Cerebral Perfusion Deficit Masked by Paget's Disease of Skull

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Prominent extracerebral perfusion on an emission angiogram of the head in a patient with Paget's disease of the skull masked an underlying intracerebral perfusion deficiency, due to occlusion of the left middle cerebral artery.

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The diagnostic difficulties arising from brain imaging of patients with concurrent intracranial and extracranial disease are well recognized. Virtually any pathologic involvement of the scalp or calvaria—post-traumatic, postsurgical, inflammatory, neoplastic or metabolic—could result in an abnormal activity pattern on brain imaging.

The most common problem encountered is differentiation between acute traumatic changes to scalp or skull, and intracranial disease, especially subdural hematoma.

Previous reports have alluded to abnormal brain scans in Paget's disease of the skull, as well as in other bone diseases (*1–3*). In some cases, additional information from skull radiographs, radionuclide bone imaging, emission angiography of the head, and comparison of immediate and delayed brain images, have helped to differentiate intracranial disease from Paget's disease (*2,4,5*).

In our patient, the presence of an acute occlusion of the left middle cerebral artery was masked in an emission angiogram by early and persistent increased extracerebral radionuclide perfusion due to Paget's disease of the skull.

CASE REPORT

A 61-year-old man was admitted to the hospital when he was found confused after a minor auto accident.

Physical examination revealed a right hemiparesis, aphasia, and right upper-motor-neuron signs with abnormal reflexes. Also noted were atrial fibrillation and a holosystolic murmur.

Past history included long-standing Paget's disease with extensive involvement of the pelvis and skull. Possible diagnoses were subdural hematoma secondary to recent auto accident, or embolus of the left middle cerebral artery secondary to mitral valve disease and atrial fibrillation.

Two days after admission, dynamic and static radionuclide brain images were obtained. Tc-99m DTPA (20 mCi) was injected intravenously with standard bolus technique. Arm-to-neck time was 16 sec, with tailing of the bolus apparently due to atrial fibrillation and borderline congestive heart failure. Although increased intracranial pressure was suggested by increased artery-to-vein circulation time, no significant asymmetry in cerebral perfusion was seen (Fig. 1). Static images were interpreted as consistent with Paget's disease, as demonstrated on skull radiographs.

A concomitant left cerebral contrast arteriogram demonstrated occlusion of the horizontal portion of the left middle cerebral artery (Fig. 2, black arrow). The middle meningeal artery, superficial temporal artery, and other branches of the external carotid were prominent, with increased vascularity to the thickened calvaria (Fig. 2, open arrows).

In retrospect, the emission angiogram shows an area of diminished activity between the circle of Willis and the genu of the left MCA (Fig. 3). On closer inspection, the activity over the left cerebral

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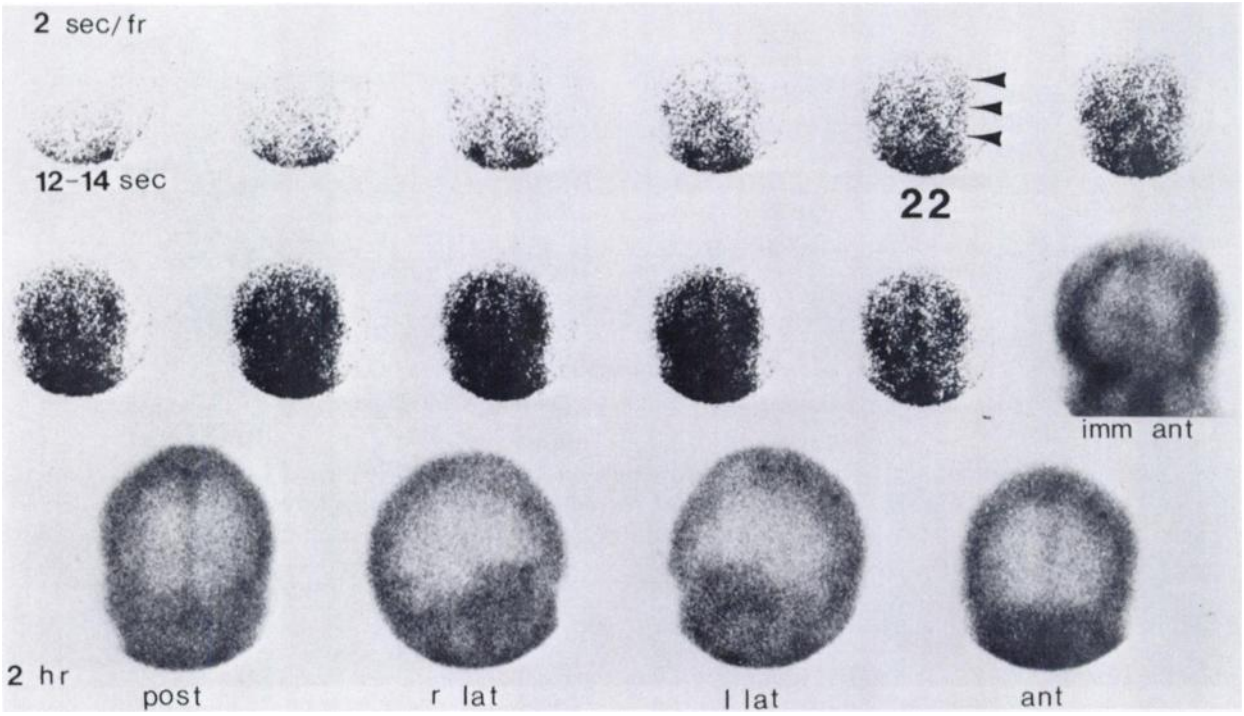

