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CT Diagnosis of Basilar Artery Occlusion

Dimitrios Vonofakos,¹ Harry Marcu, and Hans Hacker

Basilar artery occlusion was diagnosed by high-resolution computed tomography (CT) in 11 cases. The normal basilar artery imaged on plain CT scans has an attenuation value not higher than that of whole blood. If the CT attenuation value of the vessel is higher than that of blood, basilar artery occlusion is probably present. The tentative diagnosis can be confirmed by comparison of the attenuation values on the pre- and postcontrast scans, preferably using multiple slices obtained by multiplane dynamic CT.

The ability to diagnose basilar artery occlusion by computed tomography (CT) is of particular importance because this disorder is not as rare as has generally been believed and does not occur only in older, arteriosclerotic patients [1, 2]; furthermore, in most cases a correct diagnosis is not made before angiography [2]. The outcome in patients with basilar artery occlusion is not always catastrophic, as previously thought, so early diagnosis may improve outcome by indicating appropriate therapeutic measures [2].

Many authors have described characteristic CT findings in cases of dilated, tortuous, elongated basilar artery with wall calcifications (megadolichobasilar artery) [3, 4]. In reported cases of ischemic disease in the area supplied by the vertebrobasilar system, assessment of the basilar artery CT attenuation values has not been attempted. The only case, to our knowledge, with definite evidence of occluded basilar artery diagnosed by CT was reported by Kuckein [5].

We diagnosed basilar artery occlusion in 11 cases by measuring the CT attenuation values of the vessel on the plain scan alone (five cases), on the plain scan in combination with postinfusion scans (two cases), or on multiplane dynamic CT scans (four cases).

Materials and Methods

The clinical symptomatology, CT findings, and other findings in our 11 cases of basilar artery occlusion are presented in table 1. CT examinations were performed on Siemens Siretom I and Somatom 2N scanners. The plain scans were obtained with the thinnest possible slices (2 mm or 4 mm) at the level of the basilar artery in order to visualize the entire length of the vessel. When contrastenhanced scans were obtained, multiplane dynamic CT was preferred, with intravenous injection of 100–150 ml contrast medium and flow of 1–1.5 ml/sec. These scans were obtained with the Telebrix 300 (Byk Gulden, Konstanz, West Germany) or Rayvist 300 (Schering, West Berlin). During the injection period, adjacent slices 2 mm or 4 mm thick were obtained at the level of the basilar artery. The attenuation values of the basilar artery were measured in all scans. To serve as a reference, the attenuation value of whole blood (hematocrit, 44%) was measured on our CT scanner. In addition, as a control, we estimated the mean CT attenuation value of the basilar artery in 21 randomly selected routine cases examined with the Siretom I scanner and in 100 randomly selected cases examined with the Somatom 2N.

Results

The CT attenuation values of the basilar artery on the plain and contrast-enhanced scans in the 11 cases of occlusion are given in table 1.

The attenuation value of whole blood as measured on our scanner was 45 Hounsfield units (H). Other authors have reported attenuation values ranging from 52 H to 56 H for whole blood [6, 7]. The distribution of mean attenuation values for the basilar artery in the 121 control cases is presented in figure 1.

If the CT attenuation value of the basilar artery in the plain scan is higher than that of whole blood (or, according to various measurements, higher than 45–56 H), the vessel is probably occluded. The high attenuation value represents thrombotic material in the occluded basilar artery. Similar observations have been made for cerebral emboli [8] and sinovenous occlusion [9, 10]. The attenuation values on plain scans obtained in cases of basilar artery occlusion were higher than values for whole blood in nine of 11 cases (table 1; case 4: fig. 2A; case 10: fig. 3). The two other cases were case 3, in which the diagnosis was confirmed by the contrastenhanced scan, and case 11 (a brainstem infarct; fig. 4).

Although wall calcifications in arteriosclerotic disease are not as common in the basilar artery as in the cavernous portion of the internal carotid artery, calcium deposits can result in a high attenuation value and thus result in a mistaken diagnosis of basilar artery occlusion. However, the calcifications that we observed were typically located at the periphery of the vessel and could be recognized as such. Another potential pitfall is a pathologically high hematocrit. For these reasons, determination of basilar artery occlusion on the basis of a high attenuation value for the vessel solely on the plain scan is only suggestive, not diagnostic.

