Radiological Reasoning: Hyperintensity of the Basal Ganglia and Cortex on FLAIR and Diffusion-Weighted Imaging

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Objective

The educational objectives of this article are to learn an approach to analyzing studies involving abnormalities of the basal ganglia in the acute setting. The approach is based on using medical history and imaging features to narrow the differential diagnostic considerations. An emphasis on understanding the pathophysiology is provided to reinforce the causality of the imaging abnormalities.

Conclusion

Abnormalities on imaging of the basal ganglia are often nonspecific. Therefore, a focused approach to limit the differential diagnosis is based on whether the patient presents with acute or chronic symptoms. The most sensitive sequence in the acute setting to detect abnormalities within the basal ganglia is diffusion-weighted imaging (DWI). The differential diagnostic considerations in the acute setting should include hypoglycemia, cerebral hypoxic encephalopathy, carbon monoxide poisoning, encephalitis, osmotic myelinolysis, toxin exposure, vascular causes, and illicit drug use.

Case History

A 24-year-old man presented to our emergency department via ambulance in a comatose state. Brain MRI was performed after urgent clinical assessment and stabilization in the emergency department.

Brain MRI

Unenhanced MRI examination of the brain was performed using a routine (standard) protocol; the following sequences were included: T1-weighted, T2-weighted, DWI with generation of apparent diffusion coefficient (ADC) maps, FLAIR, and blood-sensitive T2*.

The FLAIR images showed hyperintensity of the bilateral caudate nuclei, putamina, globus pallidi, and thalami (Fig. 1A). Cortical swelling was seen in a temporal–occipital–parietal distribution (Figs. 1A and 1B). DW images showed restricted diffusion of the basal ganglia and of the temporal–occipital–parietal cortex (Figs. 1C and 1D). T1-weighted images (not shown) depicted hypointensity within

the basal ganglia. The findings on the remaining brain images were unremarkable.

Expert Discussion

Initial Discussion

Abnormalities on imaging of the bilateral basal ganglia are often nonspecific. Therefore, a focused approach to limit the differential diagnosis is based on whether the patient presents with acute or chronic symptoms. Subsequent to that clinical correlation, imaging characteristics then may be useful to further refine the differential diagnosis.

Metabolism—To better understand the imaging characteristics of the basal ganglia, a brief review of the physiology and pathophysiology is provided herein. The basal ganglia form the core of the extrapyramidal system and regulate motor activity. This process requires chemical energy in the form of adenosine triphosphate (ATP) [1]. To produce ATP, the basal ganglia require a rich blood supply, which typically provides oxygen, glucose, and a high concentration of minerals [1]. ATP is produced through aerobic respiration and oxidative phosphorylation within the mitochondria [1]. An acute deprivation of oxygen, glucose, or both results in insufficient and inefficient production of ATP [1]. In addition to providing oxygen and minerals, a rich vascular supply to the basal ganglia is necessary to remove metabolic by-products that may be harmful if left in situ [1]. The basal ganglia contain inherently high concentrations of trace minerals such as iron, copper, and manganese, many of which are important cofactors for metabolic activity [1].

The basal ganglia have a high metabolic rate, which makes them particularly susceptible to damage from hypoxia or anoxia [1]. Injury may occur with any process that interrupts the normal steps in ATP production including, but not limited to, decreased oxygen, blood flow, or glucose [1]. In addition, conditions altering the normal mitochondrial structure or resulting in elevated mineral deposition can potentially injure the basal ganglia and thus impair their function [1]. Furthermore, the typically rich vascular supply to the basal ganglia makes them susceptible to toxins and infectious agents [1]. The typically high metabolic

A

B

C

D

Fig. 1—24-year-old man who presented in comatose state.
A and **B,** Axial FLAIR images show hyperintensity of caudate nuclei, putamina, globus pallidi, and thalami. Note cortical swelling in temporal–occipital–parietal distribution.
C and **D,** Axial diffusion-weighted images depict restricted diffusion of basal ganglia, thalami, temporal–occipital cortex, and parietal cortex.

demand of the basal ganglia is similar to that of the cerebellum and cerebral cortex [1]. Presumably, this characteristic is why the cortex and basal ganglia are often involved simultaneously in pathologic conditions along these lines [1]. A common end point leading to cellular injury may result from an insufficient production of ATP, resulting in increased intracellular water [1]. This increase, in turn, causes the basal ganglia to show abnormal signal hyperintensities on FLAIR, T2-weighted, and DW images. If hemorrhage or mineral or protein deposition also occurs within the basal ganglia, the basal ganglia may show T1-weighted signal hyperintensity or hypointensity or may be invisible on essentially any MR sequence of the brain.

