

Case report

Microhemorrhages in the central nervous system : report of a patient with microhemorrhages in brain and spinal cord

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Summary

A 53-year-old woman with microhemorrhages in the brain and spinal cord is described. This patient was initially seen with a reversible oculomotor paresis and hypertension, a year later she developed spinal cord symptoms. T2-weighted magnetic resonance imaging showed characteristic hypointense lesions in the brain and spinal cord consistent with microhemorrhages. Although the occurrence of microhemorrhages in the brain has been described before, the combination of brain and spinal cord microhemorrhages has not been reported yet. The observations in our patient suggest that microvascular changes related to hypertension are a common cause for these microhemorrhages.

Keywords : Microhemorrhages ; brain ; spinal cord ; magnetic resonance imaging.

Introduction

Although incidental observations of clinically silent microhemorrhages were described in pathological studies (Ishii *et al.*, 1984), a reliable estimation of the frequency of these microhemorrhages in vivo is now possible with magnetic resonance imaging (MR-imaging). With this technique even small intracerebral bleedings can be identified on T2-weighted images as small hypointense areas (Fazekas *et al.*, 1999 ; Kinoshita *et al.*, 2000). These hypointense areas represent regions of increased hemosiderin deposits. The hemosiderin deposits are related to the presence of hemosiderin containing macrophages, which infiltrate the hemorrhagic tissues (Kinoshita *et al.*, 2000 ; Offenbacher *et al.*, 1996 ; Roob *et al.*, 1999). It has now become clear that asymptomatic small intracerebral hemorrhages frequently occur in combination with larger intracerebral hematomas, multiple lacunar strokes (Kinoshita *et al.*, 2000, Offenbacher *et al.*, 1996 ; Roob *et al.*, 1999) or in association with the use of anticoagulantia in older patients with ischemic stroke and other atherosclerotic diseases (Kwa *et al.*, 1998). However, sometimes these hemorrhages were observed in previously healthy elderly persons (Kinoshita *et al.*, 2000). In this report, a unique case of a younger

patient is presented who developed not only intracerebral microhemorrhages, but also microhemorrhages within the spinal cord.

Case report

A 53-year-old woman was admitted to the hospital with complaints of diplopia since a week. She also noted a dropping eyelid on the right side and complained of a numbness of her right face. The medical history of this patient was unremarkable and she did not use medication or drugs. On examination the patient was adequate and co-operative, blood pressure was 160/100 mm Hg. Neurological examination revealed a mild oculomotor paresis on the right side with ptosis but normal pupillary reflexes. Furthermore, a disturbed sensation was present in the right face, especially in the distribution area of the second trigeminal nerve. No other signs of brain stem dysfunction were observed. There were no sensorimotor disturbances in the limbs and coordination was intact. However a generalized hyperreflexia was present, right planter reflex was extensor, left planter reflex was indifferent. MR-imaging (Philips, Eindhoven, the Netherlands) of the brain showed small intracerebral areas of hyperintense foci with a rim of hypointense signal in the brain stem located bilaterally in the ventral part of the pons and bilateral paramedian lesions at the border of pons and mesencephalon (Figure 1a, Figure 1b) and similar lesions in the hemispheres consistent with microhemorrhages (Figure 1c). We consider the ventral pontine lesions responsible for the pyramidal signs, the right-sided oculomotor paresis is most probably related to the rostral right paramedian hyperintense lesion at the level of pons-mesencephalon.

Complete recovery of the oculomotor paresis occurred within two months. A mild hypertension remained present, although medical treatment for hypertension was initiated. The patient was extensively screened for vascular risk factors. This included an extensive medical and laboratory investigation. The medical examination was unremarkable except for the hypertension. No signs of teleangiectasia were found on the skin. The follow-