A positive diagnosis of this disorder can be made with a combination of plain and contrast-enhanced scans. If the attenuation value of a given part of the basilar artery remains unchanged on the postcontrast scan in comparison with the precontrast scan, while the other vascular structures opacify, the diagnosis of occlusion is definite. The distribution and extent of the thrombosis in the artery can also be estimated, as was done in our cases 4 (fig. 2), 6, 8 (table 2 and fig. 5), and 10 (fig. 3).

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TABLE 1: Summary of 11 Cases of Basilar Artery Occlusion Diagnosed by CT

Case No.: Age (years)	Medical History, Clinical Symptoms, and Outcome	CT Findings	Angiographic and/or Autopsy Findings
1:38	L-S hemiplegia, coma 3 hr preadmission. Died 2 days later.	BA att: PS, 62 H. No CS.	Ang: proximal BA occlusion. No aut.
2:47	Epileptic seizure, coma, L-S mydriasis 6 hr preadmission. Died 3 days later.	BA att (distal part): PS, 55 H. No CS.	Ang: megadolichobasilar ar- tery and distal BA occlu- sion. Aut: distal BA throm- bosis.
3:58	Brainstem symptoms, L-S hemiparesis 24 hr preadmission. Died 10 days later.	BA att: PS, 36 H; CS, no change.	No ang. Aut: BA thrombosis.
4:49	Extension spasms, coma, R-S mydriasis 6 hr preadmission. Died 4 weeks later.	BA att (distal part): PS 65 H; DS, no change.	Ang: left VA and distal BA oc- clusion. No aut.
5:72	Deep coma 12 hr preadmission. Died 3 weeks later.	BA att: PS, 63 H. No CS.	No ang. Aut: BA thrombosis.
6:58	Deep coma 12 hr preadmission. Died 6 days later.	BA att: PS (proximal 2/3 of BA), 60–70 H; DS, increased att only in distal 1/3 of BA.	No ang. Aut: thrombosis of proximal 2/3 of BA.
7:69	Deep coma, tetraplegia 12 hr preadmis- sion. Died 1 week later.	BA att: PS, 62–68 H. No CS. Extensive occipital infarcts.	No ang. No aut.
8:78	L-S hemiplegia, coma 6 hr preadmission. Died 6 days later.	BA att: PS (proximal 1/3 of BA), 42–74 H; DS, increased att only in distal 2/3 of BA.	No ang. Aut: proximal BA thrombosis.
9:67	Deep coma 6 hr preadmission. Died 1 day later.	BA att: PS, 60-64 H; CS, no change.	No ang. Aut: left VA and BA thrombosis.
10:57	Brainstem TIAs beginning 1 year pread- mission. Symptoms unchanged.	BA att: PS, 38–45 H; DS, no increase in middle 1/3 of BA.	Ang: occlusion of middle 1/3 of BA.
11:1.5	Cardiac operation 1 year preadmission. Epileptic seizure, L-S hemiplegia, som- nolence 24 hr preadmission. Patient still alive and comatose 4 weeks later.	BA att: PS, 52–58 H. No CS. Brainstem infarction. CT 16 days later: BA att: PS, 32–35 H.	No ang.

Note.—L-S = left-sided; R-S = right-sided; TIAs = transient ischemic attacks; BA = basilar artery; VA = vertebral artery; PS = plain scan; CS = contrast-enhanced (infusion) scan; DS = dynamic scan; att = attenuation value; H = Hounsfield units; ang = angiography; aut = autopsy.

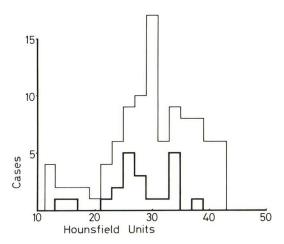


Fig. 1.—Distribution of CT attenuation values for normal basilar artery in 100 randomly selected cases examined with Somatom 2N scanner (*thin line*) and 21 randomly selected cases examined with Siretom I scanner (*heavy line*).

Discussion

The results in our study of 11 cases indicate that basilar artery occlusion can be tentatively diagnosed by direct recognition of the thrombus on the plain scan. Yock [8] reported several cases of calcified intracranial emboli that could be recognized by plain CT. Not only calcium within a thrombus or embolus but also dense fibrin

or platelet or thrombus without calcification can be imaged in venous occlusion [9]. In some instances, as in our case 11, the attenuation values of the basilar artery suggest that thrombotic material has a high density only in the first phase after thrombosis, while in time the thrombus becomes isodense (fig. 4). Unfortunately, angiography was not performed in case 11, so the possibility of recanalization of the basilar artery cannot be excluded.

