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Image Report

Microstructure of embolized capsule of chronic subdural hematoma

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ABSTRACT

Background: Chronic subdural hematomas (cSDHs) are frequent and potentially life-threatening neurosurgical conditions affecting, first of all, elderly. Few treatment options are available ranging from observation to removal thought large craniotomy. However, currently, there is tendency to minimize surgical aggression, especially considering poor general condition of elderly patients. Thus, one of gaining popularity method of neurointerventional treatment of cSDHs is medial meningeal artery (MMA) embolization. To date, large series of cases published describing favorable outcomes of this treatment approach. At the same time, few reports are available that describe microstructural changes in cSDH's capsule after embolization; meanwhile, no exact effect of embolization on pathophysiology of hematoma was determined.

Case Description: Through current paper, we present two cases of cSDH that has previously undergone embolization of MMA, after which cSDHs have been operated through minicraniotomy due to complications after artery embolization. Microstructural changes of hematoma's capsule are described and discussed.

Conclusion: Histological changes in embolized capsule suggest embolization of MMA as a valuable method for treatment of cSDHs.

Keywords: Capsule, Chronic subdural hematoma, Embolization, Microstructure

BACKGROUND

Chronic subdural hematomas (cSDHs) are widespread intracranial lesions predominantly affecting elderly and characterized by intermittent stroke-like presentation with both general symptoms and focal signs. Surgical removal of hematoma in most cases leads to complete resolution of symptoms. During past decades, there has been a paradigm shift from craniotomies with resection of inner and outer cSDH's membranes toward minimally invasive treatment strategies[7] including medial meningeal artery (MMA) embolization.[2] Although large case series of successful cSDH treatment through MMA embolization are currently available,[4,6,11] neither specific mechanism of MMA embolization's impact on cSDH is described, nor its place in treatment algorithm is determined.

Current paper describes two cases of cSDH which was previously treated by MMA embolization with n-butyl-cyanoacrylate (nBCA) followed by minicraniotomy with partial resection of

embolized capsule of cSDH. A thorough histological examination of the obtained capsules was performed.

CASE PRESENTATION

Two patients with cSDHs were treated with MMA embolization, followed by minicraniotomy with partial resection of embolized capsule of cSDH.

Case 1

A 64-year-old male suffering from liver failure admitted to the hospital with recurrent headache and left-side hemiparesis 2 weeks after the mild head injury. Computed tomography (CT) scan revealed large right-sided cSDH. Patient's general condition was poor as a result of prerequisite severe heart, liver, and kidney diseases. At the same time, the volume of cSDH was too high and neurological deterioration was significant for observation. Thereby, it was decided to perform MMA embolization to prevent subsequent hematoma enlargement. Postoperative CT scans revealed X-ray density enhancement of cSDH's capsule [Figure 1].

After embolization, the patient was transferred to ICU to continue treatment of concomitant diseases. Unfortunately, on the 6th day, after embolization occurred a hemorrhagic stroke with large intracerebral hematoma formation in the right parietal lobe. Urgent hematoma evacuation was performed accompanied by decompressive hemicraniectomy with partial resection of cSDH capsule.

Afterward histological examination of cSDH's capsule was performed [Figure 2].

Inner and outer membranes of the capsule were clearly defined. The most amount of embolized vessels were localized in outer one, which was adhere to dura mater.

Subsequent course was complicated by severe heart and kidney failure which led to refractive hypotonia and death on 34th postoperative day.

Case 2

An 82-year-old male suffering from the right side weakness, speech impairment, dizziness, and gait disturbance was hospitalized at the neurosurgical department. First signs of disorder had appeared 2 weeks before admission. Neurologist on duty suspected stroke; thus, MRI scan had been performed that reveled massive left-sided cSDH [Figure 3].

Patient's condition on admission was subcompensated, including Wernicke's aphasia, ataxia, and slight right-sided hemiparesis. Prescription of tranexamic acid was contraindicated due to related atrial fibrillation. Direct surgical treatment is supposed to be associated with high risk of complications. Thus, MMA embolization was chosen as primary treatment option.