FIG. 1. Anterior cerebral perfusion and static images. Note activity arising directly from left external carotid branches (black arrows, "22" frame). The diffusely thickened calvarial activity on static images is due to Paget's disease.

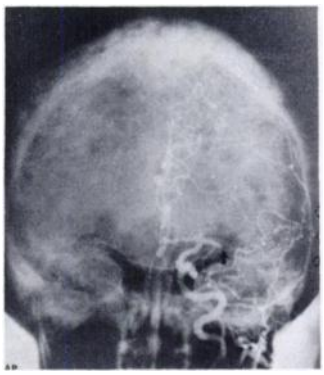

FIG. 2. Selected anteroposterior radiograph from left cerebral angiogram. Occluded left MCA (black arrow). Prominent left external carotid branches (open arrows).

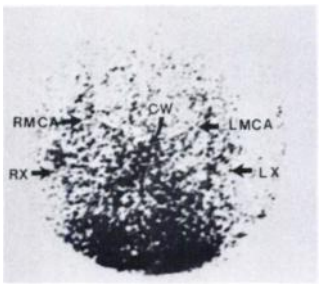

FIG. 3. Enlarged 18–20-sec frame from Fig. 1, demonstrating decreased perfusion of horizontal portion of left middle cerebral artery (LMCA). LX = left external carotid branches; CW = circle of Willis.

convexity, which had simulated left MCA distribution, is seen to arise from external carotid branches (Fig. 1, black arrows).

A repeat radionuclide brain study two weeks later showed no change in the perfusion pattern. Static images, however, showed extensive uptake indicating cerebral infarct in the distribution of the left MCA (Fig. 4).

DISCUSSION

Studies with bone-seeking agents in patients with Paget's disease of the bone show locally increased blood flow to be a factor in the abnormal uptake in pagetic bone (*6,7*). With bone-scanning agents, increased perfusion is not the predominant cause of abnormal uptake (*8*). With brain-imaging agents, the highly vascular nature of bone in Paget's disease is the probable cause of abnormal accumulation. The increased uptake is often apparent in dynamic as well as in static brain images in these patients.

Skull radiographs will usually alert the physician to this pitfall in the diagnosis of possible intracranial disease from radionuclide brain studies. Occasionally, early Paget's disease with normal skull radiographs has caused confusion with subdural hematoma on static brain images (*1*).

