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Case Report

Severe depression and behavioral disturbance, an unusual presentation of intracerebral hemorrhage in a 78-year-old man

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Abstract

We describe a case of a 78-year-old man who, on two occasions, had intracerebral hemorrhage with an atypical, predominantly psychiatric presentation: once with major depression without focal neurological signs and the second time with severe behavioral disturbance and only mild facial paralysis.

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Keywords: Elderly; Intracerebral hemorrhage; Psychiatric manifestation; Depression; Behavioral disturbance; Cerebral amyloid angiopathy

1. Introduction

Intracerebral hemorrhage (ICH) is the second most frequent subtype of stroke (15%–20%) [1,2], after ischemic stroke, with a 30-day mortality rate of 35% to 52% [3,4]. Patients with ICH typically present with sudden onset of a focal neurological deficit that progresses over minutes to hours with accompanying headache, nausea, vomiting and decreased consciousness. However, ICH sometimes is difficult to diagnose in old patients because they present with a wide variety of symptoms and signs. We illustrate this difficulty with the following case report.

2. Case report

In March 2007, the patient, a 78-year-old man, experience a mild headache that lasted for some hours. For the rest of

that day, he felt tired and depressed. The next morning, he woke up with a severe left frontal headache that disappeared later that day. During the following days, he developed multiple depressive symptoms, including anhedonia, crying fits, lack of energy, fatigue, insomnia, anorexia, extensive worrying and feelings of guilt and worthlessness. His medical and family history did not reveal any psychiatric disorders. He consulted a general practitioner, who prescribed oxazepam for an adjustment disorder, later followed by citalopram up to 30 mg/day for depressive disorder. A primary psychiatric disorder was accepted by the patient based on substantial psychosocial stressors in the past few months because of the sudden death of his sister, complicated inheritance, giving up an executive function in the board of a pension fund and conflicts within a directorate. Although his symptoms gradually diminished over the next few weeks, he requested to be referred to an academic psychiatric outpatient clinic for an evaluation of his depression.

His medical history revealed hypertension and stable angina pectoris. He had been treated with acetylsalicylic acid 80 mg/day, tildiazem 300 mg/day and isosorbide 5-mononitrate 25 mg/day. The patient did not smoke and his alcohol intake was one to two drinks per day. He had

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studied economics, had worked as financial chief executive officer of a large company, and later served as a member of the board of directors. Physical/neurological examinations and blood/urine tests did not reveal any pathology.

Cognitive and psychiatric examinations demonstrated depressed mood, problems with concentration, planning and organization, mild impairment of short-term memory and difficulty in processing both verbal and nonverbal information. The dose of citalopram was then increased to 40 mg/day. In view of the rapid development of a severe first depressive episode — in the absence of a psychiatric history — and the associated cognitive impairments, magnetic resonance imaging (MRI) of the brain was performed (Fig. 1). His MRI showed hemorrhage in the lateral right temporal lobe. Subsequent magnetic resonance angiography found no evidence of cerebral arteriovenous malformation or brain aneurysm. The diagnosis was changed to a major depressive disorder due to a general medical condition, the ICH, and citalopram was tapered off. Because of the lobar location of the hemorrhage and the absence of hypertension, cerebral amyloid angiopathy (CAA) was the presumed underlying pathology responsible for the ICH, and the patient was treated conservatively. Computed tomography (CT) scan of the brain 1 month later showed some resorption of the bleeding. On follow-up 2 months later, the patient no longer had any depressive symptoms. His mood, memory and activities of daily living had all returned to the level before the hemorrhage.

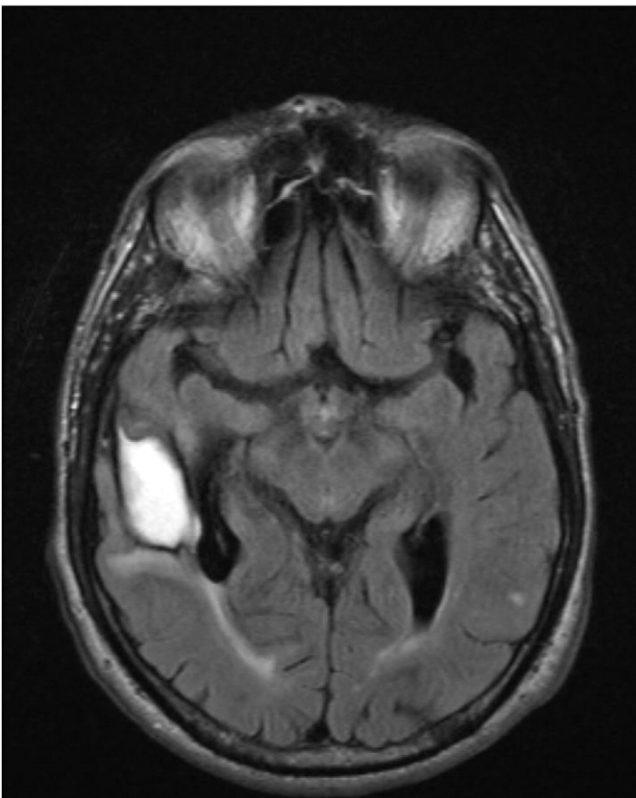


Fig. 1. MRI: Hemorrhage in the lateral right temporal lobe.

