Clinical-CT Correlations in TIA, RIND, and Strokes with Minimum Residuum

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SUMMARY An approach to the controversy of the physiopathology and classification of ischemic stroke is attempted in this study. The computed tomographies (CT) of 88 patients with transient ischemic attacks (TIA), 46 with reversible ischemic neurologic deficits (RIND) and 70 with ischemic strokes with minimum residuum (SMR) are analysed. The incidence of focal ischemic lesions on CT is 25% in TIA and RIND and 35% in SMR, when the study was performed after the first 24 hours. The incidence of cerebral infarction was much lower when the CT was performed within the first 24 hours after the clinical event. No significant differences in size or location of the infarction were found between the different groups. Deep infarctions were smaller than superficial ones. TIA duration correlated neither with the incidence of CT abnormalities nor with the size of the lesions. No correlation was found between doppler or oculoplethysmography abnormalities, clinical groups and CT findings. In reference to the structural lesions that underlie the clinical syndromes, TIA, RIND and SMR should not be considered as different groups.

THE CLINICAL CONCEPT of transient ischemic attack (TIA) is based mainly on the supposition that it is provoked by focal cerebral ischemia without infarction. This idea has been supported by studies in which no infarctions are found on computed tomographies (CT) performed in TIA cases. But there are other reports that contrariwise show a significant incidence of infarctions on CT of patients with transient neurologic deficits. Although TIAs and completed strokes are usually managed as different conditions, clinical experience shows that completed strokes with minimum residuum are in many aspects similar to long lasting TIAs or to reversible ischemic neurological deficits (RINDs). In order to study the differences and correlations between these groups we analysed the features of CT performed in patients diagnosed of TIA, RIND and completed ischemic stroke with minimum residuum (SMR). The results obtained can be useful in order to achieve a practical classification for the management and study of focal cerebral ischemia.

Material and Methods

Two hundred and four patients with the diagnosis of focal cerebral ischemia have been analysed. All were studied in the Neurology Department within a month after the stroke. The diagnosis was based upon clinical and CT findings. Patients with a history of previous completed strokes were excluded. In all the patients at least one CT was performed after the current neurologic event. Depending on clinical symptoms the ischemia was considered to occur either in the carotid system or in the vertebrobasilar system. The differences in clinical evolution permitted classification in three groups: 1. TIAs. When focal neurological symptoms lasted less than 24 hours. 2. RINDs. When neurological symptoms or signs lasted more than 24 hours but cleared completely before one month. 3. SMR.

When after one month mild neurological signs persisted which did not hamper the basic daily activities of the patient, CTs were performed in a 160 × 160 matrix. Focal ischemic lesions included parenchymatous low-density areas and focal areas of dilatation of a ventricle or a cistern. The area of the low-density lesion was measured in the slice in which the width of the lesion was greatest, giving the results in real brain dimensions. Depending on their location the lesions were considered as superficial (frontal, parietal temporal and occipital lobes), deep (thalamus, caudate, internal capsule, putamen and corona radiata) and cerebellar. Diffuse cerebral atrophy was not taken into consideration. Contrast-enhancement studies are not mentioned because they were rarely performed.

In several cases directional doppler sonography and/or oculopneumoplethysmography were performed. Quoted as pathologic are only those cases with evidence of severe stenosis or occlusion of the ipsilateral internal carotid artery. Angiographic studies were performed in few cases so it seemed not useful to analyse them. The statistical significance of differences was studied by means of Student's t test (for means) and Chi-square test, with Yates correction (for proportions).

Results

Out of 204 cases, 88 were diagnosed as TIAs, 46 as RINDs and 70 as SMR. In the TIA group 67 cases were in the carotid system (amaurosis fugax cases were not included in this series) and 21 in vertebrobasilar system. In the RIND group 41 cases were in the carotid system and 5 in the vertebrobasilar system. In the SMR group 61 cases were in the carotid system and 9 in the vertebrobasilar system. Fifty cases had only one CT performed, in each case during the 24 hours that followed the neurologic event. Two of them showed focal lesions (both SMR). In 154 patients at least one CT was performed after the first 24 hours (with a mean period between the stroke and performance of the CT of 50 days — standard deviation of 55 days —; all the patients being treated during this period by a neurologist). The incidence of CT abnormalities in the differ-
In the management of focal ischemic cerebral disease it has been considered that TIAs constitute a well differentiated group when compared with completed strokes (those with persistent neurological deficit). In the last years an intermediate group, that of RINDs, has been described. 7 In some series, 8-19 strokes with minimum residuum have been considered in the same group of TIAs. In fact, there are qualified authors who state that TIAs, RINDs and SMR should be considered as similar; 11 others consider that they are different groups; 12 others suggest that new studies are needed to identify the groups 13 and, finally, an author states that a new approach to this problem should be attempted. 14

An important question is that it has not yet been possible to establish a physiopathological difference between TIAs and completed strokes. Nevertheless, when the many experimental studies on this subject are reviewed the following conclusions can be pointed out: Transient arterial occlusions (of carotid or middle cerebral arteries) can lead to a cerebral ischemia and permanent occlusions can provoke only transient cerebral ischemia. Whether infarction develops or not depends mainly on two factors: 15-18 the severity of local cerebral blood flow (CBF) decrease (the focal CBF has to be lower than 10 cc/100 gr/min. to produce infarction); and the time hypoperfusion lasts (at least 15 minutes to produce infarction). A little higher CBF (12-18 cc/100 gr/min.) can provoke a reversible failure of neuronal activity (which duration is not still well determined), without infarction. Permanent occlusion can lead to various types of neurological deficit depending mainly on the function of collateral circulation. 19 Not always a greater extent of the infarction correlates with a more severe clinical deficit. 20 There is not a direct correlation between infarction site and clinical disability, except for infarctions located in the internal capsule which correlate with a severe hemiparesia. 21

