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"MUCOCYTES" IN THE BRAIN OF A LLAMA: A CASE REPORT

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Abstract: "Mucocytes" were found in the white tracts of the brain of a llama (*Lama glama*) which had clinical signs of central nervous derangement. A morphologic and histochemical comparison with other cerebral organic and inorganic deposits is given.

INTRODUCTION

Cerebral mucoid globules ("mucocytes", Buscaino plaques, metachromatic bodies) in man and other animals have been noted and described by numerous other authors.^{1,2-8,10} According to Smith,¹⁷ these structures were found and named "mucocytes" by Grynfeldt in 1923 in brains of persons with senile dementia, paralysis agitans, and Wilson's disease. Smith¹⁷ studied mucocytes, which she called metachromatic bodies, in the brain of a man with Huntington's chorea and in the brains of two horses with grass sickness. Innes and Hadlow¹⁰ also referred to the occurrence of mucocytes in brains of horses with grass sickness. These bodies are considered to be identical with the plaques of Buscaino^{3,4} who, as early as 1922, expressed the same difficulty prevailing today in identifying the precise etiology, pathogenesis, and chemical composition of these structures by naming their principal constituent "Substance X". Mucocytes appear in the white tracts of the brain as unilocular, sometimes lobulated, varisized, round or oval bodies that give the characteristic histochemical reactions of mucin.

The purpose of this report is to document the occurrence of these structures in the brain of a llama (*Lama glama*) and to compare them with other organic and inorganic deposits which may mimic mucoid globules.

CASE REPORT

A 102 kg male llama (*L. glama*), 1½ years old, was presented for necropsy at the Denver Zoo after the fatal termina-

tion of an acute disease in which signs of central nervous system derangement were noted.

On the first day of observed illness the animal walked into a wall, appeared blind, and occasionally stumbled and fell. Urinalysis indicated marked glycosuria, but inadvertent breakage of a blood sampling vial prevented determination of blood glucose. On the following day the animal was recumbent and hypothermic. Supportive therapy failed, and the animal died. Necropsy was performed 6 hours later.

RESULTS

Gross Findings

Despite a clinical impression that death may have resulted from enterotoxemia, intestinal contents were neither assayed for toxin nor submitted for microbiologic culture, since the time lapse between necropsy and death nullified the validity of such procedures.

There was a 2 cm ulcer on the dental aspect of the upper lip. Several abscesses were noted in the liver. A piece of metal, well encapsulated, lay under the serosa of the reticulum. The intestines contained greenish, fetid, fluid feces with some gas bubbles, but there were no visible hemorrhages in the gut walls. No other significant gross lesions were recognized in the musculoskeletal, respiratory, cardiovascular, genitourinary, endocrine, digestive, hematopoietic, nervous, or integumentary systems.

Histopathologic Findings

Representative samples of all organ systems were fixed in 10% neutral-buffered formalin, embedded in paraffin, cut at 6 μ , and stained with hematoxylin and eosin (H & E).

The labial ulcer contained plant material, and coccoid and bacillary bacterial colonies. Numerous neutrophils infiltrated the surrounding dermis and submucosal tissues. Oval erythrocytes, characteristic of the camel, were noted in focal hemorrhages. However, the lesion was well localized to a small area and was not considered contributory to the demise of the animal.

The liver had irregularly distributed abscesses in both capsular and parenchymal locations. Broad bands of dense collagenous tissue branched inward from the thickened capsule and divided the hepatic lobules into units of disparate size. There was engorgement of all vascular channels, and a thin fibrous wall was present around many central veins. In some lobules central veins were difficult to discern. Abundant fibrous connective tissue was present in most portal areas, in which hyperplasia of biliary channels was noted.

Several instances of infraglomerular epithelial reflux¹² were observed in the kidney. In many glomeruli there were proteinaceous globules in Bowman's space. No evidence of inflammation was noted in the kidney, but mild tubular nephrosis was present.

The area surrounding the metallic foreign body under the serosa of the reticulum was comprised principally of mature fibrous tissue and did not present any other salient feature.