Case Discussion

Because the patient described here presented with acute symptoms, the differential diagnosis includes but is not limited to hypoglycemia, hemolytic uremic syndrome, osmotic myelinolysis, encephalitis, carbon monoxide (CO) exposure, exposure to other toxins, recreational drug use, vascular causes, and hypoxic ischemic encephalopathy. The clinical presentation and imaging characteristics of these disease processes and of the abnormalities caused by toxin exposure or illicit drug use are reviewed herein. Chronic conditions are not discussed in this article for the sake of brevity.

Differential diagnosis: hypoglycemia—The neurologic signs of hypoglycemia (neuroglycopenia) include weakness, confusion, anxiety, and tingling of the autonomic group [2]. Seizures and coma usually occur in those with blunted responses to hypoglycemia such as patients with diabetes or tumor-associated hypoglycemia [2]. There are numerous causes of hypoglycemia, but most are related to diabetes [3]. Other causes include alimentary problems, fasting, tumors (insulinoma), endocrine disorders, hepatic diseases, and sepsis [2, 3]. Hypo-
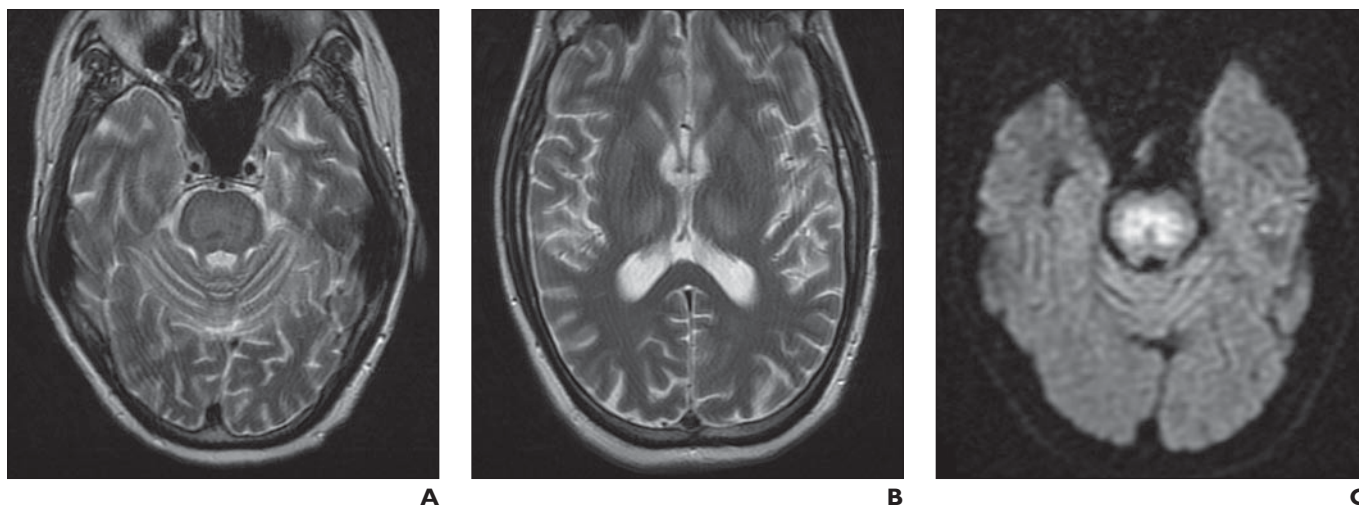
Fig. 2—Alcoholic man with ataxia.
A and **B,** Note T2 hyperintensity within pons (**A**) and basal ganglia and thalami (**B**), representing abnormal myelin signal in osmotic demyelination.
**C,** Restricted diffusion is observed within pons.

glycemia acutely results in "brain energy failure" and in membrane ionic pump failure, the latter of which causes a prominent rise of intracellular water [4]. The regions of affected brain are variable but include the temporal–parietal–occipital cortex, hippocampus, and basal ganglia, especially the caudate and lentiform nuclei [5, 6].

