Chronic Supra-and Infratentorial Epidural Hematoma. Case Report

ABSTRACT

Chronic epidural hematoma is rare and not a well-known clinical entity. We present a 10-year-old male who complained of headache, nausea and diplopia due to supra- and infratentorial chronic epidural hematoma. He was operated with two burr holes located supra- and infratentorially. The hematoma was like motor-oil liquid. Correct diagnosis and surgical intervention gave the patient a chance to recover.

KEY WORDS: Chronic epidural hematoma, Surgery, Trauma.

INTRODUCTION

Acute epidural hematoma (AEDH) is a well known clinical entity, but chronic epidural hematoma (CEDH) is uncommon, therefore, is not known clearly (5). In all epidural hematomas, the incidence of CEDH is between 3.9% and 30% (1). CEDH occur more commonly in younger ages (6).

We present a case of 10-year old male who presented with chronic supra- and infra-tentorial epidural hematoma.

CASE REPORT

A 10-year-old male presented with headache, nausea and diplopia. Although he complained of headache, diplopia and nausea, there was no papilledema or lateral gaze palsy with the neurological examination. His Glasgow Coma Score (GCS) was 15. CEDH was diagnosed on cranial computerized tomography (CT). The hematoma was more likely to be hypodense to brain parenchyma, unlike an AEDH, and there was a hyperdense line of hematoma margin without intravenous contrast administration. (Figure 1a, b).

The patient underwent operation for evacuation of supratentorial and infratentorial hematoma.

Two burr holes were opened 2 cm above and below the transverse sinus imaginary line, 2 cm lateral to the midline. The feature of the hematoma was motor-oil liquid. The patient's diplopia in the left eye persisted during the postoperative period and a lateral gaze palsy appeared. The lateral gaze palsy later recovered during his inpatient stay, but did not resolve completely.

DISCUSSION

CEDH are defined in various ways in the literature. Hooper (3) classified traumatic posterior fossa epidural hematoma as acute, subacute, and chronic, with the beginning of symptoms within the first twenty-first hour of trauma, between second and seventh days after trauma, and later, respectively. Sparacio et al (8) defined as CEDH 4

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Figure 1A: Axial CT scan revealing partial mass effect of hematoma and compression of fourth ventricle.



Figure 1B: Axial CT scan revealing supratentorial hematoma

cases of EDH diagnosed 48-72 hours after head injury. Iwakuma and Brunngraber (4) examined EDH histologically and defined CEDH as cases diagnosed more than 13 days after head injury based on the finding of capsule ossification. Bradley (1), using magnetic resonance imaging, defined CEDH as cases diagnosed more than 14 days after head injury, based on the breakdown of haemoglobin products on T1- and T2-weighted images. Tatagiba (9) and Zuccarello et al (10) defined CEDH as cases diagnosed between 12 and 14 days after trauma based on hematoma organization and calcification findings. After being involved in a fight, our patient had exceptionally fallen down and suffered a generalized tonic-clonic epileptic seizure for a very short period, two days before admission to the hospital. In our case, CEDH was diagnosed 48 hours after the head trauma. There was no other head trauma in the history. This time period is consistent with the Sparacio et al (8) study.

Headache, nausea, vomiting, memory loss, hemiparesis and unconsciousness may be the clinical manifestations of supratentorial CEDH. Neck pain, cranial nerve palsy and cerebellar dysfunction can be seen in infratentorial CEDH (9). Our patient complained of headache, nausea, and diplopia, but there was no papilledema or lateral gaze palsy on the neurological examination.

CEDH is more commonly supratentorial. Tatagiba (9) reported that only 1.5% of CEDH were localized in the posterior fossa in their series of 71 cases. In our case, the localization of CEDH was supra- and infratentorial. The CT appearance of CEDH is usually biconvex with a hypodense central area encircled by a hyperdense margin with mass effect (2,4,9). In our case, there also was a biconvex lesion with a hypodense central area and mass effect with minimal compression of the brainstem on cranial CT (Figure 1a, b).

CEDH is almost always associated with a linear skull fracture and the rate is higher than acute epidural hematoma (9). There was a linear skull fracture in the occipital bone in our patient.

Macroscopically, the CEDH appeared as motoroil liquid and there was no cerebrospinal leak and capsule formation.

CEDH has been reported to resolve spontaneously (7,9). Because our patient had some signs and symptoms of intracranial hypertension (nausea, diplopia) and compression of brainstem in the cranial CT, he underwent surgery.

In conclusion, CEDH is uncommon type of EDH and more clearly diagnosed with CT. CEDH is generally treated surgically, but conservative therapy may be employed in suitable cases.

We presented this case because the hematoma was localized supra- and infratentorial, and it appeared in the chronic phase macroscopically and on the CT scan. Treatment is generally surgical in chronic epidural hematoma but conservative management may be employed occasionally. The outcome of our patient following surgery was excellent.

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