In our case, Paget's disease of the skull was well documented; therefore, the abnormal static images

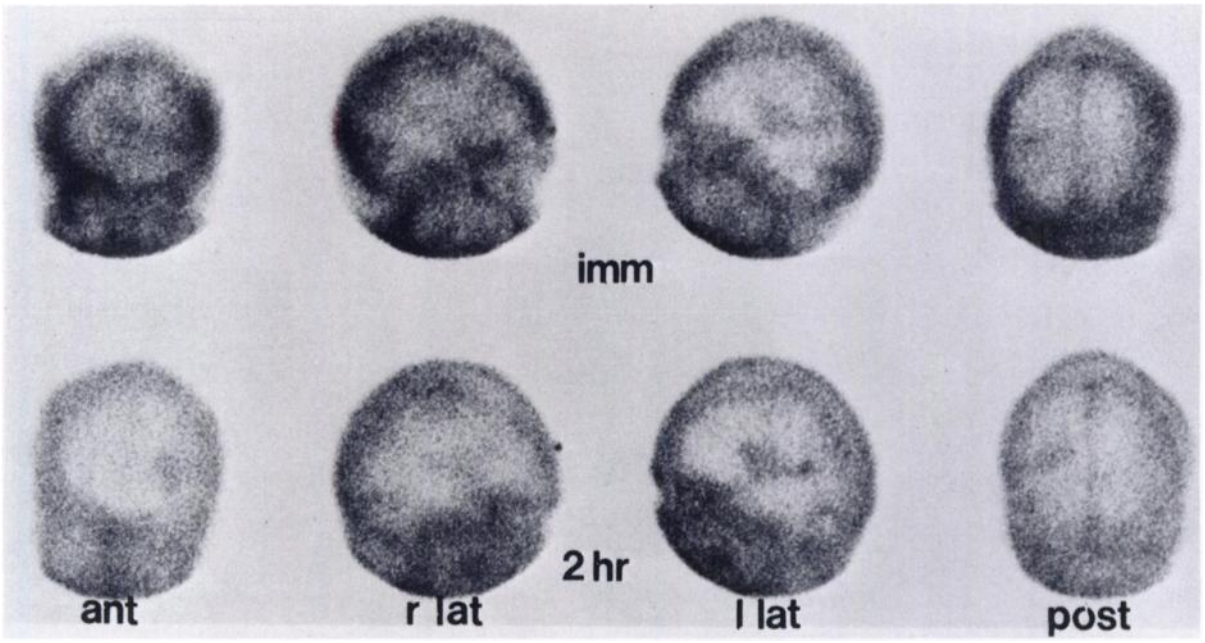

FIG. 4. Repeat static brain images 2 wk later. Cerebral infarct is seen in left MCA distribution.

were not interpreted as subdural hematoma. The early and persistent increased extracerebral activity on the emission angiogram, however, masked a cerebral perfusion deficiency.

REFERENCES

1. PREIMESBERGER KF, LOKEN MK, SHAFER RB: Abnormal brain scan in Paget's disease of bone—confusion with subdural hematoma. *J Nucl Med* 15: 880–883, 1974

2. FITZER PM: Nuclide angiography in paget's disease of the skull: Case report. *J Nucl Med* 16: 619–621, 1975

3. KIEFFER SA, LOKEN MK: Positive "brain" scans in fibrous dysplasia and other lesions of the skull. *AM J Roentgenol Radium Ther Nucl Med* 106: 731–738, 1969

4. BERNSTEIN JR, HOFFER PB: Use of the delayed brain scan in differentiating calvarial from cerebral lesions. *J Nucl Med* 15: 681–684, 1974

5. SPENCER RP, APONTE LJ: Coupling of results of cerebral blood flow and static brain studies. *J Nucl Med* 15: 32–33, 1974

6. GOLDMAN AB, BRAUNSTEIN P, WILKINSON D, et al: Radionuclide uptake studies of bone: A quantitative method of evaluating the response of patients with Paget's disease of diphosphonate therapy. *Radiology* 117: 365–369, 1975

7. SERAFINI A, ALTMAN R, SANKEY R, et al: Paget's disease: A method of evaluation of response to therapy using the anger scintillation camera on-line to a computer. *J Nucl Med* 14: 449, 1973

8. ROSENTHALL L, KAYE M: Technetium-99m-pyrophosphate kinetics and imaging in metabolic bone disease. *J Nucl Med* 16: 33–39, 1975

9. FITZER PM: Radionuclide angiography—Brain and bone imaging in craniofacial fibrous dysplasia (CFD): Case Report. *J Nucl Med* 18: 709–712, 1977