The changes in the brain caused by hypoglycemia may result in T1 and T2 prolongation in the acute phase and T1 and T2 shortening in the subacute phase [4–6]. Investigators have speculated that T1 and T2 shortening are the result of neuronal death, proliferation of glial cells, lipid accumulation, paramagnetic substance deposition, or a combination of these factors [6]. Restricted diffusion with decreased ADC values in the cerebral cortex, adjacent white matter, and basal ganglia may be seen in the acute phase [4]. Interestingly, the diffusion abnormalities in these cases may be transient and should not be confused with regions of infarction [4]. In the literature, authors have suggested that DWI may be used to define the areas of brain involved in hypoglycemia at an earlier time than conventional MR sequences [4]. Involvement of the cerebral cortex and basal ganglia has been reported to be associated with a poor clinical outcome [4]. Of note is that the absence of localized hemorrhage on MR scans is in marked contradistinction to the apparently minor hemorrhages noted from ischemic anoxic encephalopathy [6].

Differential diagnosis: hemolytic uremic syndrome—Hemolytic uremic syndrome is a form of microangiopathic hemolytic anemia that is secondary to an *Escherichia coli* infection and that results in acute renal failure, thrombocytopenia, and microthrombosis [1]. This *E. coli* infection preferentially affects the basal ganglia and may result in intracranial vascular occlusion [1]. Patients present with altered mental status, personality changes, seizures, and loss of vision [7]. Affected patients are usually younger than 5 years [1]. T1-weighted

imaging may illustrate hyperintensity within the basal ganglia from coagulative necrosis secondary to microthrombosis [7]. T2 hyperintensity may occur within the basal ganglia and cerebral cortex [7]. DW images can show reduced diffusion in the acute phase [7].