Distributed in the white matter generally, and especially in the cerebrum, cerebellum, corpus callosum, and medulla, were numerous mucoid aggregates of variable size but averaging around 50 microns in diameter (Fig. 1). These structures provoked no inflammatory changes and were mainly extracellular, lying in the tissue and surrounded by fibers of the neuropil. The bodies appeared amorphous at scanning and low magnification. With H & E they were homo-

geneously basophilic, but under high magnification they appeared to be composed of tiny globules. Under polarized light the masses were birefringent. Rarely they were intracellular and contained a pyknotic nucleus, but most were large and were not membrane-bound, delineated only by compressed fibers of the neuropil. Occasionally fibrillar strands would traverse the space occupied by the mucoid globules to give the masses a lobulated appearance (Fig. 2). The masses reacted positively to periodic acid-Schiff and alcian blue stains (pH 2.5), and proved strongly argentophilic (Fig. 3) with Gomori's methenamine silver. With Meyer's mucicarmine they stained light purple. They did not react to acid-fast, calcium, iron, or nucleic acid¹³ staining procedures. Often numerous empty cystic spaces were observed, which gave the impression that some had become dislodged and had dropped out during processing of the tissue. A clear, wide perineuronal zone was present around Purkinje cells in the cerebellum. Rounding and hyperchromatism of neurons in the cerebrum and cerebellum were frequently seen. Aside from

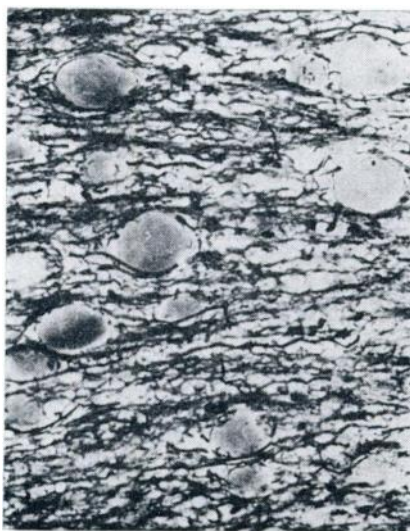


FIGURE 1. Multiple basophilic structures in white fiber tracts of the midbrain, H&E, 150X.

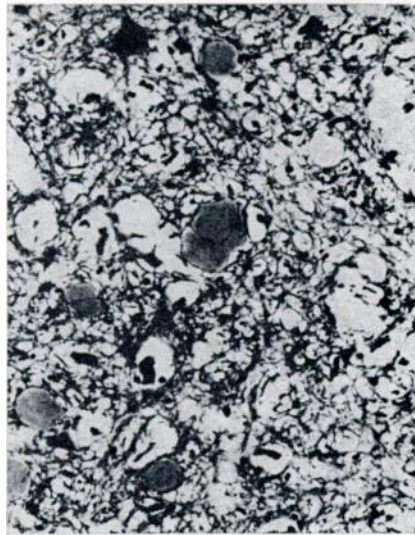


FIGURE 2. Mucocytes in white matter of the midbrain with fibers traversing one body to give it lobulated appearance. H&E. 150X.



FIGURE 3. Gomori's methenamine silver stain to depict argentophilic character of mucocytes. 150X.

extensive sieve-like, spongy areas in the white matter, no other salient encephalopathic lesions were observed.

DISCUSSION

These masses are compatible with the mucocytes of Grynfeldt,¹⁷ the plaques of Buscaino,^{4,6} and the metachromatic bodies of Smith.¹⁷

Opinions regarding the origin and pathologic significance of mucocytes are discordant. Some authors regard the masses as artifacts.^{2,16} Ferraro,⁷ as early as 1928, pointed out that the structures could best be seen by using alcohol to fix the tissue. More recent work¹ suggests that alcohol fixatives enhance the formation of mucocytes. Grinker and Stevens⁸ expressed the concept, and cited the work of others who concurred, that mucocytes were derived from degenerating oligodendroglia. These same authors⁸ were emphatic in their opinion that mucocytes were not found in normal brains, that the structures were not artifacts, and that "sensitizing" alcohol fixatives were unnecessary to bring out their mucinous constituents. They believed that oligodendroglia cells underwent a mucoid degeneration, swelled, and ruptured, liberating into the surrounding tissues globular aggregates that coalesced to form larger masses. They presented "final proof" that degenerating oligodendroglia alone gave rise to mucocytes by demonstrating mucoid material in oligodendrogliomas.

