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## Amyloid Deposition in Human Pulp

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### Summary

A tooth with a pulp having a rare degenerative change was found among numerous extracted tooth specimens. Microscopically eosinophilic diminutive granules existed around vessels in the coronal pulp. These were stained positively with Congo red and periodic acid-Schiff. Electron microscopic observation revealed that granules were composed of bundles showing one of the characteristic appearances of amyloid deposition.

### Introduction

Amyloid deposition occurs in the following varied conditions ; advanced aging, chronic infection and inflammation, disturbances of immune and autoimmune reaction, excessive tissue breakdown and wasting, and certain neoplastic lesions. Various organs and tissues, such as kidney, liver, spleen, frequently are involved in systemic or localized features (Dante and Chiga, 1985)<sup>2)</sup>. In the maxillo-facial regions the tongue and gingiva are the most common foci (Shafer et al., 1974)<sup>3)</sup>. It has been known that amyloid degeneration in the pulp could be unusually found cases of systemic amyloidosis (Ishikawa and Akiyoshi, 1980)<sup>4)</sup>. In 1922, Gräff referred to amyloid deposition in the pulp of the patient involved systemic amyloidosis, which was caused by tuberculosis<sup>5)</sup>. However, there are no recent reports about such pulpal change.

Through observations of many extracted tooth specimens, we were fortunate enough to find a case of deposition recognized as amyloid substance. The present study gives the histochemical and ultrastructural findings of amyloid deposition in human pulp.

### Materials and Methods

Removed human third molars erupted or impacted were fixed in 10% formalin and demineralized in 10% formic acid with formalin. After embedding in celloidin, specimens were sectioned and stained with hematoxylin-eosin (H-E) to observe histopathological changes including progressive, degenerative and inflammatory lesions. A case expected to display amyloid deposition was stained with periodic acid-Schiff reagent (PAS), van Gieson's and Congo red. Some celloidin sectioning specimens were treated following conventional procedures for electron microscopic examination. After being removed, celloidin specimens were reembedded in Epoxy resin and sectioned by a ultramicrotome. Ultrathin sections were stained with uranyl acetate and lead citrate, and examined using a JEOL JEM 100-B electron microscope.

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### Results

#### Light microscopic findings :

The diminutive acidophilic granules were distributed diffusely around vessels in the coronal pulp and cuspal radicular pulp, chiefly in the cervical portion. No findings of inflammatory reactions such as hyperemia and infiltration of round cells were seen (Figs. 1, 2 and 4). In the sections stained with van Gieson's, it was revealed that granules were yellow and red collagen fibers were somewhat abundant in the granules area (Fig. 5). Fig. 6 and 7 show that the granules reacted positively to PAS and Congo red. Simple and reticular atrophies were found in the radicular pulp (Fig. 3).

#### Electron microscopic findings :

Low power electron micrographs indicated that the granules had a moderate electron density and collagen fibrils were slightly increased in this region. The electron dense granules had chestnut-burr aspects, and measured approximately  $1\mu\text{m}$  in diameter (Fig. 8). They were composed of many bundles growing in all directions. A few microfibrils were also seen around the granules (Fig. 9). Although collagen fibrils were seen in the electron dense area, there was no intimate relationship between collagen fibrils and the deposits.

### Discussion

It was already known that amyloid is eosinophilic amorphous depositions stained positively with PAS and Congo red (Missmahl and Hartwiz, 1953).<sup>5)</sup> The results of the present study treated with special staining methods demonstrated that the deposits in the pulp was amyloid substance. Ultrastructurally, Cohen and Calkin (1959)<sup>1)</sup> first described amyloid as being made up of microfibrils and interfibrillar substance. The ultrastructure of the dense-bodies seems to be amyloid substances. Because sections with removed celloidin were used, we could not observe fine structure of amyloid fibrils and the relationship between amyloid deposition and cellular components of pulp tissue.

Etiology of the present case could not be suggested, so that no clinical data such as sex, age and local and systemic conditions was recorded on the extracted tooth. It is not necessary to consider chronic pulpitis as cause for deposition, but it is uncertain whether degenerative changes in the radicular pulp are concerned with the appearance of amyloid. Schulz (1977)<sup>7)</sup>, Nakagawa (1979)<sup>6)</sup> et al. suggested that endothelial cells and pericytes produced amyloid substance. The fact that granules were scattered around vessels may suggest that the origin of amyloid has a close relationship with blood vessels.

