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Presumed Venous Infarction in Spinal Decompression Sickness

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Summary: We describe the serial MR imaging findings in a patient with spinal decompression sickness. In the acute phase, the spinal cord was swollen, with increased T2 signal in the posterior part of the column; 1 month later, marked contrast enhancement was noted in the same location; and 2 months later, the swelling and T2 signal had decreased. MR imaging may facilitate the early diagnosis of spinal decompression sickness.

Decompression sickness is a clinical syndrome caused by an alteration in environmental pressure that results in the release of inert gas bubbles into tissue or blood that had previously been dissolved in the tissues (1). Decompression sickness has been reported by several authors (1, 2); however, to our knowledge, no previous reports have described MR signal abnormalities in the spinal cord associated with this syndrome. Early diagnosis and treatment of decompression sickness may lead to a better prognosis.

Case Report

A 57-year-old professional diver lost consciousness while returning to the surface after diving to a depth of 50 m. He recovered consciousness after several minutes and later reported chills. After 2 hours, quadriplegia developed, with numbness of his fingers and urinary incontinence. Three days later, the patient was admitted to our hospital for hyperbaric oxygen therapy.

Upon admission, flaccid quadriplegia was noted with diminished deep tendon reflexes of the arms and legs. Plantar responses were extensor. There was loss of position and vibration sense combined with hypesthesia below T10. CSF analysis revealed a normocytosis with an increased protein concentration of 237 mg/dL (normal, 10–40 mg/dL), an IgG level of 30 mg/dL (normal, <5 mg/dL), and myelin basic protein level of 40 mg/dL (normal, <4 mg/dL).

MR imaging was performed on a 1.5-T magnet. T1- and T2-weighted spin-echo pulse sequences revealed a swollen, edematous spinal cord extending from C2 to T2. T2-weighted images showed high signal intensity extending from C2 to T2 in the posterior and lateral aspects of the column. T1-weighted MR images of the same region revealed hypointense signal relative to that of spinal cord (Fig 1A–D).

Treatment with hyperbaric oxygen and intravenous glycerol and methylprednisolone were started, and the patient's neurologic status improved gradually, except for the disturbance of position and vibration sense. Follow-up MR imaging 1 month later showed extensive contrast enhancement confined to the posterior part of the column from C2 to C6 (Fig 2A–C). Two months after the event, MR imaging revealed reduced swelling and a decrease in size of the T2 signal abnormality and contrast enhancement (Fig 3A–C).

Discussion

Decompression sickness is classified into two groups. Type I includes joint pain, skin marbling, small patchy hemorrhages, and lymphatic obstruction; type II is further classified into four subgroups according to involvement of the CNS, spinal cord, inner ear, and lung (1). Spinal involvement occurs in 10% to 30% of cases (3). Although the pathophysiology of spinal decompression sickness is not completely understood, it is thought that bubbles of nitrogen accumulate in the intravascular space during decompression with a subsequent decrease in perfusion to the spinal cord, either by acute circulatory failure or by gas formation within the tissues (4). White matter of the upper or middle thoracic cord segments, especially in the lateral and posterior parts of the column, is reportedly affected most frequently, because it has a high fat content in which nitrogen is easily stored. Release of dissolved nitrogen gas during decompression is poor and therefore the inert pressure may become higher in white matter than in gray matter. We propose that nitrogen builds up in the white matter and that the resulting pressure causes damage. This mechanism may explain the vulnerability of white matter during decompression.

In 1995, Tournebise et al (3) reviewed 31 cases of spinal decompression sickness in which MR imaging was performed. Acutely all MR studies, except for one, were normal. The one abnormal MR examination was from a diver who had experienced paraparesis during a previous decompression incident. This MR examination showed an atrophic thoracic cord. In our patient, MR imaging revealed a swollen cervical cord and hyperintense T2 signal over nine segments from C2 to T2 in the posterior part of the column. The distribution of lesions was consistent with the autopsy findings of spinal decompression sickness reported by Palmer et al (5), in which sym-

