Short Communications:

Unilateral Neurogenic Pruritus Following Stroke

E. WAYNE MASSEY, M.D.

PRURITUS CONTRALATERAL TO capsular infarction has been described in a single patient. Since cutaneous receptors are similar to those moderating pain, it is thought that central projections from the cord to cerebrum are the same for pruritus and pain. We describe our experience with pruritus following stroke.

Case Material

Nine patients with pruritus following stroke have been seen at Duke University Medical Center Rehabilitation Unit (table 1). Six were male and three female. Four were transferred from another facility and five had been in our hospital since occurrence of the stroke. During rehabilitation, from 3-6 weeks post stroke, these patients developed excoriations of the involved side. Their rehabilitation progress was not different from other patients.

In five of our patients lesions involved the deep structures including thalamus and subcortical regions contralateral to the pruritic side. Motor and sensory involvement was variable. Computerized tomography in each patient defined the anatomic location (table 1).

The lesions were secondary cutaneous lesions due to scratching. Underlying medical disorders, which should be considered in pruritus without a primary cutaneous eruption, were excluded by history, physical examination and appropriate laboratory evaluation. Possible medications causing itching were discontinued. A seizure focus as the cause of paroxysmal pruritus was not identified on electroencephalogram. No lymphoma, renal failure, jaundice, anemia, or infectious cause could be found. All patients have been followed for at least three months.

Treatment included glycerine and olive oil local therapy in some and required Amitriptyline or Carbamazepine in five patients. Pruritus lessened in all patients over time and excoriations resolved.

D.S., C 24373, was a 67 year old white female who developed a deep neck infection with subsequent erosion of the superior right internal carotid artery requiring surgical intervention. Post-operatively she had a left hemiplegia and hemianesthesia. Head CT scan (fig. 1) revealed an infarct in the distribution of the right middle cerebral artery with extension into the internal capsule. She was transferred to rehabilitation on day sixteen and shortly thereafter she began complaining of pruritus. This occurred bilaterally but it was much more marked on the left. No definite etiology was found and she was treated with Benadryl, Neutrogena, and oil/lime water emulsion. Subsequently Amitriptyline was added at night (50 mg q.h.s.). At discharge, after eight weeks of rehabilitation the pruritus was minimal.

J.O., M 59 370, was a 76 year old right handed male who had acute onset of a left hemiparesis on 4/20/82. He had a history of atherosclerotic cardiovascular disease, hypertension, peripheral vascular disease and vascular claudication. On admission he had no movement on the left side. Sensory examination showed decreased pin prick on the left face and body which was normal on the right. CT scan (fig. 2) showed a focal low density in the right middle cerebral artery distribution with extension into the deep structures. After eight days he was transferred to the rehabilitation unit where he stayed for six weeks. He developed a nonspecific dermatitis that was localized primarily over the extensor surfaces of the left forearm and leg (fig. 3). No etiology could be found and a skin biopsy was consistent with an urticarial reaction with large numbers of eosinopils scattered throughout the superficial dermis. No specific etiology could be found and local and systemic therapy was instituted.

Discussion

King et al. have described one case in a woman with a basilar artery aneurysm with CT demonstration of infarction in the posterior limb of the right internal

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<th>Case #</th>
<th>R/L</th>
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<th>Anatomy by CT scan</th>
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<tr>
<td>1. M36</td>
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<td>5. M68</td>
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<td>9. F62</td>
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<td>neglect (?)</td>
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IC = internal capsule; MCA = middle cerebral artery.

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capsule and frontotemporal area contralateral to the pruritus. Symptoms began several weeks after discharge. This probably represents the Dejerine-Roussy Syndrome, transient hemiparesis, hemianesthesia, spontaneous pain and "burning" paresthesias, due to a lesion in the ventral posterior thalamus and internal capsule.

Yamanoto, et al. have described paroxysmal itching in multiple sclerosis in three patients. Each had presumed spinal cord involvement but double dose head computerized tomography was not reported so periventricular lesions are not excluded.

