A comatose 82-year-old woman is brought to the emergency department. Her husband reports that after dinner she began to have difficulty in speaking, lost consciousness, and fell to the floor. He tried to rouse her, but was unsuccessful and called for an ambulance.

The patient has had no recent fever, rigors, night sweats, cough, chest pain, dyspnea, palpitations, ankle edema, headaches, vision problems, syncope, weakness, paresthesias, ataxia, head injury, rash, or bowel or urinary incontinence. Her appetite has been good, and there has been no recent change in her weight. She does not smoke or drink alcohol, and she exercises regularly.

**History.** The patient was previously healthy. Her only regular medication is aspirin (81 mg/d). Her father died of a myocardial infarction at the age of 76 years, and her mother died of cervical cancer.

**Examination.** Temperature is 36.6°C (98°F); heart rate, 90 beats per minute; respiration rate, 20 breaths per minute; and blood pressure, 122/66 mm Hg. She is deeply comatose and does not respond to painful stimuli (Glasgow Coma Scale score of 3). Her hydration status is good; there is no ankle edema. Head, ears, eyes, nose, and throat appear normal. There is no evidence of anemia, cyanosis, or adenopathy, and she has no rashes or tattoos. Her neck is supple; the thyroid gland is not palpable. Jugular venous pressure is normal, with the apex palpable in the fifth lumbar intercostal space. Heart sounds are normal, and lungs are clear. The abdomen is soft, without organomegaly.

Skull and spine are normal; carotid arteries are equally palpable and without bruits; pupils are small and nonreactive; and corneal, gag, and oculocephalic reflexes are absent. Because of obtundation, it is difficult to examine her cranial nerves and motor and sensory systems.

**Laboratory studies.** White blood cell count is 9400/µL, with 70% polymorphonuclear leukocytes, 24% lymphocytes, 2% monocytes, and 4% eosinophils. Hemoglobin level is 12.2 g/dL, and platelet count is 200,000/µL; the erythrocyte sedimentation rate is 8 mm/h. Urinalysis results are normal. Her blood glucose level is 94 mg/dL; blood urea nitrogen level, 22 mg/dL; creatinine level, 1.0 mg/dL; serum sodium level, 135 mEq/L; potassium level, 4.0 mEq/L; chloride level, 100 mEq/L; and carbon dioxide level, 20 mEq/L. An arterial blood gas analysis on 2 L of oxygen reveals a pH of 7.5; partial pressure of oxygen, 9 mm Hg; partial pressure of carbon dioxide, 32.6 mm Hg; and oxygen saturation, 99%. An ECG shows normal sinus rhythm. Prothrombin time is 13 seconds, and the international normalized ratio is 1.0. Results of liver function tests and a thyroid panel are normal. A CT scan of the brain without contrast is ordered.

**Based on the clinical picture and laboratory and CT findings, what is the most likely diagnosis?**

A. Glioblastoma multiforme.
B. Brain abscess.
C. Intracerebral hemorrhage.
D. Meningoencephalitis.

**INTRACEREBRAL HEMORRHAGE: AN OVERVIEW**

Intracerebral hemorrhage (ICH) accounts for about 10% of strokes. The incidence of ICH ranges from 9 to 29 per 100,000 persons; in the United States, ICH is responsible for more than 20,000 deaths each year. African Americans and Asians are affected more often than whites.

**RISK FACTORS**

Hypertension is the most important risk factor for ICH, particularly in smokers younger than 55 years and in those who do not adhere to their medication regimens. Other risk factors include amyloid angiopathy (seen often in the elderly), excessive alcohol use, and hypocholesterolemia (total cholesterol level less than 160 mg/dL). ICH is also associated with the rupture of aneurysms and
arteriovenous malformations (especially in young, normotensive patients), anticoagulants and thrombolytics, bleeding disorders, neoplasms, and drugs (eg, cocaine, methamphetamine). Amyloid was the most likely cause of intracerebral hemorrhage in this elderly woman (the family declined autopsy, so the cause could not be confirmed).

**CLINICAL PRESENTATION**
Symptom onset usually occurs during normal daily activity, although exertion or emotional stress may precipitate the event. A gradual, progressive loss of neurological function over minutes to hours is typical, with signs and symptoms corresponding to the location and size of the lesion (**Table**). An altered level of consciousness early on is an ominous sign that suggests a large hemorrhage. Up to 25% of patients who are initially alert will experience deterioration of their mental status within the first 24 hours as a result of hematoma expansion or worsening cerebral edema. Seizures affect 6% to 7% of patients.\(^2\) Headache and vomiting occur in about half of patients because of increased intracranial pressure or meningismus resulting from blood in the cerebrospinal fluid.

**DIAGNOSIS**
CT without contrast is the study of choice to confirm the diagnosis of ICH and to exclude other causes, including infarction. A noncontrast CT scan accurately defines the size and location of the hematoma; it also identifies the presence of any mass effect, hydrocephalus, or intraventricular hemorrhage. MRI can also be used to diagnose ICH; in fact, it is better than CT at revealing underlying structural abnormalities. However, CT is more readily available and significantly less costly. Consider angiography in normotensive patients who are younger than 45 years, patients with lobar hemorrhages, and those in whom the cause of ICH is not clear. Generally, unless rupture of an arteriovenous malformation or aneurysm is suspected, angiography is not warranted. In addition, obtain a complete blood cell count, coagulation studies, and a serum chemistry panel. In certain patients, a toxicology screen may be indicated.

**TREATMENT**
There are both medical and surgical options for the treatment of ICH. Initial management includes adequate airway and respiratory support; patients with decreased levels of consciousness may require endotracheal intubation. Management of hypertension in patients with ICH is somewhat controversial; however, a systolic blood pressure of less than 180 mm Hg or a mean arterial pressure of less than 130 mm Hg is generally considered acceptable. Antihypertensive agents commonly used to treat ICH include intravenous labetalol, esmolol, and nitroprusside. In patients with increased intracranial pressure, treatment options include intravenous mannitol, hyperventilation, and barbiturate coma. Correct coagulopathies with fresh frozen plasma, vitamin K, protamine, or platelet transfusion. The use of activated recombinant factor VIIa to promote hemostasis at the site of hemorrhage holds promise for the future.\(^3\) Surgical evacuation is recommended for cerebellar hemorrhages larger than 3 cm but is more controversial in hemorrhages that affect other areas of the brain. Ventriculostomies and ventriculoperitoneal shunts are sometimes used in patients with hydrocephalus. Hyperglycemia and hyperthermia are predictors of poor outcomes and warrant aggressive management. Seizure prophylaxis should be started and continued for at least 1 month; if the patient remains seizure-free, medication can be tapered and subsequently discontinued. There is no role for corticosteroids in the treatment of ICH.

**PROGNOSIS AND PREVENTION**
Despite the various treatment options available, ICH has a 30-day mortality rate of 40%.\(^4\) Of patients who survive, only a small number are able to live independently. Factors associated with a poor prognosis include:
- Large, infratentorial hemorrhage (greater than 60 mL).
- Low Glasgow Coma Scale score (less than 9) on initial presentation.
- Presence of intraventricular hemorrhage.

Although conclusive evidence is lacking, treatment of hypertension, avoidance of excessive alcohol use, smoking cessation, and careful use of anticoagulants and thrombolytics are recommended as important strategies for preventing ICH. *

**References:**
3. Friedrich JO. Recombinant activated factor VII for...

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