Vascular Malformations and Hemangiomas of the Canine Spinal Cord

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Abstract. An arteriovenous malformation and two hemangiomas in the canine spinal cord were found in three dogs. The malformation was ventral, extramedullary and largely subdural. Chronic hypoxia, consequent upon the shunt, had led to extensive local edema, necrosis and hemorrhage at T13-L1. Prominent intramedullary perivascular fibrosis, mineralization and obliteration were the presumed consequences of the hemodynamic insult. The hemangiomas, one of which was multiple, were deep lesions that produced compressive changes in the adjacent cord tissue. The masses were regarded as benign neoplasms with central sclerosis. These conditions appear not to have been reported previously in the dog.

Non-neoplastic vascular malformations of the human central nervous system consist of capillary telangiectases, cavernous angiomas, and arteriovenous malformations [1, 3, 13]. The first two, which may be related, have groups of dilated thin-walled vessels, with appreciable intervascular nervous tissue in telangiectases and little or none in cavernous angiomas. Vessels are generally larger in cavernous angiomas and the likelihood of clinically significant hemorrhage much greater.

More complex are the arteriovenous malformations, also known as racemose angiomas, angiodysgenetic myelomalacia, and Foix-Alajouanine necrotizing myelopathy. Some recognize a venous type, but angiographic studies indicate that most of these are actually arteriovenous [1, 6]. The malformations consist of one or more anomalous arteries arising from radicular arteries along nerve roots and draining, without an intervening capillary bed, into one or more veins which communicate with veins on the surface of the cord [1, 8, 14]. Many of the anomalies form striking tangles of distended tortuous vessels on or in the cord; others are less conspicuous. Most are extramedullary, but a few are intramedullary only and some are combined. They are more commonly dorsal than ventral [5], and most frequent at the caudal thoracic and lumbar levels. Most are subdural, but epidural forms occur [8].

Hemangiomas (hemangioendotheliomas, hemangioblastomas, angioreticulomas) are regarded by most pathologists as true neoplasms of endothelium [3, 13]. They occur even less frequently in the nervous system than do vascular malformations.
These neoplasms are analogous to hemangiomas in other organs, appearing as discrete masses of tangled capillaries with or without cavernous or solid areas.

Materials and Methods

Formalin fixed, paraffin embedded tissues were stained with hematoxylin and eosin (HE), Gomori trichrome, Gomori reticulin, Mallory phosphotungstic acid hematoxylin, and luxol fast blue-periodic acid-Schiff methods.

Case Reports

Dog 1, a 10-month-old female Australian Shepherd, had an arteriovenous malformation. At the time of admission, the dog had had posterior ataxia and difficulty in getting up and down for 3 weeks. She was hypersensitive over the lumbosacral area and had a slight foreleg hypermetria. Signs were described as worse in the morning and improved after exercise. There was steady deterioration over a 3-month period with increasing ataxia and development of urinary incontinence. The dog was killed for necropsy.

The cerebrospinal fluid had a hazy xanthochromic appearance. There were 1390 erythrocytes and 18 leukocytes/mm³ and 210 mg/dl of protein. The urine had numerous leukocytes. Grossly the spinal cord at T₁₃-L₁ was black-red and soft for 3 centimeters. Leptomeningeal vessels ventrally in this area were prominent, but there was not the striking tangle of large vessels often seen in man. A few small brown areas were found centrally in the cord just cranial to the main lesion.

Microscopically, the vessels of the leptomeninges at T₁₃-L₁, especially ventrally and in the ventromedian fissure, were distended, tortuous, and thick-walled (fig. 1). Most of the thickening, which involved both arteries and veins, was collagenous, but some smooth muscle increase also occurred. Similar vessels appeared within nerve roots and epidurally about the dural sheath of a spinal nerve. There was diffuse leptomeningeal fibrosis.

The spinal cord in the affected area had many small vessels whose walls were collagenized, often to the point of obliteration (fig. 2). Their numbers were greatest in the ventral two-thirds of the cord. Many were mineralized. The walls of a few small arteries contained pink homogeneous material suggestive of fibrinoid necrosis [9]. Thrombosis was rare. There was an extensive increase in perivascular reticulin fibers throughout the damaged areas, and abundant perivascular and subpial collagen in the lesions of greatest duration. Only an occasional larger thick-walled vessel in the ventral columns might have been interpreted as being an actual part of the malformation. The abnormal, small parenchymal vessels were evaluated as altered secondarily by chronic hypoxia and not as being part of the malformation proper [1].

The parenchyma showed severe edema and necrosis in areas of greatest vascular abnormality. The damage was most severe centrally and about the ventromedian fissure. Occasional malacic foci were present. Gray and white substance suffered equally. There was widespread hemorrhage, both recent and old, in sites of edema.
Fig. 1: Arteriovenous malformation (dog 1). Anomalous vessels in ventral meninges and nerve roots. Hemorrhage in adjacent parenchyma. HE.

Fig. 2: Arteriovenous malformation (dog 1). Prominent small vessels in damaged parenchyma. HE.
Dog 2, a 6-year-old male Pointer, had had progressive ataxia for a month. The dog had difficulty standing and, when in lateral recumbency, had extension of all four legs. Weakness was progressive, the left rear leg finally becoming completely paralyzed. The head could not be raised because of neck rigidity. The dog was killed for necropsy.

