The "Hot Stroke" and Transient Vascular Occlusions

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BOB SANDERS, M.D.,t AND DUNCAN BURDICK, M.D.t

Abstract: The "Hot Stroke" and Transient Vascular Occlusions

Two patients with left middle cerebral artery (MCA) distribution infarctions fulfilled the "hot stroke" criteria on sequential 99m Tc pertechnetate flow and static gamma camera studies. The radioisotopic finding of a paradoxical relative increased flow to the affected hemisphere correlated with serial angiography showing multiple MCA branch occlusions becoming patent with a vascular blush and early venous drainage. The angiographical finding of transient vascular occlusions suggests embolism. Using the present cases together with prior observations, some transient vascular occlusions can be diagnosed by finding an increase in relative radioisotopic flow to the involved hemisphere within a few days to weeks of the infarction. This finding should launch a search for a possible embolic source.

Additional Key Words
- embolism
- gamma camera
- radioisotopic flow
- arterial branch occlusions
- cerebral infarct

The "hot stroke" phenomenon has been defined as the paradoxical transiently increased relative radioisotopic perfusion to an area of cerebral infarction as seen with 99m Tc pertechnetate gamma camera imaging. Other investigators also have seen this finding. The angiographical correlation was a vascular blush and/or early venous drainage of the involved area. Recently, patients serially studied both radioisotopically and angiographically have provided the first demonstration of the full evolution of a "hot stroke." With these cases as illustrative examples, we are able to correlate prior angiographical and radioisotopic observations and more strongly suggest etiological implications.

Case Reports

PATIENT NO. 1
A 62-year-old right-handed office worker slumped to the floor at his job without losing consciousness. On admission (day 1) he was awake and able to follow simple commands but had a dense nonfluent aphasia. Examination revealed a right hemiparesis with maximal involvement of the arm, right extensor planter reflex, right-sided inattention deficit, poor right lateral gaze movements and left ptosis and miosis. There was a history of a myocardial infarction 12 years before with full recovery. Prior hypertension, which had returned to normotension for the last several years without medication, had been recorded.

Laboratory evaluations were all consistent with a cerebral ischemic infarction. Serial 99m Tc pertechnetate radioisotopic flow and static gamma camera studies and angiography documenting his left hemispheric infarction are summarized in table 1 (figs. 1-3).

The patient stabilized and was anticoagulated with warfarin sodium. At six weeks a benign-appearing atherosclerotic plaque was removed from his left carotid bifurcation. A few months later he had a reversible episode of paresis of the left hand. No vascular lesions were demonstrated in the supply of his right hemisphere. He was placed on anticoagulation. His neurological residua included paralysis of the right hand and an expressive aphasia.

PATIENT NO. 2
A 61-year-old white man had an acute right-sided paresis, gaze deviation to the left, and inability to speak three days after aorto-coronary bypass graft surgery. He had a history of a myocardial infarct three years prior with persistent angina, and a traumatic amputation of the right leg. Spinal fluid examination was unremarkable. Serial angiographical and radioisotopic studies revealed a left parietal infarction as well as clinically unsuspected right posterior circulation infarcts. These studies are detailed in table 1 (figs. 4 and 5).

He improved and was started on oral anticoagulants after a week. On transfer to a rehabilitation hospital he was still mute and had a right lower facial paresis with his tongue deviating to the right.

Discussion

Transient middle cerebral artery (MCA) branch occlusions in cerebral infarction patients have been repeatedly verified angiographically by many investigators. It has been stressed that the opportunity to see these transient occlusions is dependent on the timing of the sequential angiograms, i.e., the earlier the angiogram, the greater the frequency of finding occlusions. The movement or disappearance of an occlusive process on subsequent angiograms is considered by some as diagnostic of intracranial emboli. Dalal, Shah and Aiyar, in a hallmark study, were able to document restored middle cerebral cir-
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Patient No. 1: left carotid angiogram, Day 1. There is occlusion of the major part of the MCA circulation. Later films showed partial retrograde filling via anterior cerebral collaterals to the parietal area.

Our patients with angiographical documentation of disappearing MCA proximal branch occlusions over a seven-day to 13-day interval most likely had cerebral embolism. In Patient No. 1, atheromatous ulcer at the common carotid bifurcation at first appeared to be the likely source. In view of his subsequent symptoms, his prior myocardial injury or other central lesions may be implicated. In Patient No. 2 the infarcts may be a neurological complication of the aorto-coronary bypass surgery or also may be related to his antecedent heart disease. The serial radioisotopic flow and static images in Patient No. 1 correlated with the vascular paucity on the initial arteriogram followed by the restored patency, vascular blush and early venous shunting in the infarcted area shown later. In Patient No. 2, the radioisotopic flow study was first done on Day 4 and thus already demonstrated the relative increased flow, while the angiographical sequence was as in Patient No. 1. Both patients had evidence of multiple sites of involvement. Patient No. 1 had a later contralateral symptom, while Patient No. 2 radioisotopically demonstrated left middle cerebral and right posterior circulation infarcts.

