

# Computed Tomographic Changes of Hypertensive Encephalopathy

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Computed tomographic (CT) scans were evaluated in 11 patients with acute hypertensive encephalopathy. Hypertensive encephalopathy is characterized by an acute, severe rise in blood pressure associated with headache, nausea, vomiting, altered mental status, and focal neurologic deficits, and rapid improvement after control of blood pressure. The systolic blood pressure range is 200–280 mm Hg; diastolic is 130–170 mm Hg. The most common CT finding was white-matter edema, diffuse or focal, affecting the supratentorial compartment in all cases and the infratentorial compartment in eight. These changes resolved after the blood pressure was lowered in all six patients studied by follow-up CT. Permanent areas of infarction were demonstrated in three patients. These abnormalities are correlated with the neuropathologic findings in hypertensive encephalopathy.

Hypertensive encephalopathy is an acute neurologic syndrome in which severe hypertension is associated initially with headache, nausea, and vomiting; later with convulsions, stupor, and coma [1–4]. Papilledema, retinal hemorrhages, and cardiac and renal failure are usually present. Complete or partial neurologic recovery is seen after institution of antihypertensive therapy. The computed tomographic (CT) findings in 11 patients with hypertensive encephalopathy were reviewed and related to the pathogenesis of this condition.

## Materials and Methods

The hospital records and CT scans of 11 patients with hypertensive encephalopathy from Montefiore Medical Center and New York University Medical Center were reviewed. In all cases, CT scans were obtained during the acute phase of blood pressure elevation. Six patients had follow-up CT scans.

The six male and five female patients were 10–59 years old (mean, 46). The diagnosis of hypertensive encephalopathy was made by the clinical criteria of an acute, severe rise in blood pressure associated with headache, nausea, vomiting, altered mental status, and focal neurologic deficits, and rapid improvement after control of blood pressure. The range of systolic blood pressure was 200–280 mm Hg (mean, 250 mm Hg); the range of diastolic pressure was 130–170 mm Hg (mean, 150 mm Hg).

## Results

The CT findings in hypertensive encephalopathy can be divided into supratentorial and infratentorial abnormalities. All cases demonstrated supratentorial abnormalities in the acute stage of hypertensive encephalopathy, characterized by edema and expansion of the white matter, compression of the ventricles, and obliteration of sulci and cisternal spaces (figs. 1–5). This diffuse white-matter edema resolved after the blood pressure was lowered in all patients in whom follow-up scans were obtained (figs. 2–5). Other supratentorial abnormalities observed during the acute phase of the disease were ganglionic edema in two patients (fig. 1), bithalamic

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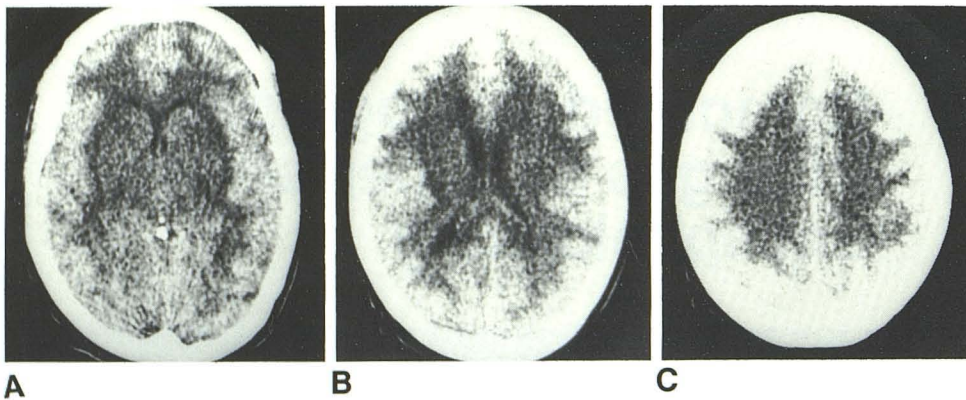


Fig. 1.—46-year-old woman with renal failure. Blood pressure = 240/150 mm Hg. Admission CT scans. Marked white-matter lucency and expansion, ganglionic low density, ventricular compression, and sulcal and cisternal obliteration. Findings are indicative of diffuse edema.