Differential diagnosis: osmotic myelinolysis—As discussed previously, the basal ganglia are particularly susceptible to electrolyte imbalances because of their high metabolic rate. Osmotic myelinolysis classically occurs from rapid correction of hyponatremia, often in malnourished alcoholics [8] but also may occur in transplant patients, secondary to polydipsia and in the chronically debilitated. Patients typically present with spastic quadriparesis and pseudobulbar palsy [8]. The imaging findings are characteristically centered within the pons, but the basal ganglia and thalami may be affected [1]. A rapid correction of hyponatremia has been purported to result in a release of myelinotoxic compounds by the gray matter compo-
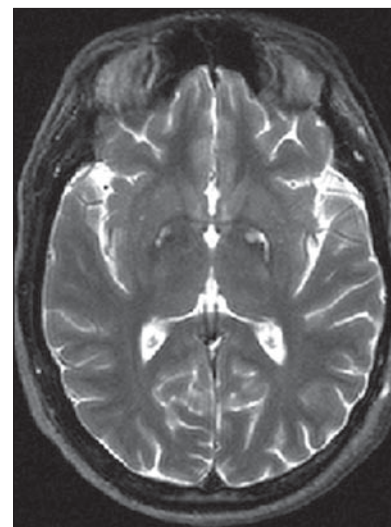
Fig. 3—Patient in whom T2 hyperintensity is observed within globus pallidi surrounded by ring of decreased T2-weighted signal. These findings are consistent with infarction of globus pallidi; surrounding hemosiderin deposition is due to hypooxygenation. (Courtesy of David Altman)
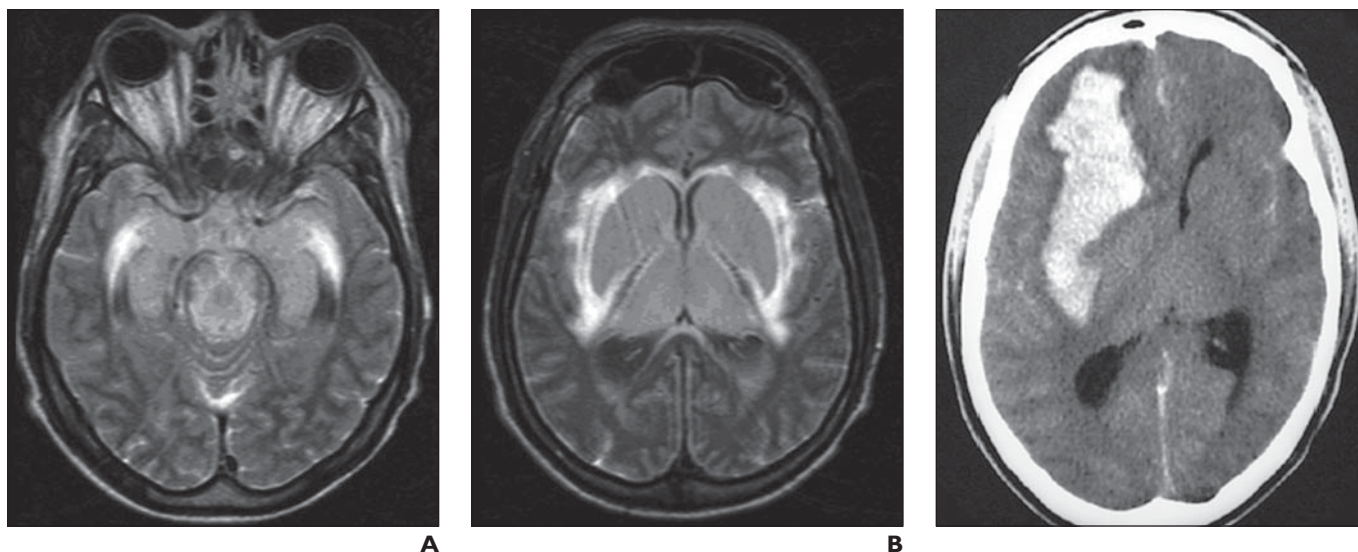
Fig. 4—Patient with ethylene glycol overdose. (Courtesy of David Altman)
A and **B**, Images show edema within hippocampi and midbrain (**A**) and within basal ganglia and thalami (**B**).
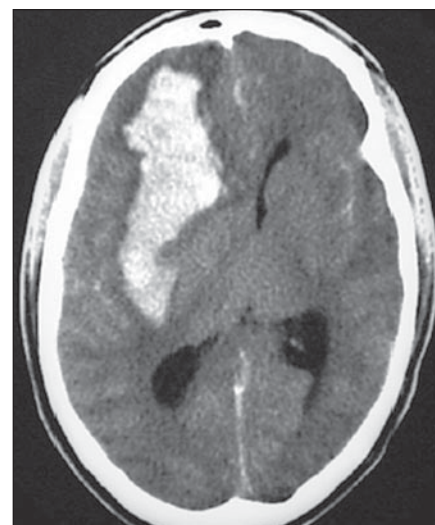
Fig. 5—Acute intraparenchymal hemorrhage in patient with cocaine overdose. Image shows hemorrhage involves white matter of right frontal lobe and corpus striatum. (Courtesy of David Altman)

nents within mixed white matter–gray matter regions [1]. This hypothesis may explain the apparent involvement of the basal ganglia and apparent sparing of the white matter and cortex on MRI. In this disease process, T2 hyperintensity may be seen within the pons, so-called "central pontine myelinolysis" (Fig. 2A); within the basal ganglia, so-called "extrapontine myelinolysis" (Fig. 2B); or within both. The hyperintensity on T2-weighted imaging supposedly results from an abnormal myelin signal [1]. Increased signal on diffusion-weighted sequences may be seen with normal, decreased, or elevated ADC values within the basal ganglia and pons and is most consistent with myelin destruction [9, 10] (Fig. 2C).

Differential diagnosis: encephalitis—Encephalitis or cerebritis in most patients is known to be or is suspected of being related to a viral infection [1]. The basal ganglia are richly vascularized by end-vessels, making them prone and susceptible to blood-borne infections [1]. Patients typically present with headaches, lethargy, anorexia, vomiting, and seizures [1]. Encephalitis may result in intracranial vascular compromise, hypoxic damage, and hemorrhagic infarction [1]. The neuroimaging characteristics are variable, in part depending on the underlying pathogen. Causes include but are not limited to cytomegalovirus, HIV, herpes simplex virus, varicella zoster virus, and eastern equine encephalitis, just to name a few [9].

