INTRACEREBRAL HEMORRHAGE IN A BOTTLENOSED DOLPHIN (Tursiops truncatus) 1

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INTRACEREBRAL HEMORRHAGE IN A BOTTLENOSED DOLPHIN (*Tursiops truncatus*)[1]

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Abstract: A neurologic condition closely resembling a common human disease, hypertensive intracerebral hemorrhage, is described in a captive dolphin. Motor deficits and the possibiltiy that behavioral changes resulted in the animal's being attacked and driven off by its herdmates, are discussed in terms of damage resulting from the hemorrhagic lesion.

INTRODUCTION

Although common in humans, massive spontaneous intracerebral hemorrhages must be regarded as distinctly rare in non-human terrestrial mammals.[4,5,9] From our own experiences and the limited published data, such hemorrhages also are rare in cetaceans.[1,2,3,8] A study of marine mammal disease, conducted over a 3 year period on the California coast, included necropsy of 13 cetaceans representing 4 species. Although 9 of these animals suffered from parasitic brain infection, no example of intracerebral hemorrhage was reported.[8]

Our own survey, conducted on the Florida coast over the past two years, has included neuropathologic examination of 75 animals from 7 cetacean species. This paper reports the clinical history and neuropathology of the only case with intracerebral hemorrhage found since the survey was initiated.

CASE HISTORY

Mabel was a large (2.44 m) female Bottlenosed Dolphin (*Tursiops truncatus*) held in captivity at a commercial oceanarium in apparent good health for six years. A tooth was not available for age determination based upon dentate rings; however, based upon her large size, she was undoubtedly an old animal. Although not highly trained, she did participate in the daily group performances. During most of her six years in captivity she had been a stable member of a herd that consisted of two adult males, a juvenile male, and an adult female.

During the last two years her primary observed social interaction occurred after the larger female gave birth to a male infant and Mabel assisted in the rearing of this infant. Her role was protective, keeping the male dolphins away from the infant while the mother was feeding or performing in shows.

No change in this pattern of interaction with the herd was observed until five days before her death when Mabel was the victim of apparently unprovoked attack by the two larger males. Although she suffered cutaneous lacerations during the attacks, she offered no observed

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resistance and eventually had to be removed to an isolation tank. At that time her appetite was severely diminished, as she was taking only 0.7 of her usual 4.5 to 5.6 kg daily consumption of mackerel and blue runner. Detailed observations of her swimming behavior were not recorded, however, she was observed to swim aimlessly on one side and to frequently bump into the wall of the isolation enclosure. She did not improve and expired four days later.

CASE REPORT

The cerebral hemispheres and diencephalon were removed and placed in 10% formalin within 12 h. after the animal's death and were sent to the University of Florida for neuropathologic examination. Although the external surface of the brain did not show any gross abnormalities, one centimeter coronal sections revealed an extensive hemorrhage in the right hemisphere which was of the greatest extent in the section shown in Figure 1. Numerous small satellite hemorrhages were evident in the necrotic tissue adjacent to the main hemorrhagic mass which extended rostral, dorsal, and medially into the right lateral ventricle. The thalamus was displaced medial and caudally by the main body of the hemorrhage and the medial ventral portion of the right temporal lobe was pushed caudally. The right internal capsule, including that portion containing major motor pathways, was destroyed and almost certainly resulted in contralateral loss of motor control. Destruction of major interconnecting white matter tracts passing in the extreme capsule, external capsule, white matter of the temporal lobe and a portion of the centrum semiovale must have resulted in severe hemispheric cerebral dysfunction.

MICROSCOPIC FINDINGS

Histologic evaluation was accomplished using Hematoxylin and Eosin, Masson trichrome, PAS and Goodpasture stains on widespread areas of brain tissue. Sections from the area adjacent to the hemorrhage had sheets of erythrocytes intermixed with swollen and necrotic brain tissue. Hemorrhage extended into the subarachnoid space and phagocytic cells were noted. The neuropil was infiltrated with large PAS positive debris-laden macrophages. Neurons in this area were damaged or dead as indicated by eosinophilic cytoplasm and pyknotic nuclei on H&E preparations. The neuropil had severe vacuolization and the perivascular and perineuronal spaces were distended. There were a moderate number of PAS negative cells with eosinophilic cytoplasm and eccentric nuclei which probably represented early astrocytic injury or reaction to tissue injury.