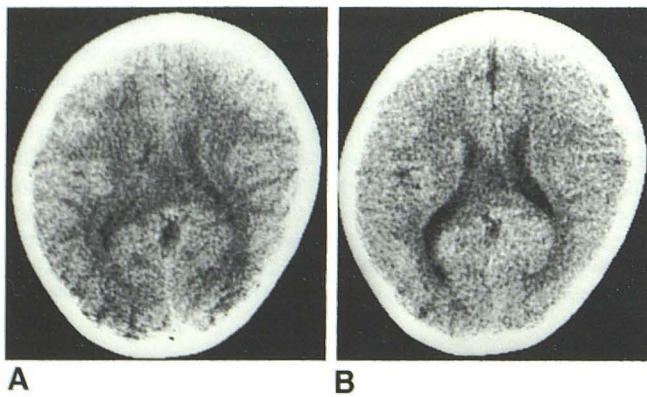


Fig. 2.—10-year-old boy with acute glomerulonephritis. Blood pressure = 230/180 mm Hg. A, Initial CT scan 8 hr after onset of symptoms. Diffuse white-matter edema. B, 10 days later. White matter is less prominent. Sulci are visible.

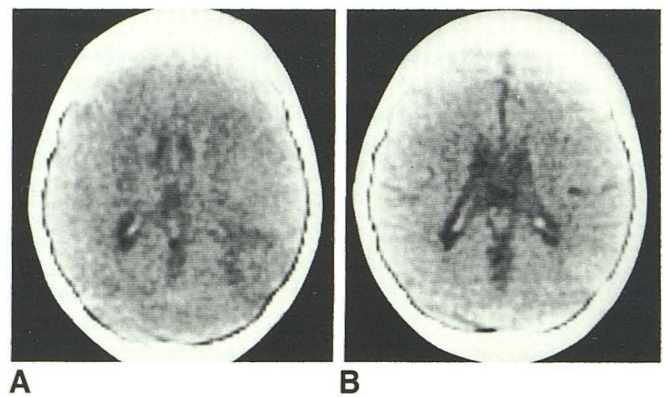


Fig. 4.—19-year-old man with lupus, headache, and blurred vision. Blood pressure = 240/150 mm Hg. A, Initial CT scan. Right parietooccipital wedge-shaped, infarctlike, focal lucency. B, 10 days later. Complete resolution of focal lesion and dilatation of ventricles and sulci indicative of resolution of edema and baseline atrophic state.

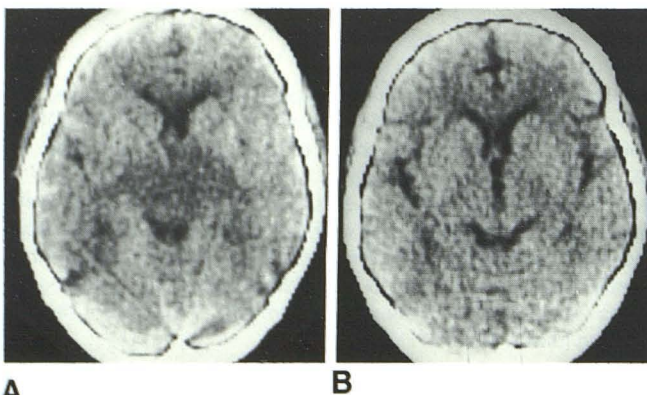


Fig. 3.—24-year-old woman with lupus. Blood pressure = 240/150 mm Hg. A, Initial CT scan. Bithalamic and brainstem edema and compression of third ventricle. Sulci demonstrated only minimally. B, 6 weeks later. Resolution of thalamic lesions. Sulci are now dilated secondary to lupus-induced atrophy.

edema in one patient (fig. 3A), and focal, infarctlike areas of lucency in three (fig. 4A). This latter finding resolved in two patients on posttreatment follow-up scans (fig. 4B). In the third patient, a permanent area of infarction remained. In three

patients, focal lesions were not seen in the acute stage but were observed on follow-up scans.

Infratentorial abnormalities were seen in eight patients. All of these cases demonstrated cerebellar white-matter edema and varying degrees of mass effect on the fourth ventricle and subarachnoid spaces. In one case, fourth ventricular compression was severe enough to produce hydrocephalus (fig. 5). Swelling and edema of the brainstem were seen in seven patients, and a focal lesion in the left cerebellar hemisphere was observed on a follow-up scan in one.