Differential diagnosis: carbon monoxide poisoning—Acute CO poisoning may lead to loss of consciousness, cognitive impairment, coma, and death [11]. Chronic manifestations of CO poisoning include poor executive function, impaired memory, impaired processing skills, apraxia, and parkinsonian symptoms [11]. CO is an odorless gas [12]. CO poisoning is most often associated with fires, portable heaters, stoves, and automobile exhaust [12]. CO toxicity reduces the delivery of oxygen by three proposed mechanisms: competitive binding to hemoglobin; direct toxicity to mitochondria; and shifting of the classical oxyhemoglobin binding curve to the left, thereby decreasing oxygen delivery to tissues [1].

Of the basal ganglia structures, the globus pallidi are preferentially affected [1]. However, the remainder of the basal ganglia may also be involved [1]. CO poisoning results in high signal intensity typically within the globus pallidi and sometimes within the caudate or putamina on T2-weighted images [9] (Fig. 3). The globus pallidi may also be surrounded by a hypointense rim on T2-weighted imaging (putatively hemosiderin) [9]. Both hyperintensity and hypointensity within the globus pallidi may be observed on T1-weighted images [9]. Changes within the globus pallidi are considered to be most likely related to infarction resulting from poor oxygenation.

Symmetric preferential white matter involvement of the periventricular regions, corpus callosum, internal capsule, subcortical white matter, and white matter of the cerebellum has also been described [13, 14]. Affected white matter may show restricted diffusion and decreased ADC values before the basal ganglia do [14]. The patient's prognosis correlates more with the apparent degree of white matter damage on MRI than with the extent of apparent pallidal damage [1]. Observations have been reported in the literature that the regions of restricted diffusion within the white matter may be reversible for poorly understood reasons [14]. The cerebral cortex is often unaffected on MRI or is affected less prominently than the basal ganglia and white matter [14]. If the cortex is involved, the temporal lobes are favored [9].

Differential diagnosis: toxins or poisons—Toxins such as methanol, ethylene glycol, and cyanide preferentially affect the basal ganglia [1]. Exposure to these toxins most often

TABLE 1: Conditions, Toxins, and Illicit Drugs Affecting the Basal Ganglia and Their MRI Appearances

Condition, Toxin, or Illicit Drug	MR Finding			Miscellaneous Notes
	Basal Ganglia Affected	Cortex Affected	Hemorrhage or Hemosiderin Present	
Hypoxic ischemic injury	+[a]	+[b]	+[c] or −	Heart attack, anoxia, anesthesia, drowning, overdose
Hypoglycemia	+	+	−	Diabetes, paresthesias
Hemolytic uremic syndrome	+	+	+[d]	Renal failure, young age
Osmotic myelinolysis	+	−	−	Alcoholics, quadriparesis
Encephalitis	+	+	+	
Cyanide	+[e]	+	+	Attempted suicide, pseudoparkinsonism
Methanol or ethylene glycol	+[e]	−[f]	+[e]	Alcoholics, vision disturbances
MDMA	+[g]	+[h]	−	Ischemic infarct, hyponatremia
Cocaine or amphetamines	+	+[i]	+	Hypertension, hemorrhage of basal ganglia
Heroin or opiates	+[g]	+	−	Ischemic stroke
Carbon monoxide	+[g]	−[f]	+[j]	Parkinsonian symptoms, poor memory, exposure to exhaust from automobiles or heaters

Note—Plus sign (+) indicates MR finding present, minus sign (−) indicates MR finding absent. MDMA = methylenedioxymethamphetamine.
[a]Caudate and lentiform nuclei.
[b]Parietal cortex, occipital, temporal lobes, hippocampi, cerebellum.
[c]Possibly in basal ganglia.
[d]Basal ganglia.
[e]Putamina.
[f]White matter typically affected.
[g]Globus pallidi.
[h]Occipital.
[i]Posterior reversible encephalopathy syndrome.
[j]Around globus pallidi.

occurs in the adult population [1]. Methanol poisoning is often seen in alcoholics imbibing low-grade alcohol [9]. Patients poisoned with methanol present classically with blindness, vomiting, ataxia, and seizures [9, 15].