### References

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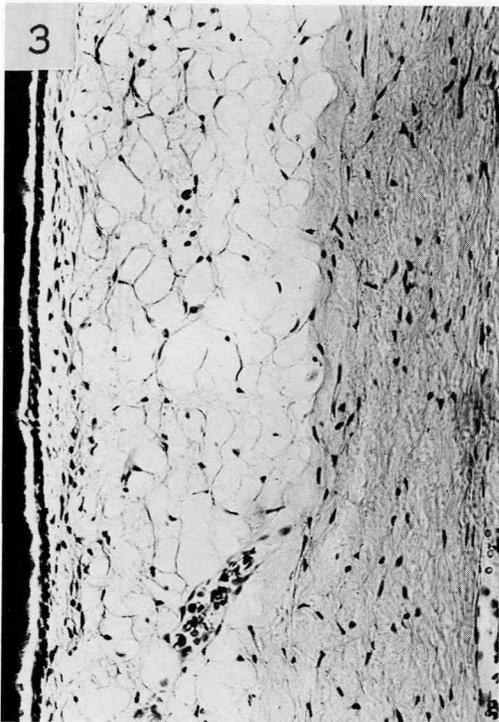
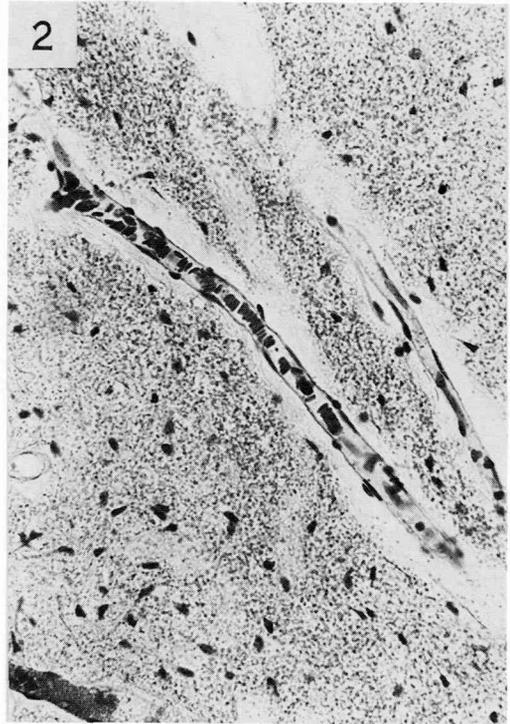
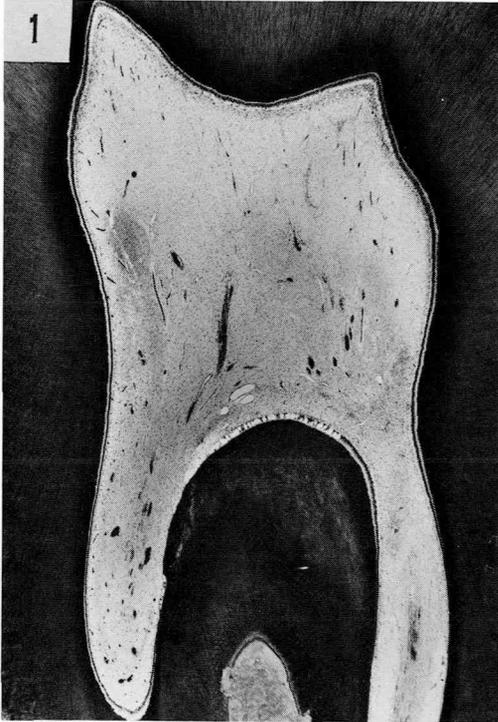


Fig.1 Deposition in the coronal pulp. H-E,  $\times 10$ .

Fig.2 Granules distributed around the vessels. H-E,  $\times 250$ .

Fig.3 Simple (left) and reticular (right) atrophies in the radicular pulp. H-E,  $\times 165$ .

