



# Adhesive arachnoiditis in a Caucasian female cadaver

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Chris N. TREINEN

Lee J. OPDAHL \*

Brent A. KRAMER

Brody D. SLOSTAD

Department of Biology & Microbiology, Box 2104, South  
Dakota State University, Brookings, SD, USA.



\* Lee J. Opdahl  
Department of Biology &  
Microbiology  
South Dakota State University  
Brookings, SD, 57007, USA.  
☎ +1 (507) 829-4368  
✉ [lee.opdahl@jacks.sdstate.edu](mailto:lee.opdahl@jacks.sdstate.edu)

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## Abstract

Arachnoid calcifications are associated with the pathologic process of chronic inflammation and are most commonly observed on the dorsal aspect of this meninx in the thoracic region. During prosection, a laminectomy was performed on an 86-year-old Caucasian female cadaver. Two clusters of white lesions were exposed on the dorsal and lateral aspects of the arachnoid between T6/T8 and L1/L2. These lesions were comprised of a collagen matrix organized into lamellar bands. Fibrocytes were rare within the matrix and margins of the plaque were supported by loose areolar connective tissue. The absence of osteoblasts or osteoid infers that these lesions are the result of chronic passive mineralization, rather than ossification. The arachnoid is thought to respond to irritation by initiating an inflammatory reaction, chronic inflammation can then lead to arachnoid calcification. Spinal stenosis, herniated discs, and osteophyte formation were readily apparent between T11-L4. All of these findings are consistent with a diagnosis of chronic adhesive arachnoiditis.

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**Key words** [arachnoiditis] [lesion] [cadaver]

## Introduction

Arachnoid calcifications are thought to be intimately associated with the pathologic process of chronic inflammation. One of the hallmarks of chronic inflammation includes the presence of macrophages and fibroblasts in the affected tissue. Not surprisingly, recent studies have shown fibroblasts to be critical to calcium deposition in arteries [1].

Calcifications of the arachnoid mater are most commonly observed on the dorsal aspect of this meninx in the thoracic region [2]. Previously, description of similar lesions was confined to incidental anatomic findings. However, with the development of computed tomography (CT) and magnetic resonance imaging (MRI), clinical diagnosis has increased [3].

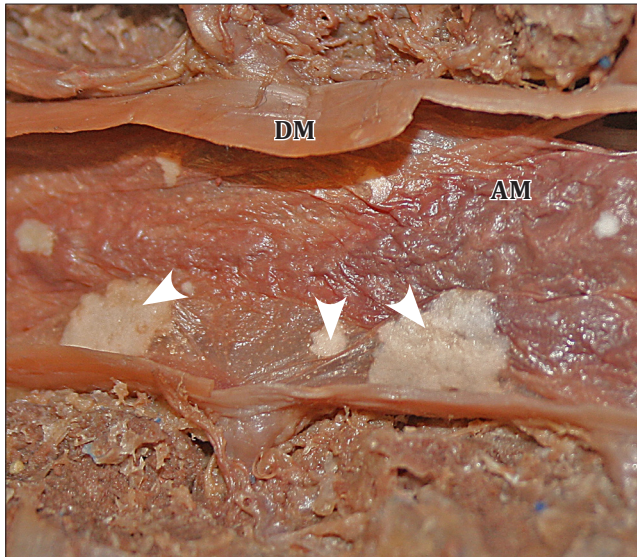
Normally asymptomatic, the calcified lesions may rapidly increase in size causing compression of the spinal cord. Excessive lesion-induced parenchymal pressure initially reduces, and ultimately inhibits, normal neurological conduction [2, 4, 5]. This review presents a case of CAA with discussion of pathogenesis, clinical presentation, related complications, and symptom management.

## Case Report

During the course of a standard anatomic prosection, a laminectomy spanning C1 to L5 was performed on an 86-year-

old Caucasian female cadaver. Upon the removal of the dura mater, clusters of white lesions ranging in size from 1 mm to 9 mm were exposed between T6/T8 and L1/L2 (Figure 1). Most lesions were located on the dorsal and lateral aspects of the arachnoid mater, while a single lesion was discovered on the ventral aspect. Lesions were removed and fixed in neutral buffered formalin, embedded in paraffin, sectioned, and subjected to an array of histological stains (Figure 2). Following Dahl's method [6], the samples were tested for calcium using 1% alizarin red. Kossa's method was followed to stain for calcium salts [7]. Masson's trichrome method [8] was used to stain collagen. Bennhold's method for amyloid was followed to stain for amyloid using 1% Congo red solution [7]. A routine Harris' hematoxylin and eosin was performed to stain nucleic acids and proteins. Periodic acid-Schiff [9] was used to stain for polysaccharides.

