

Asymptomatic interval in delayed traumatic intracerebral hemorrhage: report of two cases

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Abstract

Delayed traumatic intracranial hemorrhage (DTICH) is seen mostly in trauma to the occipitoparietal region by countercoup mechanism. It is most encountered within the first posttraumatic 10 days, particularly in the first 3. Herein, two cases of delayed traumatic intracerebral hemorrhage were discussed, first one presented with headache and vomiting who had been asymptomatic for 168 days after head trauma and the other presented with dysarthria and hyperkinesias after 92 days of asymptomatic interval, either being longer than that of the previous cases reported in the literature. Despite a long time elapse, DTICH should be considered in the differential diagnosis in the patients with history of head trauma that manifests at later stages with intracranial pressure elevation symptoms such as headache, vomiting.

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1. Introduction

Delayed traumatic intracranial hemorrhage (DTICH), which was first described in 1891 by Bollinger as ‘traumatische Spät-Apoplexie’ is encountered after severe head trauma in 2–10% of the cases [10,12]. These hemorrhages are frequently observed within the first 10 days, particularly in the first 3 [6]. In this report, two cases of delayed intracranial hemorrhage with posttraumatic intervals of 92 and 168 days, both being longer than that of the previously reported cases are discussed with relevant literature.

2. Report of cases

2.1. Case 1

A 27-year-old man was admitted to our hospital with the complaints of gradually deteriorating headache for over 2 weeks and vomiting for the last 2 days. In his previous history was found an impact on the left side of his head when he sustained a car accident 168 days ago, which had led him to be observed in another health center because of headache for 3 days. The Glasgow Coma Scale (GCS) was reported to be 15 on the initial neurological exam right after the trauma. On admission, he was neurologically intact but the cranial computed tomography (CT) revealed a hyperdense mass, not enhancing with contrast, with partial peripheral edema on the right temporal lobe. On cranial magnetic resonance (MR) images, the 3 × 4 cm mass with peripheral edema was considered to be consistent with hematoma (Fig. 1). No pathology was noted on 4-channel digital subtraction angiography (DSA), except for the elevation of M1 and M2 segments of the right

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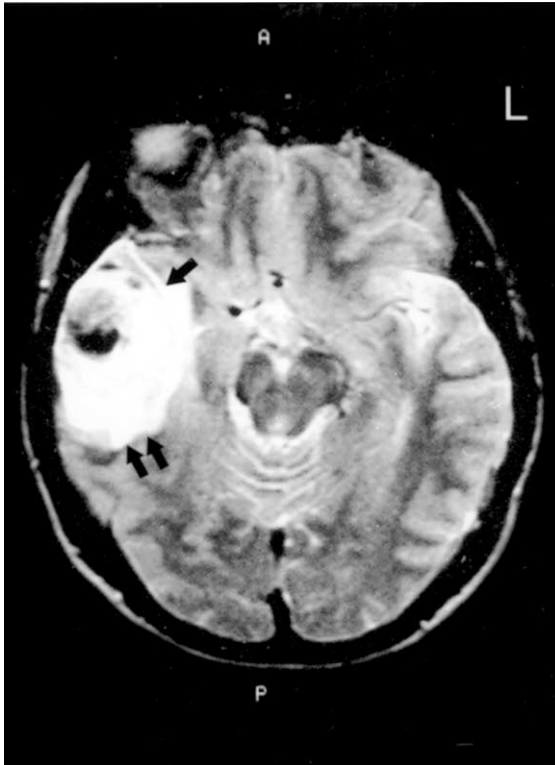


Fig. 1. On cranial MR images, edema (double arrows), hemosiderin ring (arrow) and hypo-hyperintense foci are observed.

