Dear Sir,

Insular involvement in middle cerebral artery (MCA) territory stroke is common, whereas an isolated insular stroke is very rare. We noticed isolated insular ischaemia after intravenous thrombolysis in 2 cases and here suggest that this may reflect successful intravenous thrombolysis.

Patients and Methods

Patients with possible stroke presenting to our hospital within 3 h of symptom onset are examined by a neurologist and undergo a non-contrast cerebral CT scan, a perfusion CT and a CT angiogram. Perfusion CT allows the confirmation of hypoperfusion in the MCA territory. Patients are then thrombolysed according to standard NINDS criteria [1]. Follow-up imaging is carried out by CT or MRI.

From January 2005 to September 2006, 42 patients were thrombolysed with recombiant tissue-type plasminogen activator (rt-PA) for an MCA stroke. Thirty-nine patients had a follow-up scan (27 of them by MRI, 12 by CT). The remaining 3 patients died before follow-up. We retrospectively studied this imaging series looking for insular cortex ischaemia.

We also reviewed all MRIs performed over the same period for investigation of possible stroke in patients who were not thrombolysed. These 542 MRI scans acted as a control group.

Results

Considering the 39 thrombolysed patients with an imaging control, 34 patients (87%) had cortico-subcortical infarcts with insular involvement (entire insula in 26, posterior in 6 and anterior in 2). Four patients (10%) had subcortical infarcts. In a single case, there was a cortico-subcortical infarction of the posterior MCA territory without insular involvement. In those cases where the insula was involved, the insular lesion was never distinct from adjacent lesions.

In 2 patients – a 72-year-old man (fig. 1) and a 77-year-old woman (fig. 2) – we observed in the imaging control (both with a post-treatment MRI) a particular pattern consistent with an isolated insular infarct. This finding was not observed in the 542 patients whose MRI scans were available on the stroke registry. This isolated insular infarction was visible on DWI, FLAIR and/or $T_2$ sequences. MRA (TOF) images of the MCA showed a normal, patent artery in each case. Both patients were thrombolysed between 2 and 3 h after onset of symptoms, and both had complete resolution of symptoms at a 3-month review. Before thrombolysis, a CT angiogram showed normal intracranial and cervical vessels for the man and an occlusion of the MCA at the bifurcation with patent leptomeningeal collaterals for the woman.

Discussion

The insula, the smallest lobe of the brain, is located at the base of the sylvian fissure. It consists of 4–6 gyri and is divided by the central insular sulcus into anterior and posterior portions. The insula is at the crossroads of many functional networks and has dense connections with the entire cortex and also the basal ganglia. The arterial supply of the insula arises solely from the MCA, mainly from the M2 segment.

An insular lesion can be responsible for aphasia, gustatory disorders, pseudovesicular syndromes or somatosensory deficits [2–4]. Also, involvement of the insula (and especially on the right side) in an MCA stroke may increase the risk of cardiac arrhythmia and sudden death [5], though recent case series have not supported these findings [2, 3, 6]. fMRI studies suggest that the insula processes emotions (such as empathy) [7].

Involvement of the insular cortex in MCA strokes is very common. Indeed, loss of the insular ribbon on CT scanning is a classical early sign [2]. Insular involvement on MRI scanning was found in 48% of a series of 150 acute MCA strokes [2]. In that series, the entire insula was involved in 50% of cases, the posterior insula alone in 39%, and the anterior insula alone in 11%. Insular infarction was significantly associated with lenticulostrate territory.
Infarction, larger MCA territory strokes and more severe clinical deficits [2]. Thus, insular infarction is a marker of clinical and radiological severity in MCA strokes. Isolated insular infarction, on the other hand, is rare. Cereda et al. [3] identified only 4 cases of isolated insular infarction from their registry of 4,800 acute stroke cases explored by CT or MRI. In a series of 150 non-lacunar MCA territory strokes [2], no isolated insular strokes were identified, even though 48% of the cases had insular involvement. Thus, the fact that we found 2 isolated insular infarcts amongst the 39 thrombolysed patients (5%) in our series is remarkable.

Intravenous thrombolysis with rt-PA significantly decreases the infarcted volume measured by MRI [8]. Small infarcted volume (evaluated by MRI) 3 h after intravenous thrombolysis is correlated with excellent clinical outcomes at 3 months [9]. This is consistent with the clinical course of our patients who had a very small infarcted volume and an excellent clinical outcome.

Although there is now clear evidence for DWI reversibility in the setting of early reperfusion of ischaemic areas [10], it is unlikely that it could explain the MRI pattern of our 2 patients. The presence of residual lesions after a thrombolysed stroke seems to be the rule and there was no normal follow-up imaging in our 39 thrombolysed patients. Completely normal imaging after intravenous thrombolysis is exceptional [10]. As such, normal follow-up scans may suggest that a differential diagnosis of stroke (including TIA) was wrongly thrombolysed.

Successful thrombolysis breaks the clot into many small fragments, and these migrate to downstream branches of the MCA and thus can occlude distal branches. In the case of MCA stroke, when recanalization of the MCA is observed, residual cortical involvement is often limited to the central MCA territory, and particularly the insular area [11]. An isolated insular infarct after rt-PA may be a signature of a successful thrombolysis. The vascular anatomical configuration (arteries arising mostly from the M2 segment of the MCA, absence of collateral blood supply) may make the insular region susceptible to early infarction.

Finally, given the small number of positive findings in this series, the high frequency of isolated insular infarcts may be due to chance alone. Further observations are needed to confirm ours.

References


Announcement

Fondazione Gino Galletti Neuroscience Prize, 2009

European Prize for Young Researchers in the Field of Neurodegenerative Pathologies Leading to Dementia

‘Fondazione Gino Galletti’ announces a competition with a prize of EUR 10,000 to be awarded to a young European Union researcher who is the author of scientific publications on the subject of neurodegenerative pathologies leading to dementia. Competitors must be citizens of Member States of the European Union and aged less than 40 years as of December 31, 2008.

The following documents must be enclosed with the application:
1. CV containing a full list of publications.
2. Copies (paper or electronic) of papers (published or accepted for publication, with proof, during the period January 1, 2007 to December 31, 2008) for evaluation by the committee of judges.

‘Participation in the Fondazione Gino Galletti NeuroPrize 2009’ should be indicated on the envelope or in the e-mail. Deadline for the application is July 24, 2009.

The winner will be informed by the FGG secretary to the address indicated on the application form. The prize will be awarded to the winner by the FGG Chairman or his representative on the occasion of a scientific meeting to be held at the Department of Neurological Sciences of the University of Bologna. At this meeting, the winner shall make a public scientific presentation of the results of his/her research.

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