**INTRODUCTION**

Ischemic cerebral infarction is a neurological emergency and it is important to detect it as early as possible to set up the treatment. Non-contrast CT (NCCT) is a first step diagnostic screening method in these cases (6,7). Usually, the diagnosis of cerebral infarction with CT is possible only after 24 hours. Sometimes it is possible to see increased density (brightness) along the course of one of the cerebral vessels. This is not a well known criterium and there are only a few reports about it (1,4). In the literature reviewed, bright MCA incidence was found between 1.9 and 50%. This sign is thought to be due to hyperdense thrombus or embolus. This sign is usually seen within the first 24 hours after the onset of symptoms. Here, we present two cases of bright MCA sign on non-contrast CT detected 2 and 6 hours after the onset of symptoms with follow-up MR scans showing infarction in the MCA territory.

**CASE REPORTS**

Case 1: 41-year-old male patient was admitted because of sudden unconsciousness and motor deficit of his left extremities. Neurologic examination
revealed drowsiness with left hemiparesis of 3/5. There was no sign of meningeal irritation. The medical history was unremarkable except for a transient ischemic attack which had occurred 4 years ago. It was learned that the patient had been reluctant for further investigation upon that incident.

An emergency cranial NCCT scan was performed 6 hours following the onset of symptoms and it revealed a hyperdensity on the first segment of the right MCA (Figure 1). Brain MR scan 24 hours later showed an acute infarct of the right MCA territory (Figure 2). Cerebral MR angiogram showed irregularity of the signal on the first part of the right MCA consistent with thrombus or embolus (Figure 3). Carotid Doppler sonography was insignificant. Routine blood and coagulation tests were within normal limits. Control MR was performed 6 days after administration. It showed hemorrhagic transformation medial to insular cortex (Figure 4).

The patient was heparinized immediately after the first CT scan. He regained consciousness and hemiparesis improved. He was discharged in a markedly improved neurologic state on the 11th day of the incidence.

Case 2: 75 year-old female patient with a history of hypertension and coronary artery disease was admitted with sudden deterioration of consciousness and loss of motor power of her left extremities. On admission, she had a blood pressure of 180/100 mm Hg. She also had nausea and vomiting. On neurologic examination, she was somnolent and had left homonymous hemianopsia, a gaze deviation to the right, left central facial nerve palsy with left hemiplegia.

Cranial NCCT examination performed 2 hours after the onset of symptoms showed hyperdensity of the proximal part of the right MCA (Figure 5). Cranial MR performed next day revealed an acute infarction of the right frontal, temporal and parietal lobes which also included the lentiform nucleus, caudate and capsula interna (Figure 6). A small hemorrhagic transformation of the infarct was also seen (Figure 7). Cerebral MR angiographic study showed a decreased signal intensity on right internal carotid, middle and anterior cerebral arteries. Right MCA territory infarction was obvious in the follow-up CT scan (Figure 8).

She was put on steroids, mannitol and antiaggregant treatment. The neurologic status did not change over time and physiotherapy was started.
DISCUSSION

In patients with acute neurologic signs and symptoms NCCT is proven to be the first step diagnostic tool, mostly performed to detect acute hemorrhages (6,7). Thrombus or an intraluminal blood clot in one of the major cerebral vessels causing infarction may lead to acute
symptoms similar to acute hemorrhage. Cerebral embolism is a cause of cerebral ischemia in more than 30% of the patients (6). Signs of infarction on NCCT are usually demonstrable after 24 hours following the onset of symptoms by means of density decrease (4).

Bright MCA sign is an entity that is introduced to literature in 1980's and only a few of the detected cases are reported (1). In a study of Bastianello et al. (1) 36 stroke patients with CT scans were reviewed and bright MCA was present in 50%. They confirmed that all of the patients having bright MCA sign had good correlation with angiography.

Presman et al. (4), performed NCCT examinations in patients with cerebrovascular occlusive disease and found bright MCA sign with an incidence of 1.9%. All patients eventually had clinical evidence of infarction in the distribution of MCA.

On NCCT, hyperdensity at the course of the MCA representing thrombus or embolus which is an early indicator of infarction may be seen in some patients at the first hours of the incidence (1,4,6-8). We know that blood clots and thrombi have a higher absorption value than moving blood on NCCT scans (6). Thrombus or embolus inside one of major cerebral vessels is demonstrated as having higher attenuation value and is seen brighter than the other side. This sign is most frequently seen in the first segment of the MCA as is the case in our patients (8).

Both of the cases presented here were admitted immediately after the onset of symptoms, so early NCCT examinations were available. In both cases bright MCA sign was seen that was thought to belong to thrombus or an intraluminal blood clot and treatment was started immediately.

As we conceive from the literature, bright MCA sign is detected only in a few number of the patients with acute signs of stroke, but in most of the patients cerebral infarction eventually developed. Infarction at the territory of MCA was demonstrated by MR in the following days in both cases presented here. So, when a bright MCA sign is seen in a patient having acute symptoms, cerebral vessel occlusion is a very likely diagnosis but absence of it should not be interpreted as the absence of infarction in the first 24 hours.

One should be careful when diagnosing thrombus in MCA since calcification which is usually bilateral may be misinterpreted as thrombus. In the study of Bastianello et al. (1), hyperdensity of MCA
on CT resolved after one week in 66% of the patients, excluding the possibility of calcified atherosclerotic plaques. In our study, on follow-up CT scans bright MCA was not seen, consistent with thrombus rather than calcification (1,4).

Cerebral angiography is a definitive diagnostic method to show thrombi or blood clots (2). It is an invasive procedure and is not performed until the patient’s status becomes stable. If the embolic infarction is detected within the first 6 hours, patient may have a chance of thrombolytic therapy (5).

In both of our patients, hemorrhagic transformation was observed on control MR scans. Hemorrhagic transformation is usually demonstrated after the acute period of infarction has passed. It is a sign mostly seen in embolic infarctions after clot lysis takes place and it is more likely to develop if there is wide tissue necrosis (2,3). MR is more sensitive in detecting the hemorrhagic infarctions than CT because of the presence of blood degradation products (2). This sign which is seen in both of our cases, also indicates that infarction is more likely to be embolic.

Bright MCA sign which is usually seen before the development of hypodensity at the infarcted area is an early indication of arterial occlusion and acute cerebral infarction in the first six hours. It is important to diagnose infarction as early as possible so that new therapeutic regimens such as thrombolysis can be performed. It may not always be possible to obtain MR scans which diagnoses infarct at the first 4-8 hours. One should be alert when diagnosing “bright MCA” since it may be confused with calcified MCA.

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