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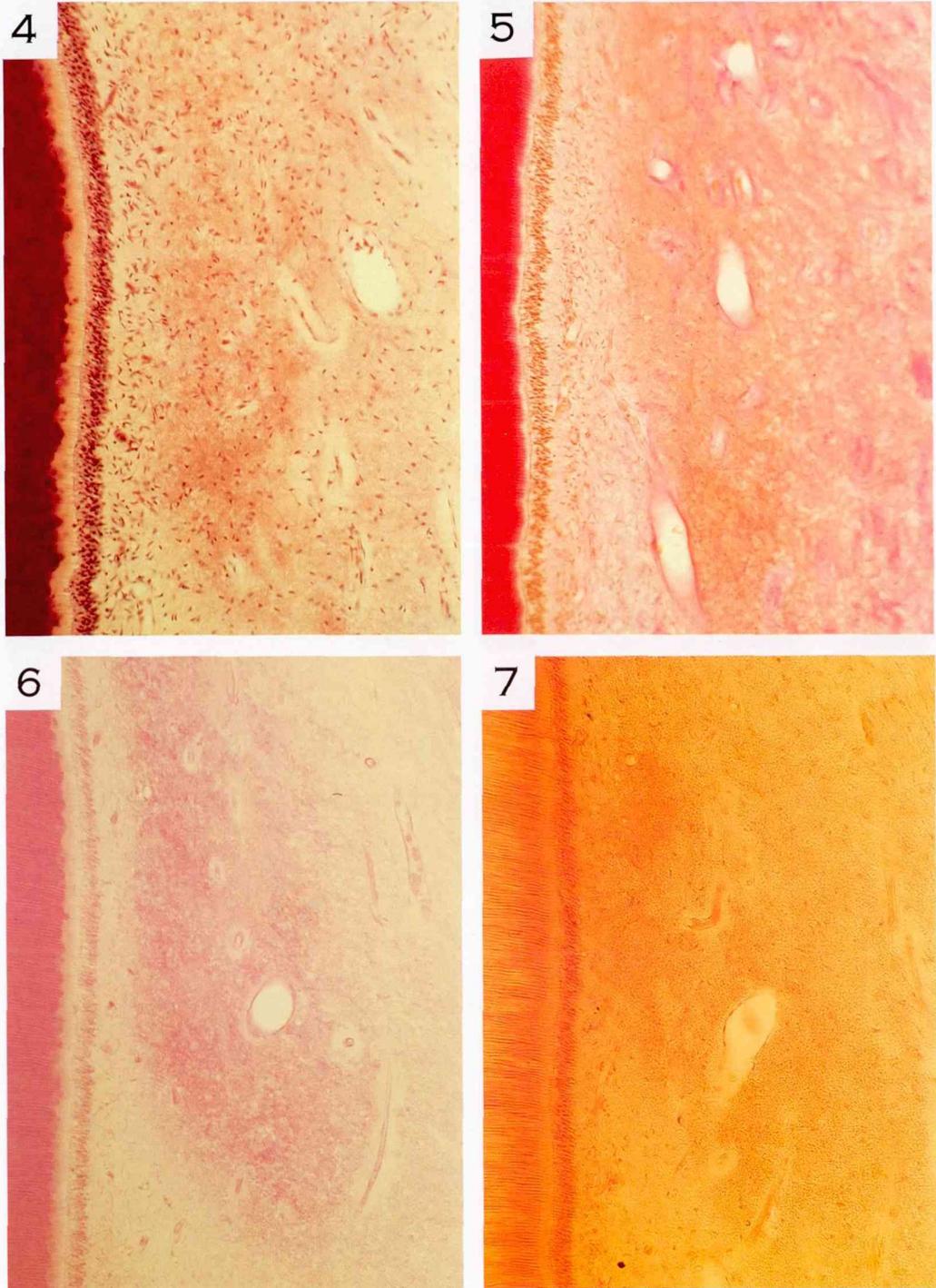


Fig.4 Eosinophilic granules in the coronal pulp. H-E,  $\times 150$ .

Fig.5 Yellow granules and red collagen fibers stained with van Gieson's.  $\times 150$ .

Fig.6 PAS-positive granules around the blood vessels.  $\times 150$ .

Fig.7 Depositions reacted positively to Congo red.  $\times 150$ .