Methanol poisoning can result in hemorrhagic or nonhemorrhagic necrosis of the putamina, subarachnoid hemorrhage, and diffuse white matter necrosis [16]. The exact cause for the preferential involvement of the putamina is not known, but some investigators believe that it may be from the excessive levels of formic acid, the main toxic metabolite of methanol, preferentially occurring within the putamina [17]. Interestingly, when patients develop putaminal necrosis, permanent visual impairment is always experienced [17]. Initially, edema, shown as increased T2 signal, and hemorrhagic foci, appearing as blooming on the blood-sensitive T2*, are observed within the putamina [17]. Also, restricted diffusion may be seen within the putamina [17]. Later, the putamina may appear T1 hyperintense because of the presence of methemoglobin [17].

An overdose of ethylene glycol may lead to edema within the basal ganglia, thalami, midbrain, and upper pons 24–48 hours after ingestion [17] (Fig. 4). Follow-up imaging may show putaminal necrosis, much like that seen in methanol intoxication [17].

Cyanide poisoning is usually the result of attempted suicide [17]. The primary long-term neurologic sequela is pseu-

doparkinsonism with extrapyramidal symptoms [17]. Cyanide deactivates cytochrome oxidase, a terminal enzyme in the cellular respiration chain, which explains the preferential involvement of the basal ganglia [17, 18]. MR findings typically show edema within the putamina, globus pallidi, and sensorimotor cortex [18]. For unknown reasons, the hippocampi are usually spared [17]. Later, MR findings consistent with hemorrhagic necrosis of the striatum and pseudolaminar necrosis of the cortex are observed [18].

Differential diagnosis: illicit drugs—Recreational drugs such as cocaine, amphetamines, methylenedioxymethamphetamine (MDMA) or "ecstasy," heroin, and opiates may affect the basal ganglia [19]. For instance, cocaine and amphetamines may result in systemic vasoconstriction that can lead to severe hypertension [19]. Vasoconstriction may result in hypertensive hemorrhage within the basal ganglia (Figs. 4 and 5) or in posterior reversible encephalopathy syndrome [19]. In addition, ischemic stroke may occur and most often involves the cerebral white matter, particularly within the middle cerebral artery territory [19].

Ischemic stroke is well recognized as a complication of MDMA and heroin use and presumably occur because of vasoconstriction by the serotonin release and μ-opioid receptor activation, respectively [19]. The distribution of serotonin receptors renders the globus pallidi and occipital cortex particularly susceptible to MDMA-induced ischemia [19]. In-

farcts in the setting of heroin use involve the watershed territories such as the cerebellum, hippocampus, and globus pallidus. MDMA can lead to hyperthermia and dehydration [19]. Aware of the potential complication of dehydration and hyperthermia, consumers of MDMA may drink large quantities of water, which can lead to hyponatremia and subsequent cerebral edema [19]. Many of these recreational drugs may also result in anoxic brain injury if drug-induced cardiac arrest or severe respiratory depression occurs [19].

Differential diagnosis: vascular etiologies—Infarction of the medial thalami and midbrain may occur from occlusion of the artery of Percheron [20]. This is a rare anatomic variant of a common trunk arising from one of the proximal segments of the posterior cerebral artery that supplies the paramedian thalami and midbrain [21]. Patients may present with a wide range of symptoms, including motor deficits and sensory and behavioral abnormalities [21]. Occlusion of the basilar tip, "top of the basilar syndrome," may also produce bilateral thalamic infarcts, but typically the superior cerebellar artery and posterior cerebral artery territories are also involved [21]. Therefore, when paramedian infarction of the midbrain and thalami is observed, occlusion of the artery of Percheron should be considered the main diagnosis [21].