Despite careful search in the hematoma, peri-hematoma tissue and other areas distant to the hemorrhage, no evidence of infection, neoplasia, vascular malformation, or parasitic infection was found.

Based on the small, but significant, number of macrophages in the necrotic tissue and the observed acute neuronal changes, the predominant damage probably occurred 5-7 days prior to death. This estimate must be regarded as somewhat speculative since data to relate the progression of histopathologic change subsequent to acute injury in cetacean brain tissue is not available.

DISCUSSION

It is unlikely that the hemorrhage was a result of traumatic attack. No skull fracture was present at necropsy and there was no evidence of subarachnoid or subdural hemorrhage.

The extreme similarity in the anatomic location of this hemorrhage to hypertensive intracerebral hemorrhage in man initially attracted our attention to this case (see Figure 1). Sixty to 80% of the hypertensive hemorrhages in man occur adjacent to the claustrum, pushing the lenticular and thalamic nuclei medially and the insular cortex and temporal lobe laterally and caudally,[11] as occurred in Mabel's case. Arterial hypertension induced experimentally in the Rhesus monkey *(Macaca mulatta)* also has been found to result in the development of a hematoma in the region of the basal ganglia and internal capsule.[6]

The similarity of the dolphin gross cortical and subcortical anatomy to that of man may be an important contributing factor to the development of a hemorrhage in this precise location. Despite marked differences in blood supply to the dolphin brain as compared to humans, similar penetrating lenticular vessels may be vulnerable to changes induced by increased blood pressure. In hypertensive humans, these vascular changes include arteriosclerosis, microaneurysms, hyalin degeneration and muscular hypertrophy.[7]

No evidence of microaneurysms, arteriosclerosis or hyalin degeneration was found in the present case, however, the possibility that one or more of these conditions might have existed focally