CBF studies in patients with TIAs show that it is not unusual to find hypoperfusion areas persisting longer than the neurological symptoms, even in cases without evidence of infarction on CT. 22 Only slight differences are found between TIAs and RINDs. 22 Angiographical studies do not show remarkable differences between TIA, RIND and SMR groups, abnormalities of extracranial vessels appearing with quite similar features and incidence in the three groups. 24-26

CT studies have provided a new viewpoint regarding the identification of focal cerebral ischemia groups. Some authors found that no infarction occurred in TIA cases. 1-3 Other reports show that CT evidence of infarction is not rare, with an incidence about 14 to 20%. 4-6, 27-29 Such incidence is clearly higher than the 6% we found in 60 patients (mean age: 60 years). Permanent occlusions can provoke only transient cerebral ischemia. Whether infarction develops or not depends mainly on two factors: 18 the severity of local cerebral ischemia. Whether infarction develops or not depends mainly on two factors: 15-18 the severity of local cerebral ischemia and the time hypoperfusion lasts (at least 15 minutes to produce infarction); and the time hypoperfusion lasts (at least 15 minutes to produce infarction). A little higher CBF (12-18 cc/100 gr/min.) can provoke a reversible failure of neuronal activity (which duration is not still well determined), without infarction. Permanent occlusion can lead to various types of neurological deficit depending mainly on the function of collateral circulation. 19 Not always a greater extent of the infarction correlates with a more severe clinical deficit. 20 There is not a direct correlation between infarction site and clinical disability, except for infarctions located in the internal capsule which correlate with a severe hemiparesia. 21

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one would expect is shown by a recent report in which infarction volume is measured in different types of focal cerebral ischemia. One of the conclusions is that neither by the clinical signs nor by prognosis can be found any difference between patients with normal and abnormal CT in cases of cerebral ischemia with slight clinical deficit.

There is not definite evidence for considering TIAs, RINDs and SMR as well differentiated groups. We do not know if a TIA is provoked by transient ischemia without infarction or it is secondary to an infarction, the mild symptoms being due to a particular location or size. In attempting to study some of these questions we analysed 204 patients with the diagnosis of focal cerebral ischemia studied with CT. Eighty eight were TIAs, 46 RINDs and 70 SMR. It surprised us the difficulty in establishing a neat difference among these groups, because of the subjective and mild symptomatology. The moment of performance of the CT was important: out of 50 cases in which the CT was performed in the first 24 hours only 2 showed a focal lesion, while of 154 cases studied after 24 hours 44 had an abnormal CT. The fact that in several cases a first and a second CT were both normal (even in SMR cases) probably reflects the low resolution of our CT scanner. TIA, RIND and SMR groups did not show any significant difference in the incidence of CT lesions or in the location of these lesions. The frequency of CT abnormalities seemed to be higher in multiple TIAs than in single TIA. No correlation was found between CT lesions and doppler or oculoplethysmography abnormalities. Deep infarctions were significantly smaller than superficial ones. Some of the former could be considered as lacunes. In fact there are reports in which lacunar infarctions are found frequently in patients with TIAs. In series of lacunar infarctions, cases with transient neurological deficits are not rare, this finding being in accordance with the current concepts that are held regarding lacunes. Our principal conclusion is that a clear cut difference in frequency or features of cerebral infarction as visualized on CT in TIAs, RINDs and SMR has not been found. Although some cases of TIA or RIND are probably secondary to a cerebral ischemia without parenchymatous lesion, other cases are provoked by a cerebral infarction which, either because of its small size or its location in areas of low clinical significance is minimized in its neurological symptoms (by mechanisms not yet completely understood).

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Failure of Flunarizine to Improve Cerebral Blood Flow or Neurologic Recovery in a Canine Model of Complete Cerebral Ischemia

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SUMMARY Ten minutes of cerebral ischemia was produced in 12 dogs by temporary ligation of the venae cavae and aorta. After reperfusion the dogs received the calcium entry blocker, flunarizine, 6 μg/kg infused over a ten minute period. Cerebral blood flow (CBF) and metabolism (CMRO₂) were measured pre-ischemia and for 2 h post-ischemia in 6 dogs. At the end of the study, brain biopsies were analyzed for cerebral metabolites. Neurologic recovery was evaluated for up to 48 h post-ischemia in an additional 6 dogs. The results of each study were compared to those previously obtained in untreated animals. The cerebral blood flows (when expressed as a percent of the pre-ischemic control value) of the flunarizine-treated and untreated groups were similar throughout the post-ischemic period. Following an initial hyperemia, the CBF fell to significantly less than the pre-ischemic control values, and remained approximately 26% of control during the final 90 min in both groups. The CMRO₂ was also the same for both groups. Cerebral metabolites were similar although abnormal in both groups. Flunarizine produced pulmonary edema in 5 of 6 dogs studied for neurologic recovery. Four of these dogs died within 12 h and another dog demonstrated severe neurologic damage. None of the untreated dogs developed pulmonary edema, but 6 of 7 dogs evidenced severe neurologic damage or were dead at 48 h. Thus, flunarizine failed to improve either cerebral blood flow or neurologic outcome when given after complete cerebral ischemia in the dog. A cardiodepressive effect of flunarizine might have contributed to the poor neurologic outcome. These results are compared to those obtained following treatment with another calcium entry blocker, nimodipine, in the same animal model.

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