There were firm swellings in the spinal cord at C₅ and T₅. These were caused by deep discrete foci, chiefly on the left side and reached the pia ventrally and along the ventromedian fissure (fig. 3). The cervical mass was 7×9 millimeters in transverse section and the thoracic mass 6×8 millimeters.

Microscopically, the lesions were well circumscribed with frequent lymphocyte accumulations and larger vessels at their margins. The adjacent cord parenchyma showed typical compressive changes, but no hemorrhage. In the masses, superficial and deep zones of differing appearance were evident. In a broad peripheral area, the
Fig. 4: Hemangioma (dog 2). Peripheral zone of abundant capillaries. Unencapsulated, with lymphocytes and larger vessels at margin. HE.

Fig. 5: Hemangioma (dog 2). Reticulin network in peripheral area. Gomori reticulin stain.

Fig. 6: Hemangioma (dog 2). Deep sclerotic zone with fewer capillaries and more fibers. HE.

Fig. 7: Hemangioma (dog 3). Peripheral and deep zones with focal necrosis (arrow). HE.
tissue consisted of capillaries and stroma in about equal amounts (fig. 4). The endothelium of the vessels was normal to plump and irregular, and was supported by a rich network of reticulin fibers (fig. 5). There was not much blood in the capillaries. The stroma consisted of fusiform or stellate cells in a fibrillar matrix. These cells varied from very elongated fusiform cells to large multipolar cells with homogeneous acidophilic cytoplasm and large single or paired nuclei. A few structures like pale nuclear inclusion bodies were seen. Occasional mitoses were seen in stromal cells in the peripheral zone. Tiny clumps of plasma cells were widely scattered in the lesion.

In the periphery of the mass, stromal fibers were not abundant, but centrally the lesion became more fibrillar and less vascular (fig. 6). Capillaries became much less frequent and there were narrow cellular cords, which suggested capillaries in the process of obliteration. In this deep zone there were abundant reticulin and collagen fibers as well as occasional necrotic foci. There was no evidence of hemorrhage into either the cord or the subarachnoid space.

Dog 3 was a spayed female Australian Shepherd, 7 years old. Over a 6-month period there had been right rear leg hemiparesis and some loss of right foreleg proprioception. A myelogram showed narrowing at C₄-₅.

The cerebrospinal fluid contained 40 mg/dl protein and 5 cells/mm³. The fluid was clear and colorless. The hemogram was normal. A laminectomy and durotomy were done at C₄-₅ and a 4×7 millimeter embedded, bulging, tan mass was removed from the right dorsolateral area of the spinal cord. The biopsy specimen (fig. 7) from dog 3 was identical to the necropsy lesions of dog 2, except that more deep necrosis was seen.

Discussion

Barring possible duplication, 20 vascular anomalies and eight hemangiomas appear to have been reported in the brain of animals [4, 7, 10, 16], but few have been described in the spinal cord. Spinal cord lesions in a dog [15] and a calf [11] seem to be cavernous angiomas because they consisted of thin-walled dilated vessels without intervening tissue. Also noted, without description, was a cavernous angioma in the ventral horn of the spinal cord of a horse [7]. Despite the statement that arteriovenous malformations of the nervous system are unknown in animals [7], it is probable that one report of such [12] in a horse qualifies, because numerous thick-walled small vessels, some obliterated, were described in the cord.

One dog had a ventral, largely extramedullary, subdural pattern of arteriovenous malformation with some epidural extension. There is evidence against direct pressure, thrombosis, and the “steal” phenomenon as mechanisms in cord damage [1]. The author speculated that the arteriovenous shunt led to raised pressure in normal extramedullary and intramedullary veins with a consequent decreased arteriovenous pressure gradient in the cord. This caused diminished intramedullary blood flow and chronic hypoxia. Damage to parenchyma and small vessels of the cord was the result of persistent and probably increasing hypoxia. Intramedullary damage can be
secondary to an entirely extramedullary malformation. Progress is usually steady but
sudden episodic advances may be caused by large hemorrhages or thrombosis.

To my knowledge no cases of intramedullary hemangioma have been reported in
the spinal cord of animals, although there is a report [16], without details, of an
hemangioma about the cauda equina of one dog and an hemangioma encroaching
on the cord from a vertebral body origin in a second. The hemangiomas in my dogs
were nearly identical to each other and closely resembled the neoplasms reported in
man [3, 13]. This tumor is considered to be a true neoplasm, growing slowly at the
periphery and sclerosing centrally. Infrequent mitoses near the margin are seen, and,
while the unencapsulated mass is not aggressively invasive, its slow growth does
interdigitate with and incorporate adjacent neural tissue into its periphery. The
decrease in vascularity and increase in stromal fibers centrally accords well with the
concept of sclerosing hemangioma in man [2]. How much of the enlargement of the
mass is neoplastic cell proliferation and how much is stromal increase is uncertain.
Even the nature of the stromal cells is undetermined, although majority opinion
labels them endothelial and neoplastic. It has been suggested that these neoplasms
may arise from the meninges and be related to angiomatous meningiomas [13].

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