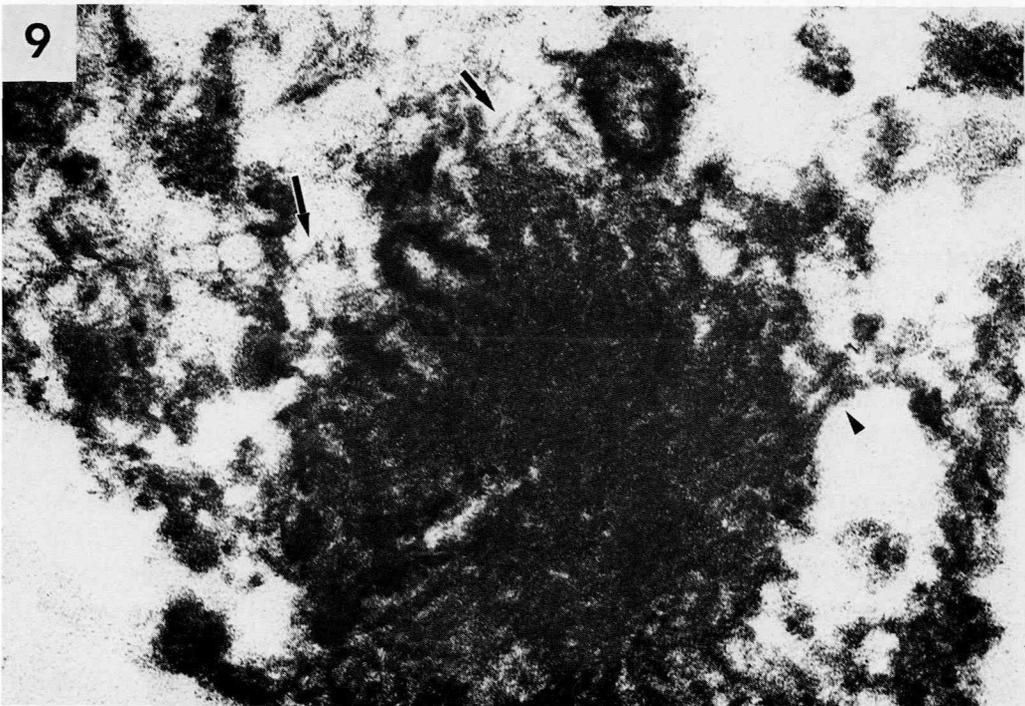
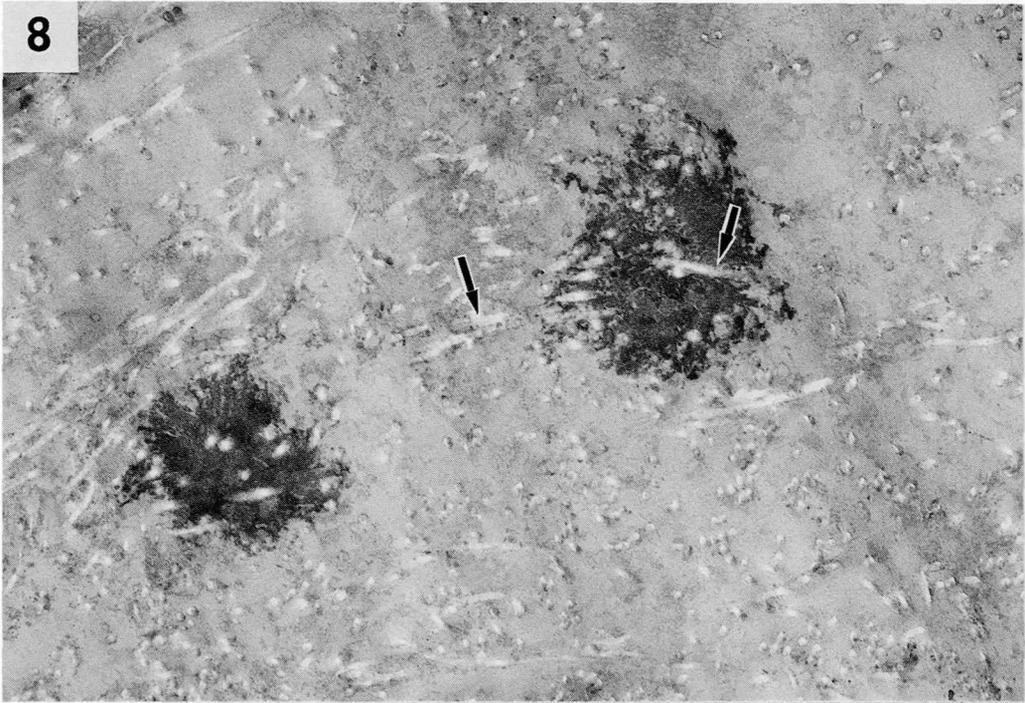


Fig.8 Electron dense bodies showing chestnut-burr aspects and slight increasing of collagen fibrils (arrows).  $\times 19000$ .

Fig.9 Bundles growing in all directions (wedge) and microfibrils (arrows).  $\times 96000$ .