## Discussion

Hypertensive encephalopathy is a syndrome consisting of a sudden, sharp elevation of blood pressure associated with severe headache, nausea, vomiting, papilledema, and retinal hemorrhages and exudates. This may progress to confusion, stupor, coma, or focal neurologic signs. During the acute phase of the disease, diastolic blood pressure is usually 130 mm Hg or greater [3]. Hypertensive encephalopathy may complicate accelerated hypertension from any cause, including essential hypertension, acute and chronic renal disease,



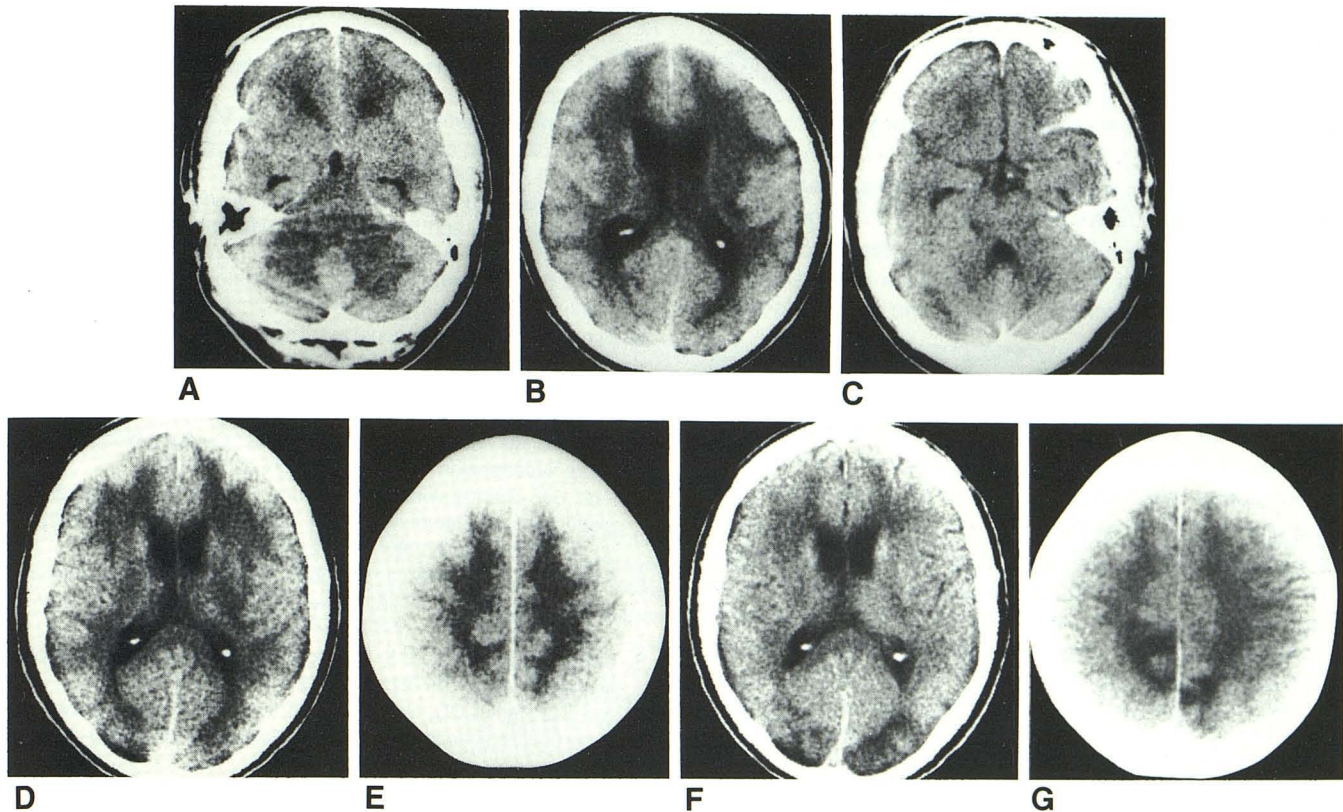


Fig. 5.—54-year-old man with headache, confusion, and retinopathy. Blood pressure = 240/190 mm Hg. **A** and **B**, Initial CT scans. Severe white-matter edema involving brainstem and cerebellum with compression of fourth ventricle and aqueduct producing hydrocephalus. Prominence and expansion of supratentorial white matter indicative of edema. **C–E**, 4 days later. Decreasing