In addition to spinal cord causes, localized peripheral nerve or root involvement can cause pruritus. Pruritus is encountered in a multitude of skin disorders, including those most often considered psychosomatic. Pruritus may derive from a variety of general medical conditions and may be the first clinical manifestation thereof. Slow conducting C fibers at the dermoepidermal junction and in the papillary dermis are probably involved; they are known to be activated by histamine, by endopeptidases, and probably by prostaglandin E. Cormia demonstrated the enlargement of a standardized histamine response and a prolongation of itching under induced stress.

The absence of an organic diagnosis, coupled with the detection on interview of significant emotional conflict, is the customary means of arriving at a psychiatric cause. Sometimes physical disorders, either specifically cutaneous (scabies, pediculosis, allergy, etc.) or general systemic (hyperthyroidism, lymphoma, obstructive jaundice, polycythemia vera, uremia) may coincide with compelling evidence of emotional conflict. Treatment may therefore be complex and difficult.

Conventional antipruritus treatment normally includes oral hydroxyzine hydrochloride, topical antipruritics, but also menthol and phenol preparations, topical steroid therapy, and various measures to effect skin cooling.

References
3. Chapman LF, Goodell H. Wolff NG: Structures and processes in-


Elimination of Subjective Bruit With Compression of Temporal Artery: New Physical Sign Indicative of Internal Carotid Artery Occlusion

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SUMMARY A patient is presented with a right central retinal artery occlusion and subjective bruit over right ear. Digital compression of the right temporal artery eliminated the bruit and release reproduced the bruit. Carotid arteriogram showed bilateral internal carotid occlusion with a prominent right temporal artery.

This sign reflects compensatory hyper-perfusion of the external carotid artery due to occlusion of ipsilateral internal carotid artery.

MULTIPLE CLINICAL SIGNS have been described in which internal carotid artery occlusion causes augmented blood flow through the external carotid system. Recognition of a dilation of conjunctival and episcleral blood vessels, neovascular glaucoma, newly formed circular vessels at the base of each iris indicate perfusion of intracranial vessels via the ophthalmic artery in a retrograde fashion from branches of the external carotid artery. Auscultation and palpation may reveal a carotid bruit which diminishes with pressure over the ipsilateral facial and temporal arteries. Ocular bruises over either eye, pulsation of the angular artery, or the brow artery, sudden disappearance of a transmitted cardiac sound, all may suggest occlusion of the ipsilateral internal carotid artery.

Case Report

The following case illustrates another physical sign indicative of occlusion of the internal carotid artery with augmented external carotid perfusion.

A 53-year-old man was seen with a history of the sudden onset of right eye blindness accompanied by minimal retro-orbital pain of 2 months duration. He became aware of a pulsating "swishing" sound over the right ear, most prominent at night. An episode of transient left hemiparesis had occurred 4 years earlier. Examination showed dilated right-sided episcleral arteries, the right pupil was 4 mm and minimally reactive, the left pupil 3 mm and reactive. An afferent pupillary defect was present on the right. There was marked attenuation of the retinal vasculature with a pale disc and a minimal retinal fiber layer consistent with prior central retinal artery occlusion. The bruit was accentuated when the examiner’s finger was inserted into the right external ear canal, and firm pressure over the right temporal artery anterior to the ear eliminated the subjective bruit. Release of this pressure led to a return of the bruit. With a modification of the stethoscope enabling the examiner and the patient to hear simultaneously, confirmed a bruit which disappeared with digital pressure over the artery. Carotid arteriograms detected bilateral internal carotid obstruction prominent right temporal artery perfusion and retrograde filling from the external carotid artery of the intracranial circulation to the homolateral cerebral cortex.

Discussion

A clinical distinction between a carotid ulcerative lesion and carotid occlusion causing cerebral or ocular symptoms is difficult. Management of an ulcerative lesion without obstruction is considered by some to require carotid endarterectomy. In the presence of complete occlusion, either medical therapy or extracranial-intracranial bypass may be under consideration. Although arteriography may be required in either case, recognition of an internal carotid artery occlusion on physical examination may be very helpful.
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