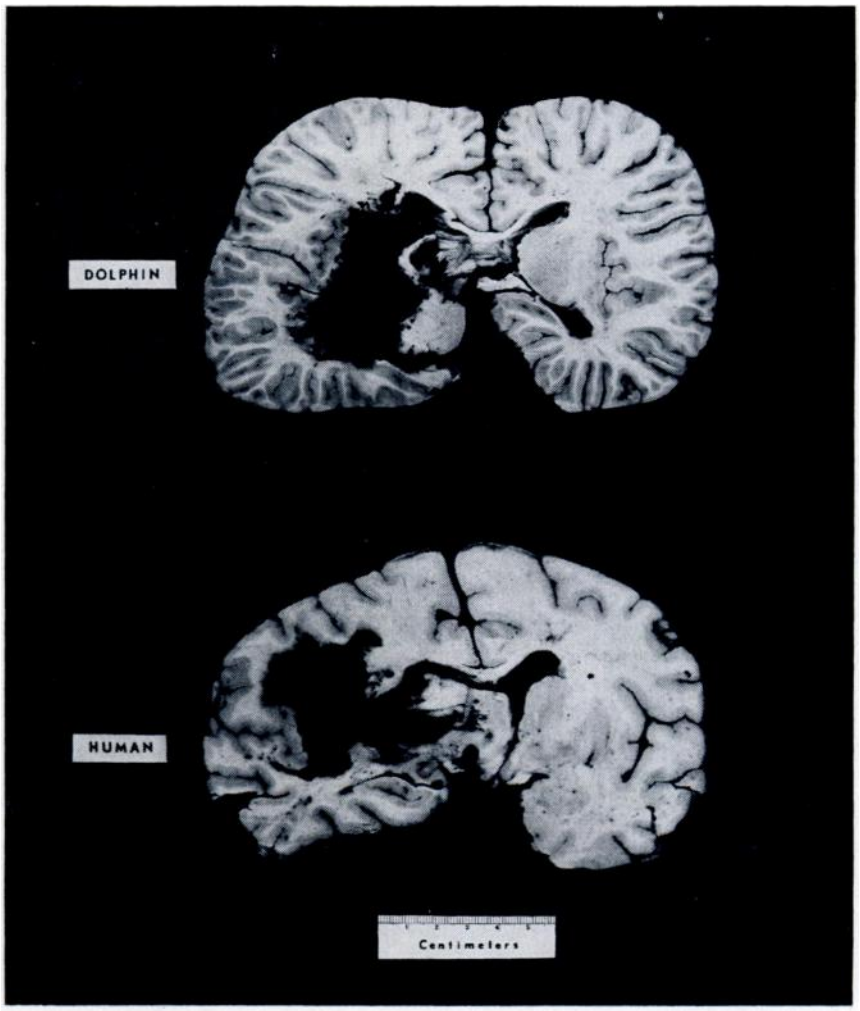

FIGURE 1. Intracerebral hemorrhage in the brain of a Bottlenosed Dolphin and its close resemblance to a case of hypertensive hemorrhage in the brain of a human.

prior to destruction by the extensive hematoma cannot be excluded. Determinations of arterial hypertrophy in humans are based on extensive experience regarding blood vessel size and the changes which occur as a result of hypertension. This data base is not present for cetaceans. We have attempted to compare Mabel's vessels to 4 *Tursiops* with no evidence of brain or cardiovascular disease. Ten arteries were randomly selected from the lenticular vessels of each animal. Following the procedure of Wagenvoort[10] the diameter of each vessel was measured and the thickness of the media expressed as a percentage of the diameter. As can be seen in Table 1, Mabel's vessels are more muscular than those from the other dolphins *(Tursiops truncatus)* we have examined.

An insufficient number of normal *Tursiops*' brains are available to make a statistical evaluation of correlations between age, sex and blood vessel size, however, it can be seen in Table 1 that comparison animals included males and females that were both larger and smaller in total length when compared with Mabel. All of these animals had smaller muscular walls encasing the arterial vessels. The extent to which Mabel's thickened vessels might be related to chronic hypertension, and therefore the genesis of this hematoma, is unknown.

Regardless of the etiology of this destructive intracerebral hemorrhage, the effect that it subsequently had on Mabel's behavior and the reaction of her long-term herd companions is of great interest. Massive hemispheric damage to intracerebral gray matter and the inter-connecting white matter tracts causes severe disorientation and behavioral dysfunction in humans, probably secondary to the concomitant failures of higher cortical function. Depending on the hemisphere involved, there may be loss or impairment of communication skills, as well as an inability to respond to previously learned social-behavioral patterns. It is therefore of interest that the earliest recognizable aberration noted by human observers was the sudden aggressive behavior of the two large male dolphins. After six years of harmonious herd interaction, this aggressive behavior raises the interesting possibility that, during the initial evolution of her intracerebral hematoma, an alteration in Mabel's behavior elicited this aggression. Subsequent to her removal to an isolation tank, she developed swimming abnormalities compatible with a hemiparesis, probably a result of hemorrhagic destruction of the contralateral internal capsule. Her disorientation and aimless swimming movements were most likely the result of a more severe generalized cerebral injury.

Despite our inability to establish the etiology of Mabel's intracerebral hematoma, the case has some marked similarities to the common human neurologic disorder, intracerebral hypertensive hemorrhage. Because of this similarity, occurrence of this neuropathologic disorder in an uncommonly reported species is of particular comparative interest.

TABLE 1. Mean percentage (±SEM) of cerebral-arterial diameter taken up by muscular media in Mabel and four normal **Tursiops.**

Species	Sex	Length (Meters)	Percentage
Tursiops truncatus (Mabel)	F	2.44	54.10 ± 4.95
T. truncatus	M	2.62	50.10 ± 3.55
T. truncatus	M	2.69	42.20 ± 4.39 $P<0.1$*
T. truncatus	F	1.80	38.90 ± 2.36 $P<0.05$
T. truncatus	M	2.44	33.80 ± 4.38 $P<0.01$

*Statistical comparisons are with Mabel.

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