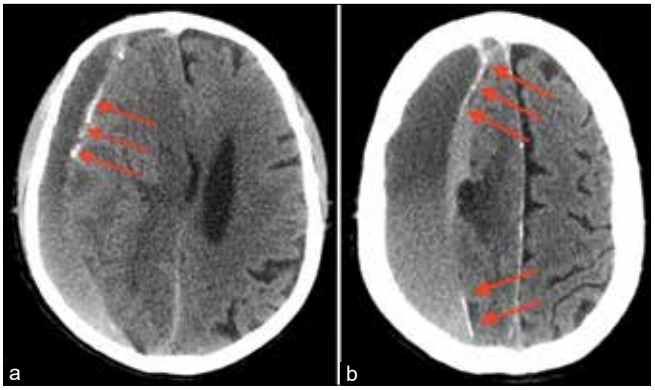

Figure 1: Head computed tomography (CT) scans of patient 1 after MMA embolization. (a) Axial plane, level of lateral ventricles; (b) axial plane, level of centrum semiovale. Note large right-sided cSDH with enhanced capsule (arrows).

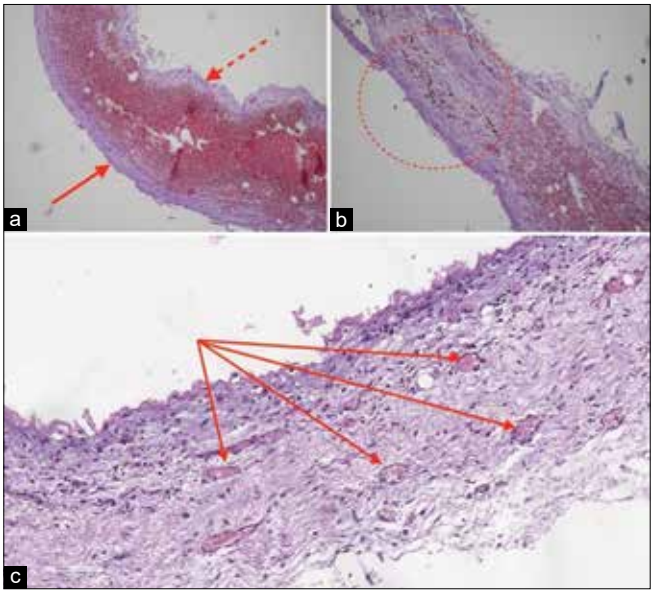

Figure 2: Histological examination of patient's 1 cSDH capsule. (a) Outer (arrow) and inner (dashed arrow) cSDH's capsule membranes, (b) embolized vessels (dashed circle), and (c) embolized vessels of the outer membrane (arrows).

After selective catheterization of MMA, angiography was performed and clearly revealed cSDH's capsule, which was being perfused by small caliber vessels [Figure 4].

Two days postoperatively, the patient was stable. CT scans revealed satisfactory pervasion of cSDH's capsule by nBCA with several prominent clusters [Figure 5].

At the 3rd day of admission, neurological deterioration noted with onset of headache and impaired consciousness (GCS 12). Urgent head CT revealed no dynamics of hematoma's volume

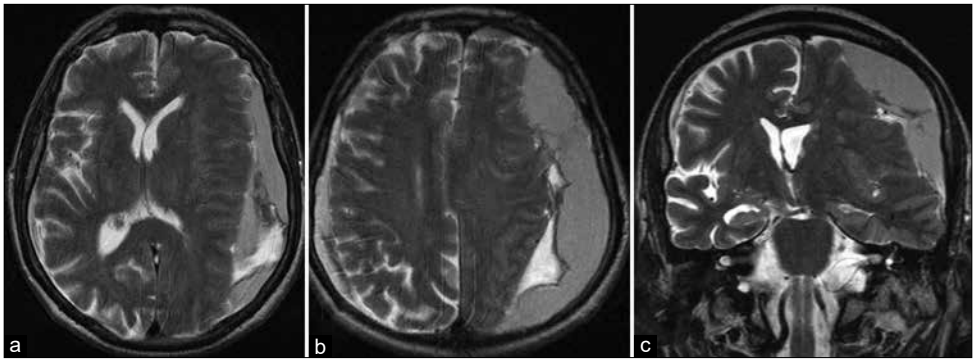

Figure 3: Initial MRI scans of patient 2. (a and b) T2 MRI, axial plane, (c) T2 MRI, coronal plane. Massive cSDH presented with slight compression on the left hemisphere.