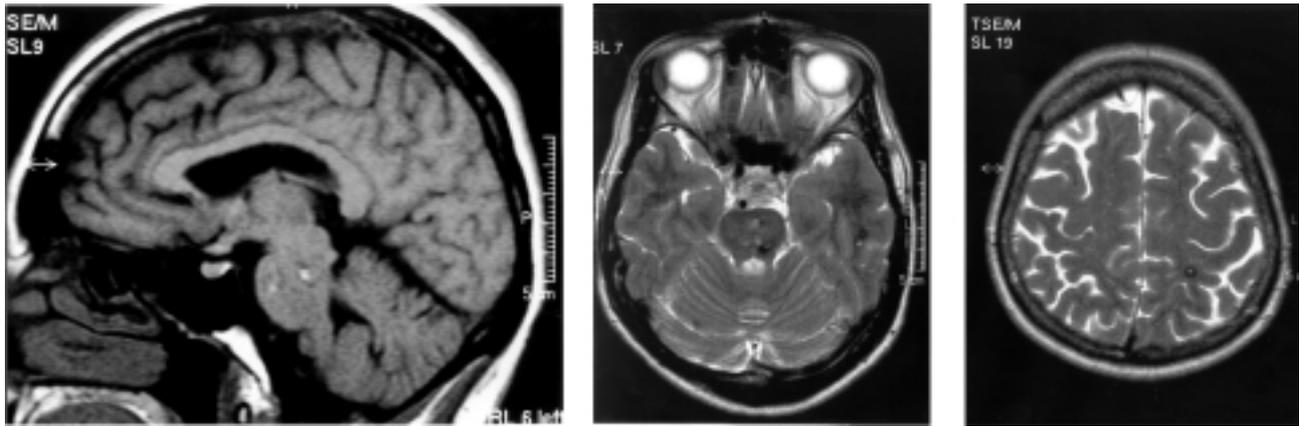


FIG. 1. — a : Saggital T1-weighted image demonstrating the presence of several microhemorrhages in the brain stem as is illustrated by the hyperintense signals surrounded by a hypointense rim of hemosiderin deposits. b : Axial T2-weighted image at the level of the brainstem showing central hyperintense lesions with hypointense rim in the brainstem located bilaterally in the ventral part of the pons and bilateral paramedian lesions at the border of pons and mesencephalon as a result of different stages of bleeding. c : Axial T2-weighted image of the brain showing a focus with hyperintense signal in the center and a very low signal rim typical for hemosiderin deposits.

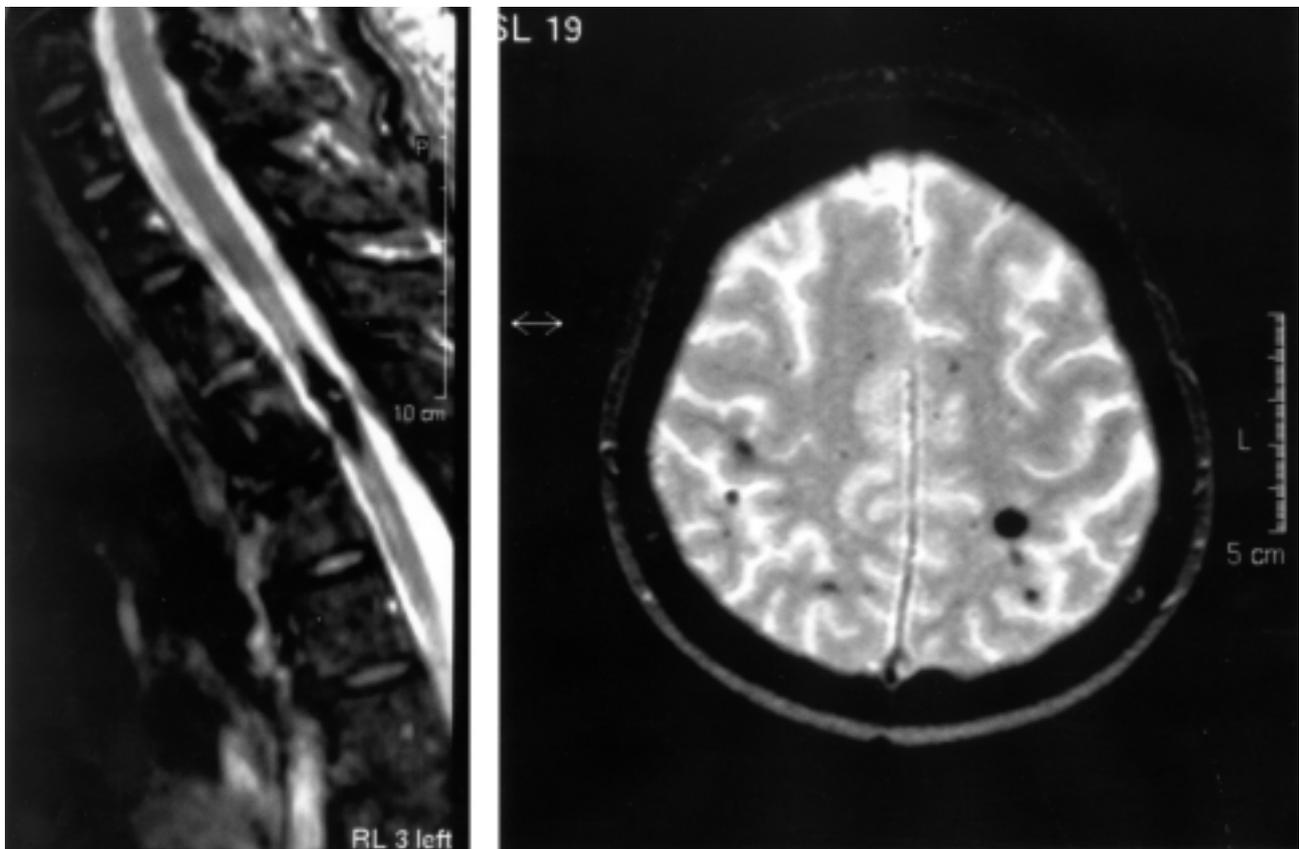


FIG. 2. — a : Saggital T2 (echo-gradient) -weighted image at the level of the seventh vertebral body. There is a large hypointense rim around the hyperintense focus in the myelum indicating hemosiderin depositions. b : Repeated axial T2 (echo-gradient) -weighted image of the brain at the same level as Figure 1c two years later showing an increase in number of microhemorrhages as is illustrated by widespread multiple hypointense signals.

