STEROID EFFECT ON THE BRAIN SCAN IN A PATIENT WITH CEREBRAL METASTASES

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A case of cerebral metastases is presented in which the initial brain scan did not demonstrate the lesion because steroid medication had been started 18 hr earlier. The lesion was seen after the patient had been off medication for several days. Therefore, brain scans should be performed prior to starting or following the removal of steroid medication for several days in order to avoid false-negative results secondary to steroid effects.

In the preoperative management of central nervous system lesions, steroid preparations are occasionally given in critical cases especially if cerebral edema is assumed to be present. The clinical benefits are well known but the effect on diagnostic evaluation of a particular case may be overlooked. This case illustrates the difficulty that may be encountered.

CASE REPORT

A 47-year-old man was admitted to the hospital with a 2-day history of left-sided focal seizures. The physical exam revealed an emaciated black man who complained of severe headaches. There was a hard mass in the right supraclavicular area. The neurologic exam showed that both optic discs were blurred and there was decreased sensation and motor strength on the left side. A chest x-ray film revealed an irregular 3-cm density in the lateral portion of the right midlung field and a lytic lesion in the right eighth rib posteriorly. The day after admission, 4 mg of dexamethasone was started orally every 6 hr. Eighteen hours later, a brain scan with $^{99m}$Tc-DTPA (Fig. 1) was done in which vague patchy activity was seen but not a discrete abnormality. An electroencephalogram done 6 days after the scan localized an irritative focus in the right posterior temporal and occipital areas. The day following the EEG, dexamethasone was discontinued and a right cerebral arteriogram demonstrating stretching of posterior temporal and parietal arteries consistent with a mass lesion was done. A week after the angiogram, a second brain scan was done (Fig. 2). The angiogram should have no effect on the scan after this period of time (1). Steroid medication was immediately resumed. A third scan 5 days later (Fig. 3) showed the lesion to be less prominent but still visible. A biopsy of a cervical lymph node was interpreted as an undifferentiated

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large-cell carcinoma. The patient died 1 month later but an autopsy was not authorized.

**DISCUSSION**

The mechanism whereby steroids assist in the management of patients with cerebral tumors and edema is unknown except to say that the benefits seen clinically are most likely the result of a reduction of cerebral inflammation and edema associated with a general anti-inflammatory effect of steroid therapy. The onset of action is rapid, and the evidence of improvement, both objective and subjective, may be noted within 4 hr (2). A scan done soon after the start of steroid therapy may thus be affected. Moreover, dexamethasone was shown to have an effect in cats when given before or after the production of an experimental “standard” subcortical lesion. The result was a decrease in the transudation of radioiodinated albumin across blood vessels adjacent to the lesion. Up to 4 days, there was a difference in isotope concentration between cats treated with dexamethasone and those not treated. Thereafter, no significant difference was noted (3). This may account for the partially suppressed activity seen on the scan 5 days after steroids were readministered. Previous work has shown that edematous brain tissue may have a higher uptake of radioactivity than normal brain tissue (4). In addition, tumor localization with pertechnetate may often be because of its combination with protein (5). Nonlocalization of pertechnetate in tumors in patients treated with steroids may be related to the effect of steroids on one or both mechanisms. We postulate similar mechanisms for the localization of 99mTc-DTPA in the area of the tumor and, depending on the relative degree of protein binding, one mechanism may predominate. That is to say, the radioactive material bound to protein may be carried across damaged vessels or may passively diffuse across vessel epithelial cells with edema fluid that collects around the tumor because of changes in osmotic pressure in extracellular brain fluid.

Normally tight junctions are considered to be impermeable to molecules of molecular weight greater than 1,800 and selective permeability is a function of the intact endothelial cell wall (6). Steroid preparations such as dexamethasone probably do not physically block protein transfer into the extracellular fluid but may affect repair mechanisms or active transport from extracellular fluid to the blood of the endothelial cell wall. A previous report of false-negative scans in 3,600 cases does not suggest steroid effect as a reason for false-negative results, the reason for some false-negative scans being unknown (7).

**REFERENCES**