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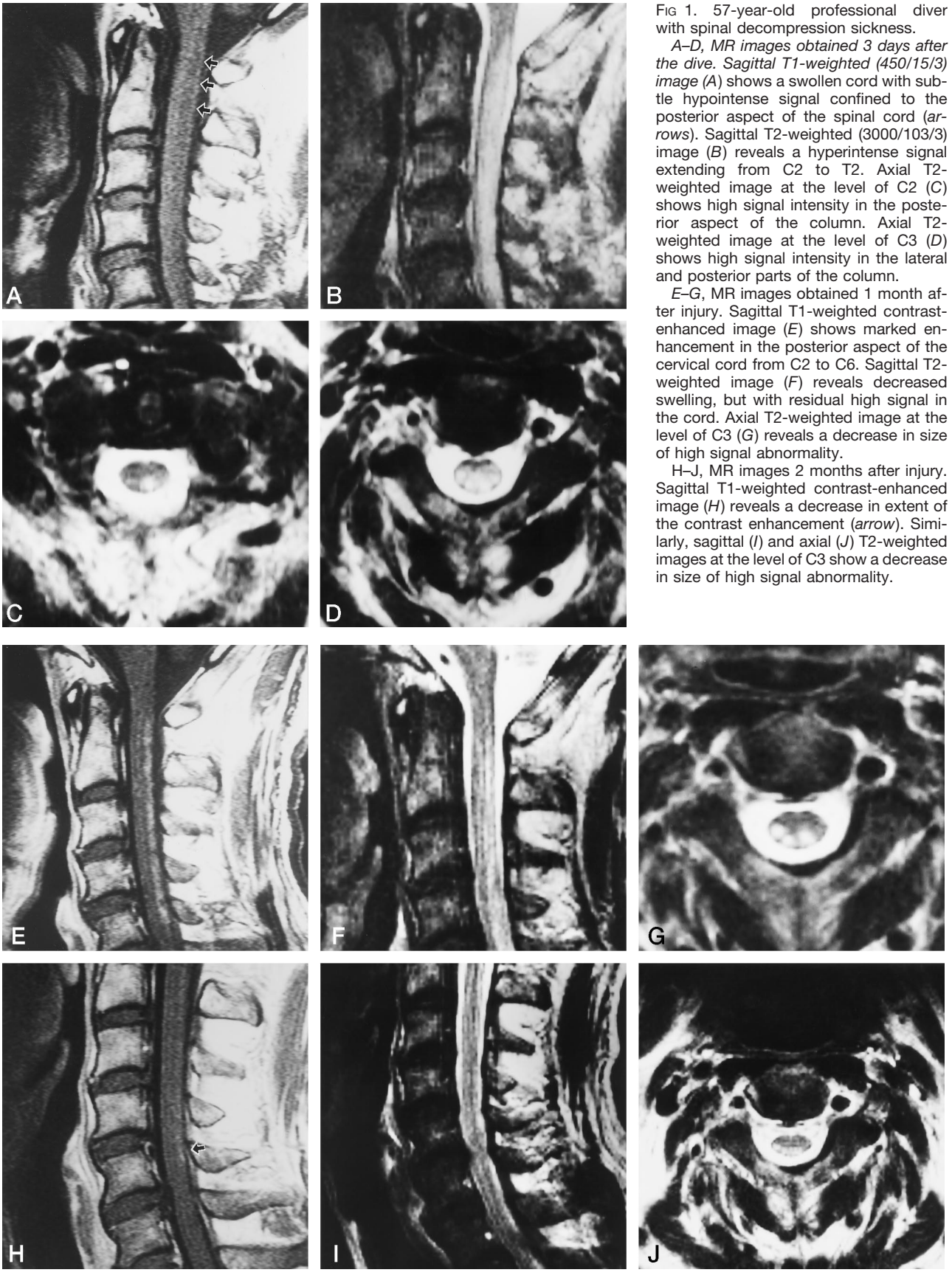

FIG 1. 57-year-old professional diver with spinal decompression sickness.

A–D, MR images obtained 3 days after the dive. Sagittal T1-weighted (450/15/3) image (A) shows a swollen cord with subtle hypointense signal confined to the posterior aspect of the spinal cord (*arrows*). Sagittal T2-weighted (3000/103/3) image (*B*) reveals a hyperintense signal extending from C2 to T2. Axial T2-weighted image at the level of C2 (*C*) shows high signal intensity in the posterior aspect of the column. Axial T2-weighted image at the level of C3 (*D*) shows high signal intensity in the lateral and posterior parts of the column.

E–G, MR images obtained 1 month after injury. Sagittal T1-weighted contrast-enhanced image (*E*) shows marked enhancement in the posterior aspect of the cervical cord from C2 to C6. Sagittal T2-weighted image (*F*) reveals decreased swelling, but with residual high signal in the cord. Axial T2-weighted image at the level of C3 (*G*) reveals a decrease in size of high signal abnormality.

H–J, MR images 2 months after injury. Sagittal T1-weighted contrast-enhanced image (*H*) reveals a decrease in extent of the contrast enhancement (*arrow*). Similarly, sagittal (*I*) and axial (*J*) T2-weighted images at the level of C3 show a decrease in size of high signal abnormality.

metrical softening and later sclerosis of the lateral and posterior regions of the column were found. They also reported that the pathologic features of spinal decompression sickness included perivascular hemorrhage and edema of the affected areas. MR images in our patient were compatible with the findings of these authors in regard to the distribution of hemorrhage and edema and to the nature of the injury.

From an imaging standpoint, spinal cord tumor, myelitis, arterial infarction, and multiple sclerosis must be considered in the differential diagnosis. In our patient, the clinical course and MR findings involving the posterior region of the column over nine segments helped differentiate infarction from other disorders (6–9). Although an arterial infarction may have been considered in our case, a venous infarction was more likely, because spinal decompression sickness has been induced experimentally by obstruction of venous drainage in the cord (10). Similar spinal cord lesions have been reported in cases of venous infarction (11). Venous infarction stemming from other causes usually has a subacute onset, whereas spinal decompression sickness develops acutely, as it did in our patient. In spinal decompression sickness, it has been reported that nitrogen bubbles lodge quickly in the spinal veins (4). The spinal lesion may therefore develop acutely, even though it is produced by venous infarction.

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