ing laboratory investigations were all normal: X-films of the chest, ultrasonography of the kidneys, renal arteriography, electrocardiogram, a complete blood count, hepatic and renal function tests, urinalysis, glucose, electrolytes, cholesterol, urine catecholamines and their metabolites, Waaler-Rose test, tests for rheumatic factor and antinuclear antibodies, tests for anti-neutrophilic

cytoplasmatic antibodies, anti-perinuclear factor and complement C3 and C4, APTT (activated partial tromboplastin time), INR (international normalized ratio), fibrinogen, AT3 (antitrombine III), protein C and protein S, tests for antiphospholipid antibodies and lupus anticoagulants. Cerebral spinal fluid analysis included cell, protein, pyruvate, lactate, glucose, hemoglobin and bilirubin

measurements, but no abnormalities were found. The family history was negative for vascular diseases. Antihypertensive drugs (captopril) were prescribed but not regularly taken by the patient.

A year later the patient was again seen, now complaining of weakness and numbness of both legs. On examination, a mild paresis of both legs was noted with sensory disturbances reaching up to the level of Th10. Again there was a generalized hyperreflexia, now both planter reflexes were indifferent. The blood pressure was still elevated (ranging from 175/105 mm Hg to 150/110 mm Hg).

MR-imaging showed two areas of focal signal loss in the spinal cord at level of the thoracic vertebral body 7 and 12 on T2-weighted images typical for small spinal cord hemorrhages (Figure 2a). Repeated MR-imaging one year later showed an increased number of lesions in the brain (Figure 2b).

Discussion

A unique case of a patient is reported, who not only developed intracerebral microhemorrhages but also hemorrhages in the spinal cord. The presence of small intracerebral haemorrhages has recently been described by several authors (Kinoshita *et al.*, 2000 ; Roob *et al.*, 1999 ; Kwa *et al.*, 1998). These hemorrhages usually were asymptomatic and frequently occurred in patients with ischemic strokes. In fact a relation between higher age, the presence of white matter lesions and lacunar infarcts and cerebral hemosiderin deposits was demonstrated (Kwa *et al.*, 1998). However, also in healthy elderly people cerebral microhemorrhages were observed. These lesions appear as small areas of signal loss on gradient echo T2-weighted images (Kinoshita *et al.*, 2000).

Comparison of the intracerebral lesions with histopathological analysis revealed that these areas showed focal hemosiderin deposition indicative of previous extravasation of blood (Fazekas *et al.*, 1999). To our knowledge, no patient with a combination of intracerebral and spinal cord microhemorrhages has been reported before.

Intracerebral hemorrhages are usually massive and result in serious neurological disturbances. The single most important clinical risk factor for intracerebral microhemorrhages is hypertension and related secondary changes in the vessel walls (Roob *et al.*, 2000), other major risk factors related to this condition are the use of anticoagulantia, cerebral amyloid angiopathy (Fazekas *et al.*, 1999), and the presence of vascular malformations. Spinal cord haemorrhages are usually associated with vascular abnormalities such as spinal cord arteriovenous malformations, cavernoma or hemangioblastoma (Odom, 1962 ; Yu *et al.*, 1994 ; Deutsch *et al.*, 2000). Incidentally, both brain and spinal cord hemorrhages are described, e.g., as a late effect of

craniospinal radiotherapy (Allen *et al.*, 1991) or in angioplastic disorders of the central nervous system (Kadoya *et al.*, 1994). These hemorrhages were usually large and resulted in considerable neurological damage.

The combination of intracerebral and spinal hemorrhages in our patient indicates that both the cerebral and spinal vascular system were equally prone for hemorrhages, suggesting a common pathophysiological mechanism. No vascular malformations or other abnormalities were observed in the spinal canal or in the brain of our patient. There was also no history of radiation. Therefore, the most obvious etiologic factor related to both the intracerebral and spinal cord hemorrhages is the persistent presence of hypertension. Although the cerebral microhemorrhages were clinically silent or resulted in a reversible oculomotor paresis, the spinal hemorrhages were symptomatic in our patient and there was no complete recovery. Moreover, repeated MR-imaging showed that there was an increased number of lesions intracerebral, illustrating that uncontrolled hypertension may lead to disease progression.

In conclusion, this report illustrates that microhemorrhages in the central nervous system are not restricted to the cerebral structures but may also occur in the spinal cord. The primary risk factor for this condition seems to be hypertension and related vascular changes. MR-imaging is an excellent technique to detect these hemorrhages.

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