supratentorial and infratentorial edema and resolving hydrocephalus. Fourth ventricle and quadrigeminal plate cistern no longer compressed. **F** and **G**, 10 days later. Focal biparietal and bioccipital lucencies have developed due to infarction. Edema has resolved.

disseminated vasculitis, eclampsia, and pheochromocytoma [1, 4]. The cerebrospinal fluid pressure and protein values are generally elevated. With prompt and vigorous blood pressure reduction, this neurologic syndrome is reversible [2–4].

Two divergent theories have been invoked in the pathogenesis of hypertensive encephalopathy. Under normal conditions, a rise in blood pressure is accompanied by autoregulatory cerebral arteriolar vasoconstriction, a mechanism to maintain constant cerebral blood flow [1, 4]. Some investigators postulate that, after a severe rise in blood pressure, diffuse vasoconstriction of cerebral arterioles occurs [1, 2, 5, 6]. Uncontrolled cerebral vasoconstriction and spasm leads to arteriolar necrosis, abnormal vascular permeability, and cerebral edema. Vasoconstriction and resultant cerebral hypoperfusion are thought to cause focal and diffuse areas of ischemia and infarction. The basal ganglia and deep periventricular white matter have been shown to undergo selective ischemic necrosis in hypoperfused states [7].

Others [2, 6, 8, 9] consider high-pressure autoregulatory failure and forced cerebral vasodilation to be the initial pathophysiologic event leading to hypertensive encephalopathy. As blood pressure rises, the upper limit of cerebral autoregulation is exceeded, cerebral hyperperfusion occurs, and cer-

ebal edema, hemorrhage, and infarction ensue.

Pathologically, either of these processes may lead to fibrinoid necrosis and thrombosis of cerebral arterioles and microinfarctions and petechial hemorrhages in the brain parenchyma [1–4]. These changes occur most often in the basal ganglia, deep cerebral white matter, and brainstem [2].

CT findings correlate well with these pathologic changes. Diffuse symmetric, well demarcated, low-density areas in the white matter that resolve after blood pressure control represent edema rather than infarction [10]. Ventricular compression with sulcal and cisternal obliteration also represents the effects of diffuse cerebral edema.

The degree and duration of blood pressure elevation correlate well with the severity of edema. Mild cases of hypertensive encephalopathy demonstrated supratentorial white-matter edema with little or no involvement of the infratentorial compartment. Severe cases demonstrated more extensive supratentorial edema and obvious brainstem, basal ganglionic, and cerebellar edema. Infratentorial edema is probably present in mild cases, but CT is insensitive to its detection. While supratentorial edema is seen in many conditions (anoxia, metabolic encephalopathy), bilateral cerebellar and brainstem edema in addition to supratentorial edema is an



uncommon finding and is characteristic of hypertensive encephalopathy. Focal cortical and white-matter hypodense lesions distributed asymmetrically and seen concurrently with diffuse cerebral edema usually represent reversible areas of edema and are not a prognostic indicator of a fixed neurologic deficit. Only when focal lesions appear initially or progress after resolution of diffuse cerebral edema do they signify areas of permanent infarction. Recognition of this finding could lead to a more aggressive therapeutic approach in patients with acute hypertensive encephalopathy in the face of focal lesions.

If hypertensive encephalopathy is treated promptly and adequately, the patient usually makes a complete neurologic recovery. The distinction between hypertensive encephalopathy and other neurologic syndromes associated with hypertension (lupus cerebritis, uremia) must be clearly drawn, because therapeutic decisions about rate and degree of blood pressure lowering depend on the specific diagnosis. Aggressive lowering of the blood pressure should be avoided in these latter conditions, since a rapid decrease in cerebral blood flow may cause cerebral ischemia. Close clinical correlation and the characteristic posterior fossa edema of hypertensive encephalopathy should help make this important distinction.

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