From our earlier study and subsequent observations, the relative increased flow to the infarcted side usually does not appear at the clinical onset. Rather, the relative increased flow may be seen as early as the second day and then persists for about two weeks. This is consistent with the immediate

FIGURE 1

Patient No. 1: left carotid angiogram, Day 13, subtraction view. The arrows outline the area of vascular blush seen on this late arterial (three-second) film. Most of the previously occluded middle cerebral branches are now patent. The early regional venous drainage was best seen on the four-second film.

FIGURE 2

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"HOT STROKE" AND TRANSIENT VASCULAR OCCLUSIONS

Angiographic and Radioisotopic Correlation

<table>
<thead>
<tr>
<th>Patient No. 1</th>
<th>Angiogram</th>
<th>Radioisotope study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1:</td>
<td>Arch and bilateral carotid angiograms revealed multiple occluded L MCA branches (fig. 1). There was retrograde anterior cerebral collateral circulation to the parietal area. Atherosclerosis at the L CCA bifurcation with a question of ulceration was noted.</td>
<td>Day 2: Slight decreased L flow. Normal static images.</td>
</tr>
<tr>
<td>Day 13:</td>
<td>L carotid angiogram revealed that most of previously occluded MCA branches were now patent. There was a vascular blush in the MCA suprasylvian distribution (fig. 2), and early parietal and deep venous drainage, i.e., a &quot;luxury perfusion.&quot; Retrograde collaterals were no longer present. A definite L CCA bifurcation ulcer was seen.</td>
<td>Day 8: Increased L flow. Moderate static uptake L frontoparietal and anterior frontal areas.</td>
</tr>
<tr>
<td></td>
<td>Day 13: Question of ulceration was noted.</td>
<td>Day 13: Increased L flow (fig. 3). Marked static uptake L.</td>
</tr>
<tr>
<td></td>
<td>Day 16: Regression of increased L flow laterally with now decreased L flow mediially (mixed pattern). Marked static uptake L.</td>
<td>Day 16: Increased L flow (fig. 3).</td>
</tr>
<tr>
<td>Day 21:</td>
<td>Decreased L flow. Moderate static uptake L frontoparietal.</td>
<td>8 mo: Decreased L flow, slight static uptake L frontoparietal.</td>
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<td></td>
<td>Day 7: L carotid angiogram revealed a filling defect in the proximal posterior MCA trunk (fig. 4). There was a paucity of parietal branches with anterior cerebral retrograde filling collaterals to this area.</td>
<td>Day 10: Increased L flow. Marked L parietal, R occipital and R posterior fossa static uptake.</td>
</tr>
<tr>
<td></td>
<td>Day 13: Regression of increased L flow laterally with now decreased L flow mediially (mixed pattern). Marked static uptake L.</td>
<td>Day 14: Slightly increased L flow.</td>
</tr>
<tr>
<td></td>
<td>6 wk: Decreased L flow. Trace L parietal uptake residua only.</td>
<td>Static images as Day 10, i.e., marked L parietal and R posterior circulation static uptakes.</td>
</tr>
<tr>
<td></td>
<td>Day 21: Decreased L flow. Moderate static uptake L frontoparietal.</td>
<td>8 mo: Decreased L flow, slight static uptake L frontoparietal.</td>
</tr>
</tbody>
</table>

L = left, R = right, MCA = middle cerebral artery, CCA = common carotid artery, mo = month, wk = week.

Angiographical finding of proximal middle cerebral branch occlusions which then reopen and show luxury perfusion on subsequent angiography. Pathologically, this fits the hypothesis that embolus lysis or distal migration explains cerebral infarction without observed occluded vessels, as proposed by Fisher and Adams. Furthermore, Lhermitte, Gautier and Derouesné in a pathological survey postulated that embolism is a major cause of MCA territory infarction. This was particularly true in the absence of middle cerebral or internal carotid artery occlusion at postmortem examination. However, it should be noted that embolus is still only an inferential diagnosis when transient vascular occlusions are either seen.

**Figure 3**

*Patient No. 1: vertex 99m Tc pertechnetate flow study. Day 13. In this sequence of successive four-second intervals, there is a prominent relatively increased left-sided perfusion (the dot is on the patient's right)*

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Patient No. 2: left carotid angiogram, Day 1. There is a filling defect (arrow) in the proximal portion of the posterior trunk of the MCA with branch occlusions. Later films showed partial retrograde filling via anterior cerebral collaterals to the parietal area.

angiographically or suspected pathologically. There may be other as yet undefined processes that cause temporary obstruction, as such as possibly a non-atherosclerotic thrombosis with subsequent lysis.

Serial radioisotopic flow and static gamma camera studies are a nontraumatic method of studying cerebral infarction. Those patients who incur an increase in the relative flow to the infarcted hemisphere over a few days to weeks are believed to have had transient vascular obstructions. This "hot stroke" evolution should then launch a search for the source of this process, especially seeking a source of emboli. Unfortunately, there will be a significant group of infarct patients in whom the cause of obstruction remains "unknown" using our current investigative techniques. This should serve to stimulate further development of methods to find the etiology of these transient vascular occlusions.

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References
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