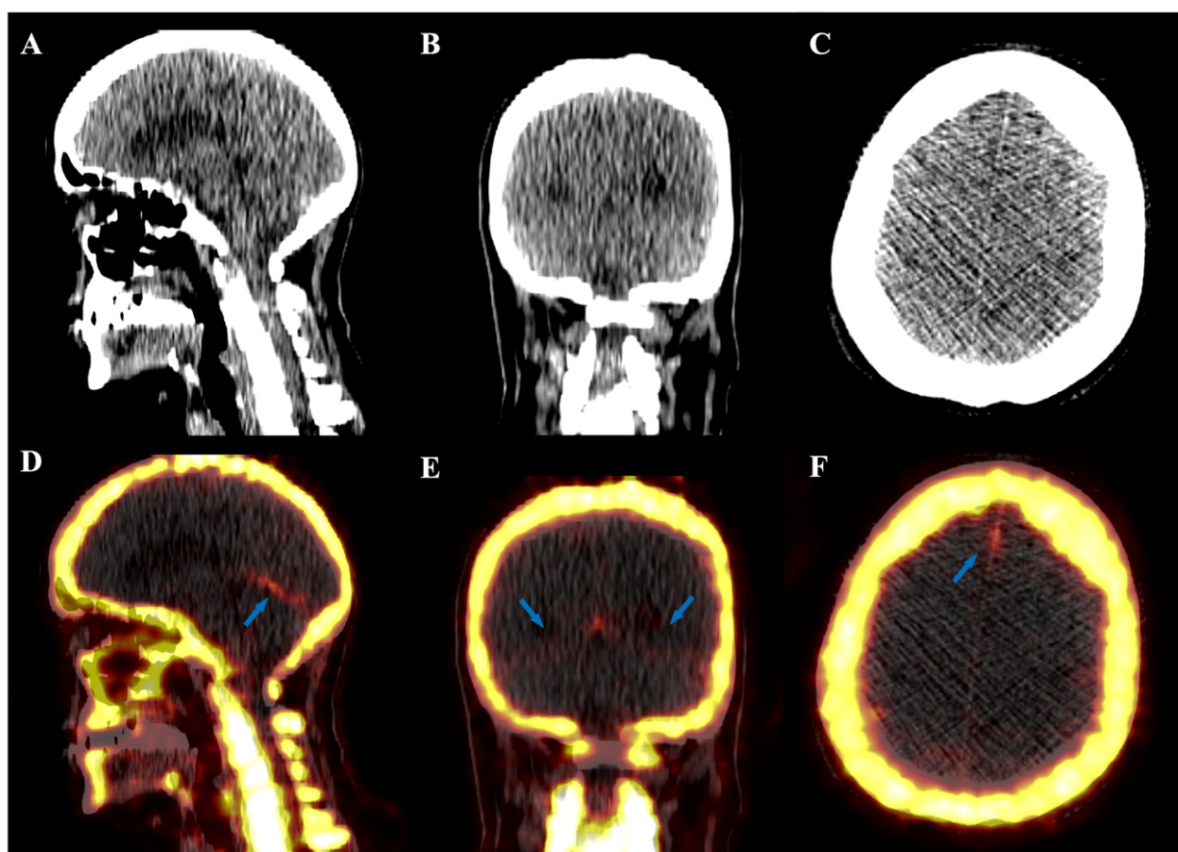
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## Abstract

Dural calcification is frequently observed on computed tomography (CT) as an age-associated finding, yet its biological activity and potential for pathological progression remain poorly characterized. Fluorine-18-sodium fluoride ( $^{18}\text{F}$ -NaF) is a positron emission tomography (PET) radiotracer that can detect biologically active microcalcification and may thus enable monitoring of such lesions. We describe a 73-year-old woman with hypertension, hypercholesterolemia, and atrial fibrillation who was incidentally found to have focal  $^{18}\text{F}$ -NaF uptake in the falx cerebri (mean standardized uptake value (SUVmean) 1.29, SUVmax 2.02) and tentorium cerebelli (SUVmean 1.08, SUVmax 1.76). This case highlights the novel capability of  $^{18}\text{F}$ -NaF PET to characterize dural mineralization at the molecular level and serve as a biomarker of lesion progression.



**Figure 1.** (A) Sagittal, (B) coronal, and (C) axial computed tomography (CT) images. (D) Fused fluorine-18-sodium fluoride ( $^{18}\text{F}$ -NaF) positron emission tomography (PET)/CT sagittal, (E) coronal, and (F) axial images reveal focal radiotracer uptake (in red) along the dura (blue arrows). The subject is a 73-year-old woman with a body mass index (BMI) of 31.4 and a medical history significant for hypertension, hypercholesterolemia, atrial fibrillation, and prior smoking. She was recruited to the Cardiovascular Molecular Calcification Assessed by  $^{18}\text{F}$ -NaF PET/CT (CAMONA) clinical trial as a subject identified at elevated risk for cardiovascular disease. Dural calcifications were incidentally identified CT, where hyperdense signal is localized along the falx cerebri and to a lesser extent in the tentorium cerebelli. Fluorine-18-NaF PET revealed focal uptake along the falx cerebri (SUVmean 1.29, SUVmax 2.02) and tentorium cerebelli (SUVmean 1.08, SUVmax 1.76). Although typically regarded as benign age-related findings, dural calcifications may arise from diverse pathological processes: chronic inflammation or tumors, trauma and hematoma formation, neurosurgery, metabolic disturbances, or congenital pathology [1, 2]. Fluorine-18-NaF PET enables *in vivo* visualization of active microcalcification by binding to exposed hydroxyapatite on mineralized tissue [3, 4]. While conventionally used to evaluate tumors of bone,  $^{18}\text{F}$ -NaF PET has become a modality of interest for the imaging of heterotopic calcification, including atherosclerotic plaques [5-9]. Uptake of  $^{18}\text{F}$ -NaF reflects ongoing calcification at the molecular level rather than quiescent mineralized tissue [4], thus providing a unique view into the progressive nature of dural calcification that structural imaging alone cannot capture. While the significance of active dural mineralization remains to be determined, this finding demonstrates a clinically relevant application of  $^{18}\text{F}$ -NaF as a measure of dural microcalcification. Future studies are warranted to determine contributing factors to this process and its association with physiological or pathological states.

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