Deep cerebral venous thrombosis involving the internal cerebral veins, vein of Galen, or straight sinus may cause cytotoxic or vasogenic edema within the thalami, sometimes extending into the caudate nuclei and deep white matter [22]. Patients may present with seizures and symptoms secondary to increased intracranial pressure caused by mass-effect upon the third ventricle leading to hydrocephalus [22, 23]. Hemorrhage is noted in 19% of patients [22]. Rarely, unilateral thalamic edema may be seen secondary to thrombosis of the single internal cerebral vein [24]. Acute thrombus is predominately T1-weighted isointense and T2-weighted hypointense. The T2-weighted hypointensity may be mistaken for a flow void [22]. During the subacute and chronic phases, thrombus is typically T1-weighted and T2-weighted hyperintense and T1-weighted and T2-weighted hypointense, respectively [22]. Furthermore, contrast enhancement may occur in the thrombosed sinuses because of organized thrombus [22]. Therefore, MR venography or CT venography is usually necessary to confirm the diagnosis [22]. Diffusion abnormalities may be transient, which may be secondary to recanalization of thrombus or improvement of collateral drainage [23]. In a study by Mullins et al. [23], when restricted diffusion occurred in the absence of seizure activity, the lesions showed abnormality on follow-up imaging.

Differential diagnosis: cerebral hypoxic encephalopathy—Cerebral hypoxic encephalopathy results primarily from a failure of adequate blood flow or oxygen delivery to the brain from failure of the heart and circulatory system or the lungs and respiratory system [25]. Causes include reduction of blood flow from a myocardial infarction, arrhythmia, blood loss, or shock; hypoxia from drowning,

strangulation, compression of the airway, or airway obstruction; CO poisoning; failure of the respiratory muscles (e.g., Guillain-Barré syndrome); high altitude sickness; and exposure to oxygen-deficient agents during anesthesia [25]. Presentation can vary from vague symptoms of inattentiveness to stupor or coma [25].

On MRI, the pattern of brain injury from ischemia may differ from that of anoxia by affecting the watershed zones between the major vascular territories rather than the cerebral cortex, cerebellum, and deep gray nuclei [25]. However, the patterns may be mixed [25]. Cerebral anoxia disrupts the Krebs cycle and ion transport, thereby leading to cell death (cytotoxic edema) [25]. Anoxic brain injury is usually complete if prolonged for more than 5 minutes [25]. The degree of anoxic damage may be modified by continued cardiac activity or body temperature cooling, apparently mitigating the anoxic injury to the brain [25]. Therefore, clinical presentation may vary depending on the amount of time the patient is anoxic and how soon treatment is instituted.

The MRI findings of anoxic brain injury vary depending on the time at which MRI takes place after the anoxic event, the length of time of the hypoxia or anoxia, and the severity of the insult [26]. Acutely, edema is shown by T2 and FLAIR hyperintensity within the cortex, thalami, and basal ganglia [26]. Cortical involvement is typically within a parietooccipital and cerebellar distribution [26]. DWI performed during the acute phase (< 24 hours after the event) is more sensitive to the detection of abnormal basal ganglia and cerebellar and cortical involvement than is conventional MRI [27]. This observation may be related to the increased sensitivity of DWI to restricted water movement [27]. The cerebellum on DWI is typically more hyperintense on DWI sequences than are the basal ganglia and cerebral cortex [27]. Cortical involvement on DWI in this setting is typically located in the occipital or perirolandic region [27].

During the early subacute phase, predominantly gray matter and cortical abnormalities are seen on DWI, whereas in the late subacute stage, DW images may show mostly white matter involvement. Reperfusion occurs in the subacute phase leading to insufficient cerebral blood flow [28]. This causes vasogenic edema leading to increased signal on conventional imaging and exacerbates cytotoxic edema resulting in increased diffusion signal, especially on higher b-value diffusion images [28, 29]. Spectroscopy may show elevated lactate levels in affected areas [29]. At around day 7 after the anoxic or hypoxic event, the diffusion abnormalities and metabolic ratios may normalize in regions of ischemia [29]. This phenomenon is known as "pseudonormalization" [29]. However, metabolic ratios may not always normalize, making spectroscopy useful for difficult cases [29].