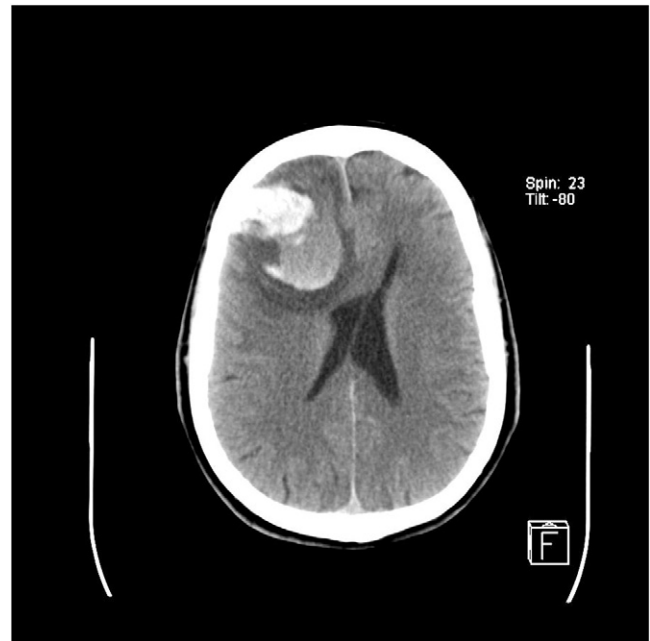


Fig. 2. CT: Hemorrhages from different dates (old and recent) in the right frontal lobe with midline shift.

In January 2008, the patient was admitted to the emergency department because of sudden behavioral disturbance. His wife mentioned a sudden headache that had lasted a few minutes 2 days before admission. The following day, he had developed behavioral disturbances such as walking around the house naked, reading the newspaper in the shower and urinating everywhere. He had become indifferent. Loss of consciousness, weakness or sensory impairment and history of head trauma were not reported. Results of a general physical and neurological examination were unremarkable, except for a mild left-sided central facial paresis. Blood and urine tests were normal. Cognitive and psychiatric examination showed deficits in orientation, concentration and memory. There was no spontaneous speech and his answers were short. Brain CT demonstrated hemorrhages from different dates (old and recent) in the right frontal lobe with midline shift (Fig. 2).

Acetylsalicylic acid was discontinued, and the patient was treated conservatively at the neurosurgical intensive care unit. The behavioral disturbances gradually disappeared, and 2 weeks later, he was transferred to a rehabilitation center for physical and cognitive rehabilitation. In October 2008, he was discharged home without neurological impairment but still with memory deficits. His cognitive functions continued to deteriorate, and at the last follow-up in 2010, vascular dementia was suspected; however, he and his wife have thus far refused further diagnostic evaluation.

3. Discussion

The presence of CAA should be suspected clinically in patients older than 60 years old who have multiple

lobar hemorrhages in the absence of an obvious cause [5]. Old patients with ICH may present with a wide variety of symptoms and signs, but almost always invariably, these include focal neurological deficits. Cerebral amyloid angiopathy is found most frequently in association with clinical Alzheimer's disease (AD) [6,7] but also occurs in some nondemented old people [8]. Our patient had neither a history of dementia nor psychiatric disorders.

Patients with AD also often have a history of arterial hypertension; CAA and hypertension are the two major known risk factors for ICH [9,10].

The last years have seen a growing debate on the role of aspirin in patients with dementia. Aspirin use has been associated with lower risk for AD [11], but recent studies could not confirm this effect [12,13] and its role in vascular dementia is unclear [14]. However, a meta-analysis of 22 randomized trials of low-dose aspirin has found that low-dose aspirin increases the risk of major bleeding, major gastrointestinal bleeding and ICH by 1.7- to 2.1-fold [15]. At the center of this debate is the question whether the presence of CAA is causing this increased risk for ICH with anticoagulation [16]. Furthermore, aspirin has been associated with increased ICH volume growth and poor outcomes at 3 months [17]. The continuation of aspirin in our patient may thus have contributed to the occurrence of the rebleeding.

The exact etiology of neuropsychiatric symptoms in patients with ICH is not fully understood. The etiology has been divided in two mechanisms: first, as a psychological reaction to the physical impairment that results from stroke; second, as a biological reaction caused by the neuronal injury [18,19]. In this patient, the first mechanism was not a very likely explanation, as he had no physical or functional impairment due to his stroke. Concerning the second mechanism, ICH can cause primary and secondary neuronal injury. Immediate effects of ICH are that extravasated blood accumulates and compresses surrounding brain tissue, thereby increasing intracranial pressure and leading to primary injury. A subsequent effect of ICH is the inflammatory process, characterized by the accumulation and activation of inflammatory cells and mediators within the hemorrhagic brain, which contribute to secondary injury [20]. Brain imaging has increased our knowledge about the neuroanatomical structures involved in neuropsychiatric symptoms and mood regulation [21]. Although this knowledge may stimulate research into the lesion–location theory linking poststroke depression to specific neuroanatomical locations, empirical evidence remains inconclusive [22]. Future studies should explore whether this can be explained by a large number of brain regions involved or whether neuropsychiatric symptoms are primarily caused by compensating secondary mechanisms such as an inflammatory response.

4. Conclusion

Intracerebral hemorrhage is a medical emergency with high mortality and morbidity rates; therefore, it should be

promptly recognized and diagnosed. Neuroimaging is not advocated routinely in old-age psychiatry for patients with classic, nondementing psychiatric disorders. However, when an old patient without a psychiatric history suddenly presents with a late-onset, classic psychiatric disorder, cerebral pathology should be ruled out.

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