Interhemispheric Subdural Hematoma in a Patient with Chronic Renal Disease: Unusual MR Demonstration

Kronik Böbrek Yetmezliği Olan Bir Hastada İnterhemisferik Subdural Hematom: Olağandışı MR Bulguları

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Abstract: Despite the fact of widespread availability of computed tomography and magnetic resonance imaging, interhemispheric subdural hematomas are still rare. An unusual magnetic resonance appearance of an interhemispheric subdural hematoma in a patient with chronic renal disease is presented here. Etiological factor, shape of the interhemispheric hematoma and different aged blood products differ the case from previously published cases in the literature.

Key Words: Chronic renal disease, interhemispheric subdural hematoma, magnetic resonance imaging

Özet: Bilgisayarlı tomografi ve manyetik rezonans görüntülemenin yaygın olarak kullanılmasına rağmen interhemisferik subdural hematomlar halen nadir olarak görülmektedir. Burada, kronik böbrek hastalığı olan bir hastada gelişen, değişik manyetik rezonans bulguları olan bir interhemisferik subdural hematom olgusu sunulmuştur. Etiolojik unsur, interhemisferik subdural hematomun şekli ve farklı dönemlerdeki kan elemanlarının varlığı olguyu daha önce yayınlanmış olanlardan farklı kılmaktadır.

Anahtar Sözcükler: İnterhemisferik subdural hematom, kronik böbrek hastalığı, manyetik rezonans görüntüleme

INTRODUCTION

Interhemispheric subdural hematomas (ISDHs) are of special interest due to their rarity and need for precise definition prior to surgery. It was first described by Jacobson in 1955 (1,3,5,7,10). Recent review of Borzone et al. revealed that there are few cases demonstrated by magnetic resonance (MR) (1). Presented case has a different appearance from the previously reported cases which were usually located at the parasagittal region with an inner convex shape. Also chronic forms of ISDH are quite rare (1,3,5,6,7,10). Most likely etiological factor was chronic renal disease in the presented case.

CASE REPORT

A 69-year-old male was admitted to the emergency room with sudden unconsciousness. He had hypertension for 22 years and was diagnosed as having chronic interstitial nephritis a year ago. Additionaly, he had a history of tuberculosis lymphadenitis. After hospitalization for 3 weeks with the diagnosis of tuberculous infection, he was discharged two weeks ago. He was on

antihypertensive and antituberculous therapy. No neurologic deficit was noticed during hospitalization. He returned to the emergency room after two weeks with sudden unconsciousness. No trauma was noticed in his history. His hemoglobin was 10.8 g/dL and hematocrit 30.4 %.

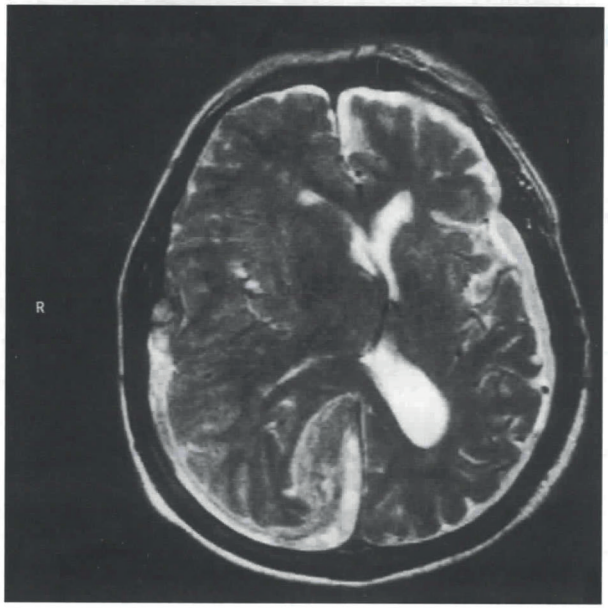
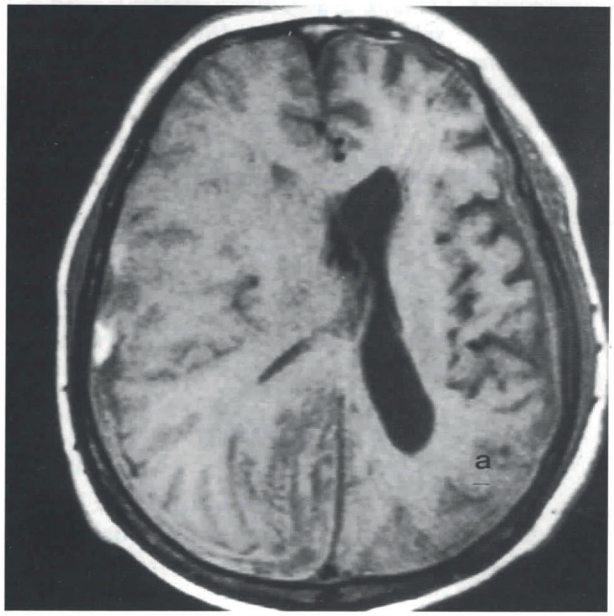


Figure 1: ISDH with remarkable midline shift. Two different signal characteristics are noticed which are more bright on the outer side than on the inner larger intermediate component (turbo SE, axial, TR/TE 2880/15 msec).