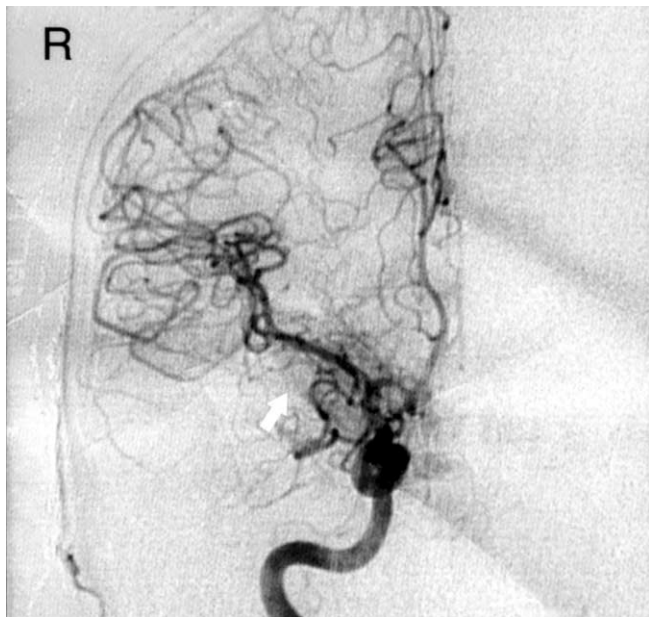


Fig. 2. On angiography, elevation of the M1 and M2 segments of right middle cerebral artery is demonstrated (white arrow).

middle cerebral artery (Fig. 2). Blood count, blood chemistry, and bleeding and coagulation times were found as normal. The patient had no systemic disease, and subsequently was operated on. Pathological investigation confirmed the diagnosis of hematoma. On 1-

year follow-up, he was doing well and postoperative CT was unremarkable except the postoperative changes.

2.2. Case 2

A 74-year-old woman was admitted with the complaints of dysarthria and hyperkinesias for over 1 week. Her previous history revealed a car accident 92 days ago, which caused swelling and color change on the left side of her forehead due to the impact. At that time the emergency service in another hospital found GCS as 15 and her cranial CT unremarkable. On the day of admission to our department, neurological status was normal except for hyperkinesias. On cranial CT, a hyperdense mass was demonstrated on the left temporal lobe. Cranial MR images revealed a hyperintense mass with partial hypointense foci with surrounding edema, which was consistent with hematoma. 4-Channel DSA was unremarkable. Bleeding and coagulation times and blood pressure were found in physiological ranges. Blood sugar was elevated due to preexistent diabetes mellitus demanding oral medication for over 10 years.

3. Discussion

DTICHs usually develop in contusion areas in temporal and frontal regions due to countercoup mechanism following occipitoparietal trauma [4,5,13]. Diagnosis is made on the basis of deterioration in the neurological status and serial cranial CT's because of intracranial pressure elevation symptoms [2,6]. However, as was in our first case delayed intracranial hemorrhage may well be seen in cases without any alteration in neurological status or intracranial pressure [1,12]. At about 6 months follow-up, of the case cranial CT revealed a delayed hematoma in the right temporal lobe opposite to the impact site, similar to those previously reported [4,5,13]. In our second case, post-traumatic CT taken in the early posttraumatic period was also normal. Hemorrhage was in the temporal lobe, which surprisingly was on the same side as the impact. Hemorrhages of both cases obtained on MR images were consistent with the subacute stage and the time interval for diagnosis was consistent with the interval needed for the onset of complaints [9].

Fukamachi et al. [4,5] reported that mechanical damage to vessel walls by trauma and the related connective tissue neuropills, was the main causative factor in the development of delayed intracranial hemorrhages. Secondary factors in the development of these hemorrhages are reported to be the reduction in the intracranial pressure either medically or surgically, hypoxia, hypercarbia, hypotension, hypertension, vasoparalysis, bleeding diathesis [11], and alcohol intake [5–7,13]. Trauma was not severe enough to alter GCS score

in our cases, and the absence of secondary factors may have also played a role in the prolongation of this interval. Diabetes mellitus has an effect of increasing fragility of the cerebral vessels and in turn, tendency to bleed by aging [8]. Besides the factors mentioned above, we believe that diabetes could have been such a secondary promoting factor in the development of hematoma in our second case.

Traumatic ‘Spät-Apoplexie’ was originally based on four criterias regarding the pathophysiological mechanisms. (1) A definite history of the minor head trauma and young age. (2) An asymptomatic interval longer than 24 h. (3) An apoplectic event. (4) Absence of vascular disease [3]. This description based on four criteria has become controversial particularly after the advent of CT [3,4,13]. Recently, delayed traumatic intracerebral hematomas have been reported to occur rather following severe head traumas and in elderly people even within the first 24 h [4–6,13]. Hematoma location and/or early diagnosis with the help of CT before development of any neurological deterioration may prevent symptoms of apoplexia to be included in the above mentioned criteria. Both cases of us did not correlated completely with these criteria and we believe that the term ‘traumatic Spät apoplexie’ does not cover any longer all types of delayed traumatic intracerebral hematoma.

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