Large striatocapsular infarcts: clinical features and risk factors

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Abstract

We defined large striatocapsular infarcts as subcortical softenings of more than 20 mm in diameter involving the territories of the lateral and medial groups of lenticulostriate arteries. The aim of this study of 56 patients was to compare the clinical features and risk factors of these infarcts with those of cortical and lacunar infarcts. On the whole, our data suggest that both the clinical features and risk factors of large striatocapsular infarcts are similar to those of cortical infarcts, but significantly different from those of lacunar infarcts. The clinical manifestations of large striatocapsular infarcts with a maximum diameter of less than 50 mm may sometimes resemble those of lacunar infarcts because neuropsychological disorders are less frequent; however, our study indicates that, even in these cases, cardioembolic sources and artery-to-artery embolism are significantly more frequent in large striatocapsular than in lacunar infarcts, thus suggesting a different pathogenesis.

Keywords

Large striatocapsular infarcts • Lacunar infarcts • Cortical infarcts • Cerebrovascular diseases

Introduction

Large striatocapsular infarcts (LSCIs) are typically comma-shaped subcortical softenings involving the putamen, the anterior limb of the internal capsule and the caudate nucleus [3–5, 10, 17, 23, 24]. They correspond to the territories of the lateral and medial group of the lenticulostriate, the Heubner, or the anterior choroidal arteries [23]. Small subcortical lacunar infarcts or “lacunae” are often located in the same vascular territory [1, 4, 12–14] but usually have different clinical features and risk factors [5, 10, 17].

The aim of the present study was to investigate the clinical features and the risk factors associated with LSCIs in a relatively large series of patients.

Patients and methods

Among the 1053 patients with first-ever cerebral infarcts consecutively admitted to our department between 1 January 1988 and 31 December 1993, the clinical records of the 56 patients having the clinical and CT features of LSCIs were reviewed.

All of the patients were hospitalized within 60 h of stroke onset and underwent at least one CT scan 1–10 days after the onset. The infarcts were diagnosed by means of CT, their distribution being identified by means of cerebral territory templates [6, 15]. The area of the infarction had to include at least two elements of the striatocapsular area: the head of the caudate plus the internal capsule, or the putamen plus the internal capsule (Figs. 1–3). All of the patients had single large (>20 mm diameter) subcortical infarcts in the territory of the lenticulostriate arteries, sparing the cortex.

The patients all underwent neurological and routine blood examinations, electroencephalography, and Doppler ultrasonography with spectral analysis of the large extracranial vessels. Twenty-four selected patients also underwent angiography, which was only performed in those patients showing clinical recovery in the period shortly after the stroke; the pathophysiological mechanism of the infarct in these patients was considered likely to be related to the extracranial vessels on the basis of clinical indicators (transient ischaemic attacks), or ultrasonographic findings of stenosis or ulcerated plaques. Two-dimensional echography was performed in the patients with a history of heart disease or with possible cardioembolism; the same patients also underwent a 24-h Holter recording.

Language was evaluated by means of the Boston Diagnostic Aphasia Examination (16) and the Token test [9]; other neuropsy-
Psychological tests were performed 20–30 days after admission using the items proposed by Bisiach et al. [2]. Motor function was tested according to the Canadian Neurological Scale [17].

The risk factors for LSCI were defined as the presence of arterial hypertension, cardiac or artery-to-artery embolic disease, carotid stenosis, diabetes mellitus, dislipaemia (high levels of cholesterol or triglyceride), alcohol abuse and smoking. The patients were classified as hypertensive if they had chronic elevated blood pressure (>140/90 mmHg). The Doppler and angiographic findings were classified as occlusion, more than 80% stenosis, 60–80% stenosis, non-haemodynamically significant changes, or no detectable changes. Possible embolic plaques of the carotid arteries were defined by means of echography and angiography (ulcerated plaques). Embolism from the heart was diagnosed in patients with a potential cardiac source, especially when angiography revealed occluded arteries. A number of cardiac diseases were considered as potential embolic sources, including rheumatic and non-rheumatic atrial fibrillation, acute myocardial infarct, ventricular thrombi and aneurysm unrelated to myocardial infarct, non-ischaemic cardiomyopathies, prosthetic cardiac valves, non-bacterial thrombotic endocarditis, and infective endocarditis. The patients were classified as diabetic if they had either a clear history of active diabetes mellitus or if, during their stay in hospital on a starvation diet, their blood glucose levels were repeatedly found to be higher than 110 mg/dl. A fasting cholesterol level of more than 200 mg/dl and triglyceride levels of more than 170 mg/dl were considered abnormal. The subjects whose daily alcohol intake had been greater than 2.5 g/kg for at least 5 years were considered heavy drinkers. Smokers were defined as those currently smoking any number of cigarettes on at least a weekly basis.

The clinical features and vascular risk factors present in the 56 LSCI patients were compared with those of 356 age-matched patients consecutively admitted during the same period, 206 of whom had had their first-ever, single cortico-subcortical infarct (CSI); 150 had had subcortical lacunar infarcts, 18 which were infratentorial locations. The remaining 651 patients were not included in the study because they had multiple infarcts or because they had incomplete anamnestic or clinical data. CSIs were defined as the presence of a cortical syndrome characterized by a unilateral motor or sensory deficit (or both), associated with signs of