Adornato and Lampert¹ considered Buscaino bodies to be incidental findings in normal and abnormal brains and believed that they arose from the artifactual disintegration of myelin. Cancilla and Barlow⁹ described procedural electron microscopic techniques that resulted in artifactual formation of Buscaino bodies. Blackwood *et al.*⁸ indicated that the chemical nature of mucocytes may be a glycolipid resembling cerebroside, but they did not identify precisely the condition under which the Buscaino bodies are formed.

We are not qualified to explain the precise cellular phenomena that lead to the formation of mucocytes, but their identification with Huntington's chorea,

TABLE 1. Characteristics of some organic and inorganic deposits in the brains of animals.

DEPOSIT	PHYSICAL PROPERTIES			BIREFRINGENCE	STAINING CHARACTERISTICS					SUGGESTED SIGNIFICANCE	REF
	SIZE	SHAPE	LOCATION		H & E	PAS	ALCIAN BLUE	CALCIUM	SILVER		
Corpora amyacea	15-50 μ	spherical	extracellularly in: (a) subpia (b) subependyma	neg.	basophilic	pos.	pos.	neg.	neg.	an aging change	14
Corpora arenacea	variable	lobulated	extracellularly in: (a) olfactory lobe (b) cerebellum (c) midbrain (d) pineal	neg.	basophilic	pos.	pos.	pos.	neg.	manifestation of involution	7
Mucocytes	10-150 μ	spherical, sometimes lobulated	(1) within oligodendroglia throughout white matter (2) or extracellularly	pos.	basophilic	pos.	pos.	neg.	neg.	(1) autolytic change (2) grass sickness in horses (3) Regenerative change	13 3
Argentophil plaques	5-100 μ	irregular	extracellularly in: (a) outer layer of cerebral cortex (b) Ammon's Horn (c) Purkinje cell layer	neg. unless center of plaque contains amyloid	occasionally the center of this plaque contains amyloid and thus stains accordingly			neg.	pos.	a senile change	3
Dystrophic axons	5-50 μ	ovoid	medulla oblongata and other areas in white matter	neg.	eosinophilic	pos.	neg.	neg.	pos.	(1) a degenerating axis cylinder (2) Vit E def (3) a senile chg	5 8 9
Extracellular global vacuoles	variable	round	cross section of white fiber tracts	neg.	In routinely processed sections these appear as empty punched out holes that occasionally contain a mucopolysaccharide complex					(1) found in normal brains (2) numerous in demyelinating process	7

senile dementia, and Wilson's disease of man¹⁷ and with grass sickness of horses¹⁷ indicates that mucocytes are not always artifacts. It seems that the sequence of events that ultimately lead to the formation of mucocytes may be triggered by a variety of factors leading to edema, myelinolysis, and injury to oligodendroglial cells.

Because the material in the globular masses appears mainly extracellularly, we do not regard "mucocyte" as a proper term to describe the aggregates. "Mucoid degeneration" does not adequately describe their unilocular occurrence. Whereas the term "mucoid globules" would circumvent the problem of attempting to

coin a term that describes both their intra- as well as extracellular location, we feel that their older name, mucocytes, is too firmly entrenched in the literature to permit a change.

Special stains can be used to good advantage to differentiate mucocytes from other organic or inorganic structures occurring in brains of animals with or without clinical evidence of central nervous involvement. Table 1 presents a comparison of morphologic and histochemical properties of some common deposits with those characteristic of mucocytes. Note that, of those listed, mucocytes are the only ones that are birefringent.

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