On routine hematoxylin and eosin staining, the arachnoid plaques were comprised of a collagen matrix which was highly organized into lamellar bands or, less commonly, tight whorls (Figure 2A). Fibrocytes were rare within the matrix itself, but margins of the plaque were supported by loose areolar connective tissue with regularly spaced fibrocytes and sporadic small vessels. The collagenous composition of the plaque was confirmed by Masson's trichrome staining (Figure 2B). The matrix was diffusely mineralized by calcium



**Figure 1.** Photograph of lesions in situ at T6-T7. (**DM:** dura mater; **AM:** arachnoid mater; **arrowheads:** calcifications)

and calcium salts, as shown by alizarin red and Von Kossa staining (Figure 2C). Periodic acid Schiff and Congo red staining failed to demonstrate polysaccharides or amyloid within the matrix (Figure 2D).

### Discussion

Chronic adhesive arachnoiditis is typically caused by inflammation, adhesion, or distortion of the nerve roots within the spinal cord. A differential diagnosis for such cases should include Behcet syndrome, isolated central nervous system angiitis, systemic lupus erythematosus, Sjogren syndrome, and Wegener granulomatosis [10]. However, we speculate that the immediate cause of CAA here was chronic spinal stenosis and disc protrusion.

Several pathologic processes induce calcification and are broadly grouped into one of two forms. Dystrophic calcification occurs in tissue undergoing the process of coagulative, caseous, or liquefactive necrosis. This occurs regardless of normal serum calcium levels and without the presence of calcium metabolism disorders. Metastatic calcification occurs in tissues due to hypercalcemia caused by a variety of conditions such as parathyroid tumor or paraneoplastic hypercalcemia due to ectopic secretion of PTH-related protein from malignant neoplasms [10].

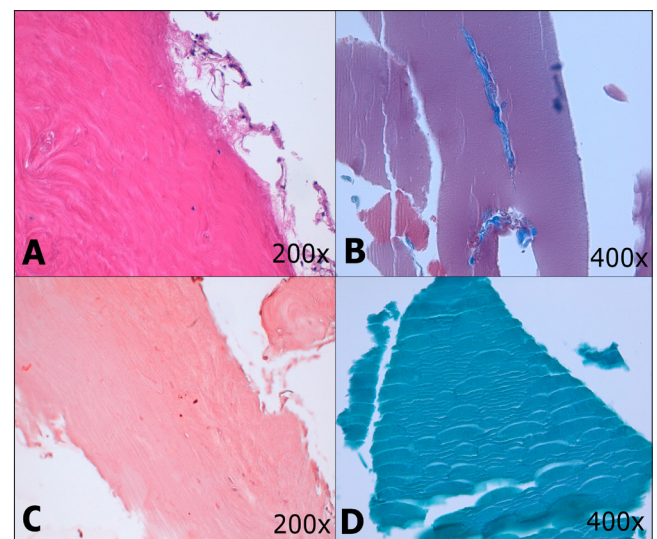
Generally, calcifications of the arachnoid mater are not thought to be due to either dystrophic or metastatic calcification. Instead, the arachnoid mater is thought to respond to irritation by initiating an inflammatory reaction, which may progress to chronic inflammation if untreated [11]. Chronic inflammation can lead to arachnoid calcification, first through the recruitment of macrophages which is then followed by fibroblasts. Macrophages release platelet-derived growth factor (PDGF), fibroblast growth factor (FGF) and

transforming growth factor beta (TGF- $\beta$ ), all of which recruit fibroblasts to the site of inflammation. The fibroblasts then produce collagen which contributes to scar tissue, which is normally broken down in the remodeling phase but persists in CAA [10].

One proposed pathogenesis of dystrophic calcification involves the formation of crystalline calcium phosphate similar to bone hydroxyapatite. Damage to the cell membrane causes membrane bound vesicles to concentrate calcium in a process that involves the binding of calcium ions to vesicular phospholipids and the subsequent creation of calcium binding phosphate groups by phosphatases. The cycle then repeats and cellular concentrations of calcium increase which create calcium deposits near the cellular membrane. Calcium and phosphate groups then rearrange to form a microcrystalline structure, which leads to additional calcium deposition [10]. We believe our samples provide evidence for passive mineralization involving calcium deposition during long-term chronicity rather than ossification, due to the absence of lamellar rings, osteoblasts, or osteoid in the lesion.

In addition to CAA, prosection revealed protruding disks and severe scoliosis associated with L2/L3 and L3/L4. Large osteophytes were present on T11 and to a lesser extent on adjacent vertebrae. Scoliosis is associated with spinal stenosis, herniated discs, and osteophyte formation – all of which are consistent with the development of CAA. In this case, the other acquired malformations of the spinal column may have masked any potential symptomology of the observed CAA.

Spinal cord calcifications are observed in roughly 76% of autopsies and are generally thought to be asymptomatic, however, these calcifications can occasionally lead to nerve root clumping which necessitates pain management. There is



**Figure 2.** Histological staining of lesion with hematoxylin and eosin (A), Masson's trichrome (B), alizarin red (C), and periodic acid-Schiff (D). This particular portion of the lesion was removed from the ventral surface of this meninx, adjacent to T8.

some disagreement in the literature concerning the treatment of CAA [3]. It has been suggested that removing intrathecal ossifications produces little or no clinical improvement of symptoms [3]. Decompression of the spinal canal may offer better symptom relief after performing decompression laminectomies [12].

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