At neurologic exam he was comatose and had anisocoria. Pupillary reflex was negative on the right and slow on the left. He had mild flexory response on the right to painful stimulation and hemiparesis on the left. Babinsky was positive on the left.

MR examination demonstrated interhemispheric subdural hematoma which was extending to the right temporal subdural space which had an inner intermediate and outer increased intensity with remarkable midline shift on T2 weighted (T2W) transverse images (Figure1). It was isointense to brain tissue on T1W images with a superior hyperintense component (Figure 2). On coronal and axial gradient echo images, it was occupying the whole right middle cranial fossa causing superior and medial displacement of right temporal lobe, resembling a sandwich (Figure 3). It was mainly dark on gradient echo images with a posteriorly small outer rim with slightly increased intensity (Figure 3). Intensity characteristics was suggesting chronic interhemispheric subdural hematoma with acute and subacute components due to multiple episodes of rebleeding. Hypointensity on gradient echo images represented the acute component, and hyperintensity on T1W images represented the subacute component. Thin left sided subdural hematoma with low intensity on T1W and higher intensity on T2W images was also noticed representing chronic subdural hematoma (Figures 1, 2a).



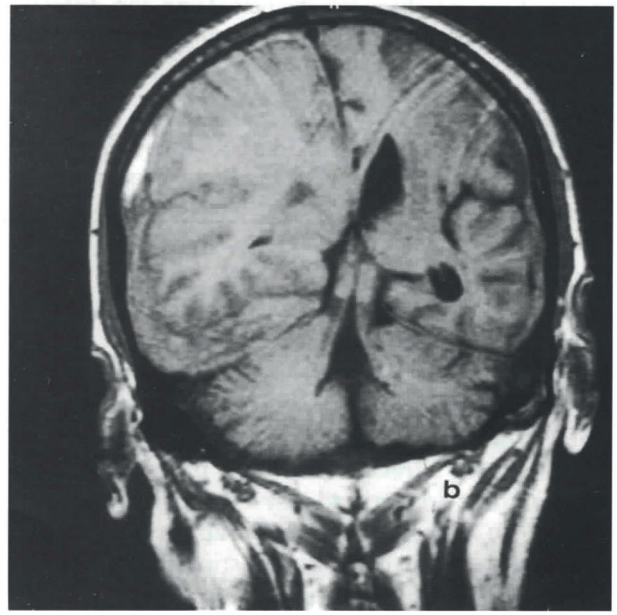


Figure 2, a) T1W axial image with a hyperintense small area suggesting rebleeding of the right subdural hematoma. Also, notice the left sided isointense subdural lesion with an apparent thick membrane (TR/TE 500/15 msec), b) T1W coronal image (TR/TE 600/15 msec) shows bilateral subdural hematomas isointense to brain tissue which is larger on the right with a superior hyperintense component.