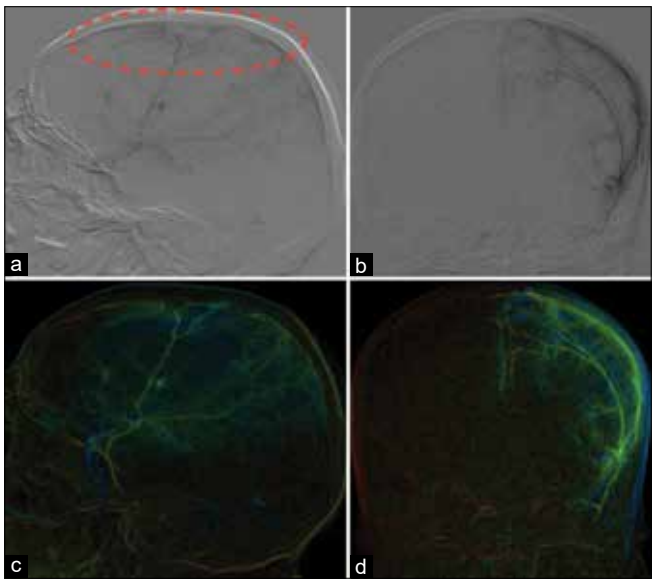

Figure 4: Selective angiography of patient 2. (a) Digital subtraction angiography, catheter in MMA, sagittal plane, capsule of cSDH highlighted by red dashed line, (b) digital subtraction angiography, coronal plane, (c and d) color reconstructions of angiograms, sagittal, and coronal plane, respectively.

but slight increasing of brain edema in adjacent areas. Decompensation of cSDH was proposed and decision made to evacuate hematoma through minicraniotomy. Surgery was uneventful, small piece of hematoma capsule with cluster of nBCA according to the previous CT scans was incised and histologically examined [Figure 6].

Postoperative course of patient 2 was uneventful; he was discharged at postoperative day 7 without any neurological deficit.

DISCUSSION

According to Rand, cSDHs capsule consists of two membranes: the outer (on the dural site) and the inner (on the arachnoid site).[8,13] Our findings confirm this statement.

Despite numerous histological and ultramicroscopic studies, the exact pathomorphology and pathophysiology of CSHs remain controversial. One theory proposes that blood in the thin space between arachnoid and dura, through profound inflammatory response, leads to creation of highly vascularized outer subdural membrane attached to the dura and an inner collagenous membrane up against the brain arachnoid.[1] The pathophysiological consequence of this hypothesis is the statement that *de novo* formed that vasculature of the outer membrane of cSDHs is the main source of internal bleeding and expansion of cSDHs.

Scanning electron microscopy revealed changes in cSDH's fibrocellular structure developing during its evolution.[5] Thin fragile capillaries were observed in hematomas removed between 15 and 40 days after trauma, although, in "older" hematomas, they were accompanied by patent, larger diameter blood vessels.

The other theory possesses that cSDHs are not in fact "subdural," but "intradural."[13] Histological examination revealed thin layer of boundary dural cells on arachnoid membrane without any subdural space between them. On the contrary, there is space between that boundary cells and main dural layers with dense vascular net in (on the) cranial site. According to these findings, hematomas could be originated from damaged intradural vasculature and localized between dural boundary cells and main dura without any true capsule. It allows to divide cSDHs into two types: primary chronic intradural hematoma which originates from intradural vasculature after mild head trauma and secondary cSDH that represents a product of evolution of acute properly subdural hematoma. According to this theory, both our patients suffered from primarily cSDHs which developed after mild head injury without prerequisite acute subdural hematomas.

