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Rapid Development of Basal Ganglia Calcification

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The pathologic and radiologic appearance of calcification of the corpus striatum is now well recognized. This calcification can be associated with endocrine, congenital/developmental, inflammatory, and anoxic/toxic conditions. Although the association of the basal ganglia calcification with these conditions is well establised, its pathogenesis is poorly understood. We found no specific answer in the literature as to how long it takes for the pathologic basal ganglia calcification to occur. However, we got the impression that it takes at least several months to years. We recently encountered a young patient in whom the basal ganglia calcification was demonstrated on sequential computed tomography (CT) scans after only 31 days.

Case Report

A 29-year-old woman was admitted after an alcoholic binge of 2 days. She was nauseated and complained of epigastric pain. Laboratory studies revealed decreased serum levels of sodium, calcium, and phosphate. Her condition deteriorated and she was believed to have hemorrhagic necrotic pancreatitis. An exploratory laparotomy confirming this was complicated by a stormy postoperative course. She developed diabetes mellitus, respiratory difficulty, and acute tubular necrosis.

Serial serum calcium and phosphate levels remained quite low for 2–4 weeks after the operation (serum calcium levels were as low as 4–5 mg/dl and serum phosphate levels as low as 1 mg/dl). During this time she sustained two cardiopulmonary arrests with successful resuscitation. Postoperative neurologic status varied from decorticate and decerebrate posturing to the ability to speak only short sentences. Whereas she could execute spontaneous movements in all her extremities, her tone was considerably increased and she had tremors of both extremities (extrapyramidal signs). Her other medical problems were adequately controlled.

An initial CT scan at admission was normal (fig. 1A). A scan 31 days later showed extensive basal ganglia calcification (fig. 1B). A lateral skull film done 3 weeks after the second scan revealed no basal ganglia calcification.

Discussion

Basal ganglia calcification was initially reported independently by Virchow [1] and Bamberger [2] in 1855. Its radiographic visualization was first described by Fritzche [3] in 1935. Since the reports of Eaton and Haines [4] and Sprague et al. [5], who showed relation of basal ganglia calcification to hypoparathyroidism and pseudohypoparathyroidism, respectively, many other reports have discussed the association of basal ganglia calcifications with other conditions [6–17]. From these reports it is clearly evident that in the central nervous system, the basal ganglia, particularly the globus pallidus, show a great proclivity for calcification. Our case further supports this premise. From the clinical summary it is perhaps fair to conclude that the underlying cause of basal ganglia calcification in our case was anoxia.

The vulnerability of the basal ganglia, particularly of the globus pallidus, to anoxic injury (pallidal necrosis) has long been recognized [17, 18]. It is rare for the corpus striatum (putamen and caudate nucleus) to suffer this type of anoxic injury without concomitant pallidal injury. The *pathoclisis* (susceptibility to involvement by toxins) theory of Vogt and Vogt [19] was based on the assumption that certain parts of the brain had physiochemical properties that made them more susceptible to anoxic damage [20, 21]. Several factors have been postulated to explain this susceptibility of the globus pallidus and corpus striatum to anoxic injury and consequent dystrophic calcification. These factors include:

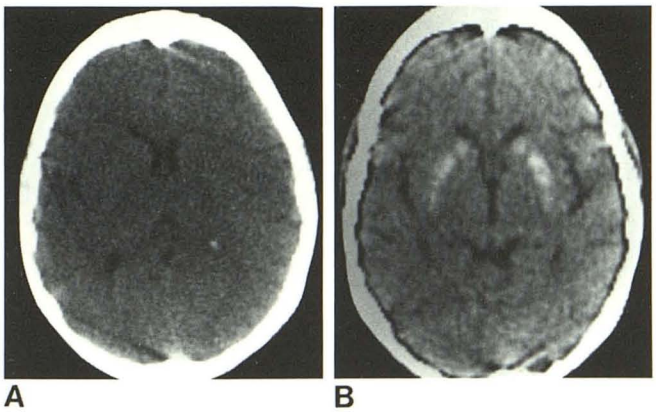

Fig. 1.—**A**, Normal admission scan. **B**, 31 days later. Extensive basal ganglia calcification.

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(1) abundance of oxidative enzymes [20–22]; (2) peculiarities of the arterial supply [23–29]; and (3) disturbance of vasomotor regulation [30]. Physiologic calcification of the globus pallidus after the age of 40–50 years gives further credence to this theory [16]. It is difficult to ascertain if the relative abundance of iron in the globus pallidus increases its susceptibility to calcium deposition [31].

Pathologically the calcification is located in and around the finer blood vessels [32]. This calcification follows colloid deposition in the substrate; the colloid then changes into a gel that accepts calcium salts [32].

We were unable to find information regarding the time required for secondary basal ganglia calcification to occur. However, our case suggests that the basal ganglia are not only highly susceptible to anoxic injury and consequent dystrophic calcification, but also that basal ganglia calcification can occur in as short a time as 31 days, much more rapidly than generally believed.

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