During the subacute phase, laminar necrosis may manifest as T1 hyperintensity or contrast enhancement within the cortex [26]. T1 hyperintensity in this setting has an apparent predilection for the depths and sides of the gyri

rather than the crests of the gyri [26]. T1 hyperintensity of the cortex has been hypothesized to be caused by microhemorrhage or fat-laden macrophages [26]. T1 hyperintensity of the basal ganglia may be related to a minor hemorrhage resulting from RBC diapedesis after reperfusion [30]. Also, contrast material enhancement typically occurs in the basal ganglia, hippocampus, cortex, or cerebellar tonsils. This contrast enhancement presumably results from a breakdown of the blood–brain barrier.

White matter involvement in hypoxia or anoxia is variable and may be seen in a subcortical, deep distribution or in a diffuse distribution [29]. White matter is usually involved during the subacute and chronic phases [27]. Increased signal on diffusion sequences may be more pronounced than on conventional imaging because of limited brownian motion from ischemic tissue and Wallerian-like degeneration [27]. Also, the phenomenon of late neuronal death may contribute to the increased T2 and diffusion signal predominately affecting the neocortex, hippocampi, and basal ganglia [27].

Many of the findings noted in the subacute phase may also be seen in the chronic phase [27]. Additional findings during the chronic phase include severe atrophy resulting in cortical and ventricular prominence and white matter involvement [27]. During the chronic stage, cellular death and destruction have likely occurred resulting in an increase in the extracellular space, cavitation, and increased brownian motion. These changes appear as decreased signal on high-B-value DWI [27].

Commentary

The patient presented here suffered an anoxic brain injury secondary to a near-drowning episode. The MR finding of restricted diffusion in the basal ganglia and parietal–occipital cortex are presumably the most sensitive finding for the diagnosis of an acute anoxic event (based on the available literature) [27]. This imaging finding is by no means specific. Because the hippocampi, basal ganglia, cerebellum, and occipital cortex are the most metabolically active regions of the brain and use a rich blood supply, glucose, and oxygen to ultimately maintain ATP production, they may be affected by any process that disrupts or involves this relationship. Therefore, imaging abnormalities of the basal ganglia often overlap in appearance and may manifest as a result of numerous causes. An accurate and thorough history and an assessment of discriminating imaging findings are crucial in obtaining a limited differential diagnosis in the setting of an acute process such as this one. Please see Table 1, which summarizes the conditions, toxins, and illicit drugs affecting the basal ganglia and their imaging appearances.

Clinical Management

Approximately 1% of emergency department admissions consist of unresponsive persons [31]. In a prospective study by Forsberg and colleagues [31], the cause for the unconscious person presenting in the emergency department was as follows: poisoning, 38%; focal neurologic lesion (i.e., stroke or brain tumor), 24%; metabolic disturbance or diffuse neurologic lesion, 21%; an epileptogenic cause, 12%; a psychogenic cause, 1%; and no clarified cause, 4% [31]. Of the patients with poisoning, the cause was as follows: ethanol, 36%; ethanol and sedative hypnotics, 17%; narcotics (mainly heroin), 10%; miscellaneous, 17%; and near drowning, 0.2% [32].

Poisoning was the cause of unconsciousness in 80% of the cases when the patient was younger than 40 years and only 11% of the time when the patient was older than 60 years [31]. Unless the diagnosis is established at once by history and physical examination, laboratory workup is necessary [25]. This workup should include CT or MRI of the head; consider lumbar puncture; blood and urine toxic screening; tests of blood concentrations of anticonvulsants, opiates, benzodiazepines, barbiturates, alcohol, and other toxins; urinalysis; venous blood gases; analysis of blood gases such as carboxyhemoglobin; basic metabolic profile; and possibly electroencephalography [25].

Treatment of an unconscious patient is centered on treating the cause and providing supportive measures to restore cardiac activity and respiratory function [26]. For instance, naloxone may be given to reverse opiate overdose [26]. In the patient we describe, the cause of the anoxic brain injury was known from the history. Treatment of anoxic brain injury is supportive consisting of prompt restoration of cardiac and respiratory function through cardiopulmonary resuscitation with the use of cardiac defibrillation or pacemaker placement and proper airway management, ventilation, and oxygenation [26]. Hypothermia has a slight benefit in the early outcome and may prevent delayed worsening [26]. The randomized study by Bernard and Buist [32] showed a doubling of survival and a better outcome with reduction of the core body temperature to 91°F.

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