Lee describing the natural history of cSDHs proposes that cSDHs could be originated from subdural hygromas in patients with suitable premorbid condition – sufficient potential subdural space.[3] Microhemorrhages from fragile new vessels of the neomembrane of unresolved subdural

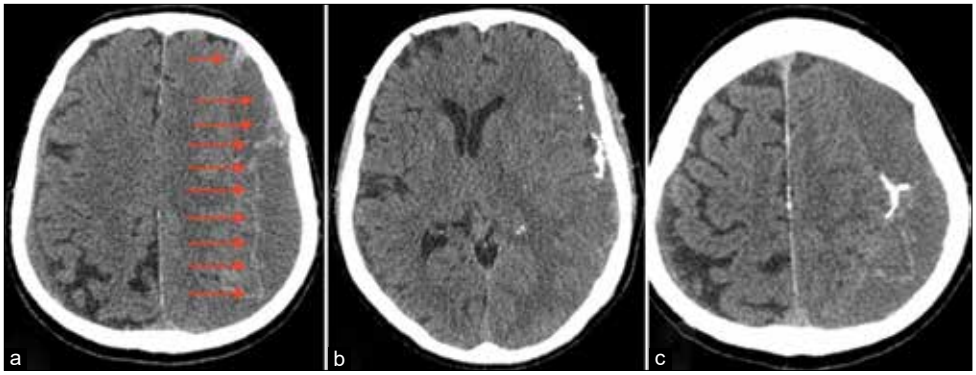

Figure 5: Postoperative CT scans of patient 2, axial planes. (a) Embolized capsule highlighted by red arrows, (b and c) clusters of nBCA inside cSDH's capsule.

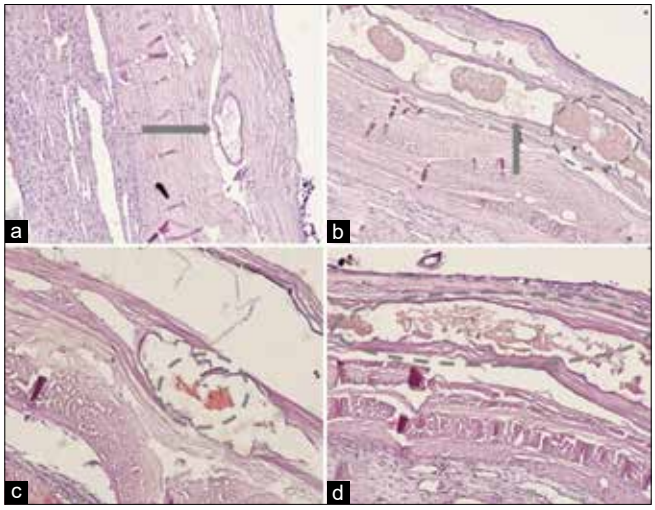

Figure 6: Histological examination of cSDH's capsule stained with hematoxylin and eosin. A – cross section of embolized dural vessel (gray arrow). (b) Longitudinal section of embolized dural vessel, nBCA fragments (gray arrow) and clumped red blood cells (dashed circle). (c and d) nBCA within dural vessels (dashed circles).

hygroma transform it into cSDH. Interesting radiographic predictor of etiology of cSDHs was additionally described – if the cSDH develop on the opposite side of the flat skull side, it would likely been originated from subdural hygroma; otherwise, it is probably originated from acute SDH.

Osmotic theory suggests dependence of cSDH's growth or recurrence on its osmolarity instead of origin.[12] Therefore, increasing of serum osmolarity could potentially enhance resorption of hematoma.

Proposed mechanism of hematoma formation has direct impact on preferable treatment strategy. Shapiro *et al*. demonstrated that anatomical considerations supporting rationality of MMA embolization which is preferable in cSDH.[10] At the same time, almost 9% of cSDHs did not resolve after embolization as a primary treatment method. Saito *et al*. explain this phenomenon by persistence of neoangiogenesis among the inner membrane and septum of cSDH's capsule.[9]

CONCLUSION

Our findings accompanied with published data on pathophysiology of cSDHs that suggest MMA embolization as a valuable treatment method.

Declaration of patient consent

Patients' consent not required as patients' identities were not disclosed or compromised.

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Nil.

Conflicts of interest

There are no conflicts of interest

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