Todd's Paralysis: A Cerebrovascular Phenomenon?

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Abstract: Todd's Paralysis: A Cerebrovascular Phenomenon?

Postictal transient focal neurological deficits, i.e., Todd's paralysis, at times are correlated with early veins and/or vascular stain angiographically. Radionuclide gamma camera images show that they also may be accompanied by a relative flow hyperperfusion and a cortical rim static image uptake. Using these observations some portion of Todd's paralysis may be explained as the result of focal epileptic discharges that lead to local vasomotor and/or metabolic changes. The functional arterial venous shunting that results could contribute to cortical ischemia and the subsequent clinical deficits.

Additional Key Words
- flow hyperperfusion
- gamma camera
- postictal angiography
- cortical ischemia
- early veins
- radioisotope

Jackson, writing in 1876 to explain the temporary paralysis following “strong epileptic discharges,” noted that Robertson in 1869 had written: “But I am inclined to think that the late Dr. Todd was correct in supposing that severe and protracted convulsions may themselves in some instances be causative of palsy of a few hours’ or days’ duration, through simply the exhausting influence exerted on the cells of the central ganglia without much, if any, appreciable change of tissue. This explanation is especially applicable to some cases of hemiplegia following epilepsy in which the paralysis passes away in a few days.”

Clinical observations of patients with focal seizures and prolonged but reversible postictal deficits coupled with angiographical and radioisotopic correlation may offer some additional pathophysiological explanations of Todd’s paralysis.

Patients with a benign illness characterized by repetitive focal motor seizures and prolonged Todd’s paralysis have been studied recently. At the peak of their illness, angiography had demonstrated early venous drainage and/or vascular stain, while intravenous radiopertechnetate gamma camera images showed relative hyperperfusion with a mild static cortical rim uptake. These angiographical and radioisotopic findings normalized as the patients improved clinically.

In focal seizure disorders, arterial venous shunting has been visually observed; “red venous blood” was reported during seizure discharges. This functional bypass of the capillary circulation could contribute to the cortical ischemia that was also observed, and the alteration of the blood-brain barrier could explain the static radioisotopic cortical rim uptake. The early veins and/or stain observed angiographically and the relative hyperperfusion noted on radioisotopic studies fit Lassen’s luxury perfusion hypothesis during which blood is shunted around affected tissue. Presumably this shunting is due to a local metabolic acidosis which also has been shown to occur in seizures.

All of these abnormalities are usually correlated with a transient disruption of function, i.e., Todd’s paralysis. However, the static image uptake at the cortical rim may be prolonged and present after the flow

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abnormality has normalized. The fact that it diminishes in intensity only over several weeks’ time can be presumptive evidence of cerebral infarction.\textsuperscript{12}

The following case report is illustrative.

**Case Report**

Three years after severe left brain trauma, a 19-year-old ranch worker was admitted because of several days’ duration of right-sided focal and generalized seizures. A marked

*FIGURE 3*

Anteroposterior radiopertechnetate gamma camera flow study. Note the marked left-sided relative hyperperfusion early in patient’s illness. The patient’s left side is to the reader’s right.

*FIGURE 3*

Radiopertechnetate gamma camera static studies showing the left-sided cortical rim uptake which gradually diminished over two months. Clockwise: anteroposterior, left lateral, right lateral and posteroanterior views. The dot represents the patient’s right side.
expressive aphasia and right-sided apraxia resulted, which took six weeks to improve. Electroencephalography revealed frequent epileptiform discharges over the left hemisphere that persisted during his first few hospital weeks.

Angiography on admission showed a prominent vascular gyral stain with marked early venous drainage (fig. 1). Serial radiopertechnetate gamma camera images showed initially a relative left brain flow hyperperfusion (fig. 2), resolving to a hypoperfusion after two months. An initial left cortical rim static uptake (fig. 3) diminished in intensity over the next six weeks.

At nine-month follow-up, repeat carotid angiography revealed only a dilated left ventricular system while the radioisotope study showed persistent relative decreased left-sided flow. Clinically the patient had a moderate expressive aphasia and organic mental deficits.

Summary
Using our angiographical, radioisotopic observations, Todd's paralysis may be considered the result of something more than "exhaustion" per se. It may be postulated that focal epileptic discharges lead to local vasomotor and/or metabolic changes and that these are associated with functional arterial venous shunting and cortical ischemia. By definition, the postictal disability is transient. Rarely, a permanent deficit ensues, probably dependent on the total amount of ischemia and the state of the bypassed cerebral tissue.

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References

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