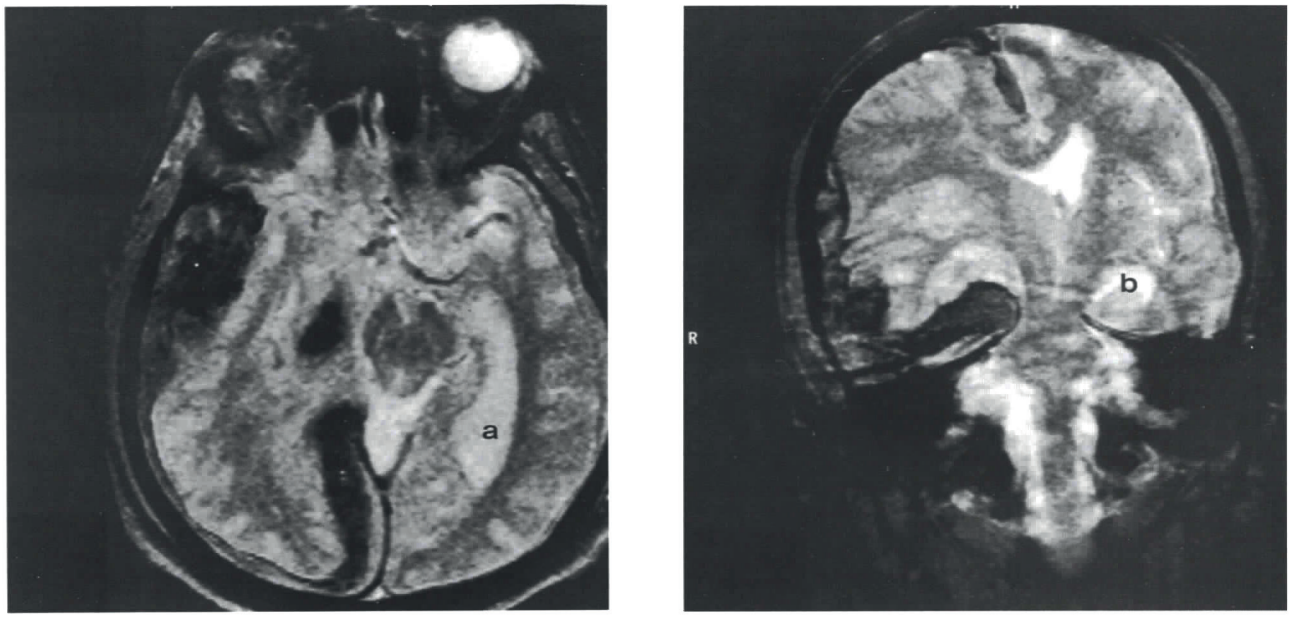


Figure 3, a) Transverse gradient echo (TR/TE 800/35 msec. FA 20) image revealed outer thinner hyperintense and inner thicker dark component. See compressed right temporal lobe resembling a sandwich, b) flash T2 coronal section (TR:800 msec, TE:35 msec) shows right sided hematoma in the subdural space filling middle cranial fossa with an interhemispheric component. Apparent midline shift and ventricular compression with superior displacement of the right temporal lobe are present.

Emergent surgery was performed with right temporoparietal craniectomy and interhemispheric subdural hematoma was evacuated. Patient did not improve and died on the third day of the surgery.

DISCUSSION

ISDHs are rarely seen entities, though the widespread availability of CT and MR. For the neurosurgeon, the most important point of ISDH, is the site of the craniotomy prior to surgery for appropriate management. Recent review of Borzone et al. revealed that there was only one case which was demonstrated by MR among 33 patients (1).

Trauma, anticoagulant therapy and rupture of aneurysms are the known etiologic factors for ISDH (1,7). Among the causes of ISDH, hemorrhagic diathesis due to renal disease, has not been mentioned previously, which was the most likely cause in our case. Chronic renal disease patients have a tendency to bleed because of their abnormal hemostasis (2,6).

The configuration of the ISDH was another interesting point of the presented case (Figures 2b, 3b). They are usually located on the convexity of the cerebral hemispheres at the parasagittal area with an inner convex margin (7). The shape of the temporal lobe was resembling a "sandwich" due to its compression by the laterally, medially and inferiorly placed components of the subdural hematoma (Figures 2b, 3b). Even after the surgery the primary site of bleeding was not clear. It could have started from the interhemispheric side and leaked into the laterally and inferiorly placed subdural space or vice versa (1).

Recurrent bleeding phenomenon from the hematoma capsule as an etiological factor for the development of chronic subdural hematoma (SDH) was first assumed by Putnam and Cushing in 1932. Sato and Suzuki's ultrastructural observations of the capsule of chronic SDH showed that inner capsule of chronic SDH presents no significant vascularization. The external membrane is very rich in blood vessels and contains giant capillaries. Inflammation and vascular degeneration lead to bleeding easily and repeatedly contribute to chronic SDH (6). Although chronic subdural hematomas make up 25 % to 50 % of all subdural hematomas, chronic ISDHs are quite rare. Fruin et al. explained the paucity of chronic ISDH as they tend to migrate over the convexity as it liquifies. According to them, Ogsburg et al. reported a similar case, and Hirahawa et al. found no cases of chronic ISDH in their large

series. Fruin et al. arises the question of some chronic convexity subdural hematomas may originate as ISDH with a persistent interhemispheric component (4). Although trauma is not the issue of this particular case, it is not possible to show the source of the bleeding during surgery in ISDH, but some authors define a parafalcian bridging vein laceration due to linear acceleration of the brain at the time of trauma (1,4).

MR of the case has different signal properties which shows rebleeding of the ISDH. Inner part of the layering had low signal intensity in both gradient and turbo SE T2W images and was isointense to the gray matter in T1W images which is consistent with the presence of deoxyhemoglobin of the acute hematoma. Outer part of the right sided hematoma had higher signal in T2W images which was similar to the left side suggesting chronic hematoma (8). High intensity is seen on T1W images in chronic intracerebral hemorrhage but not in chronic subdural hematomas, the lesions are isointense to the gray matter or hypointense than the gray matter (9) . Furthermore, hemosiderin is rarely seen in chronic subdural hematomas due to the clearance of the blood products in the absence of blood-brain barrier. Small hyperintense area in the right side can be explained by the presence of extracellular methemoglobin of the subacute component (8). Wilms et al. explained the high signal intensity in one of their chronic subdural hematoma cases as a result of impaired clearance of blood degradation products, so that high signal from methemoglobin may persist in the chronic stage (9).

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