Confirmation of a tentative diagnosis of basilar artery occlusion is possible only by comparison of the attenuation values on the preand postcontrast scans. Multiple thin slices should be obtained and a high-resolution scanner must be used. Injection of contrast material increases the iodine concentration in blood; thus, the difference in attenuation values of blood vessels before and after contrast medium administration is evident on dynamic CT [11]. If no change in attenuation values is observed, the diagnosis of basilar artery occlusion is confirmed.

Awareness of the possibility of CT evaluation of this disorder can prevent unnecessary angiographic investigation in hopeless cases. Even more valuable is the ability to make an early, definite diagnosis of basilar artery occlusion by means of this noninvasive method, which could result in early intervention and an improved prognosis.

REFERENCES

- Latchaw RE, Seeger JF, Gabrielsen TO. Vertebrobasilar arterial occlusion in children. *Neuroradiology* **1974**;8:141–147
- Thompson JR, Simmons CR, Hasso AN, Hinshaw DB Jr. Occlusion of the intradural vertebrobasilar artery. *Neuroradiology* 1978;14:219–229
- 3. Hammer B. Computertomographische Diagnose der Megadol-

A

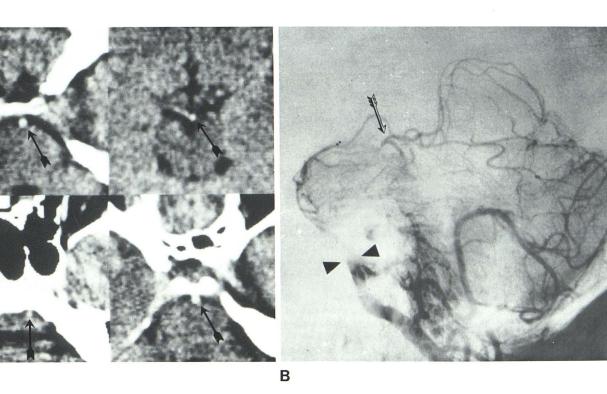


Fig. 2.—Case 4. Distal basilar artery occlusion. A, Plain CT scans. Attenuation values of basilar artery (*arrows*): *upper right*, 60 H; *upper left*, 65 H; *lower right*, 67 H; *lower left*, 43 H. First three readings represent occlusion of distal part of artery. Reading in scan at lower left represents normal

attenuation value of unoccluded part of artery. **B**, Left-sided vertebral angiogram. Distal occlusion of basilar artery (*arrowheads*). Retrograde filling of superior cerebellar artery (*arrow*).

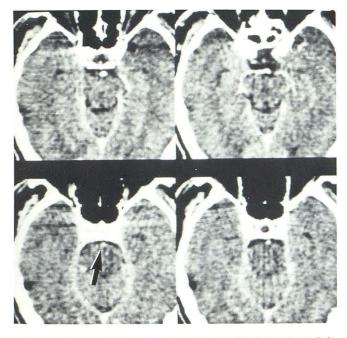


Fig. 3.—Case 10. Midbasilar artery occlusion. Contrast-enhanced dynamic CT scans. Attenuation values of basilar artery: *upper right*, 92 H; *upper left*, 96 H; *lower right*, 78 H; *lower left*, 45 H. Occlusion in scan at lower left (arrow).

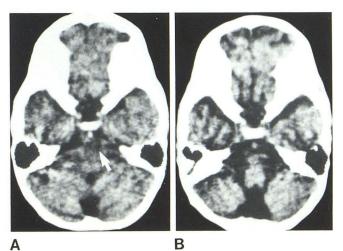


Fig. 4.—Case 11. Brainstem infarction. Plain CT scans. **A**, On admission. Attenuation value of basilar artery, 58 H (occlusion). Infarct at *arrow*. **B**, 16 days after onset. Attenuation value of basilar artery, 33 H.

 TABLE
 2: CT Attenuation Values in Proximal Basilar Artery

 Occlusion (Case 8)
 (Case 8)

	Basilar Artery Density (H)	
Imaging Position —	Plain Scan	Contrast-Enhanced Scan
76	48	88
80	51	86
84	50	95
88	66	91
92	71	74*
96	74	72*
100	42	44*
104	48	120

Note.—Both the slice thickness and the imaging table position incrementation for adjacent slices were 4 mm. Attenuation values are given in Hounsfield units (H). * Occluded part of basilar artery is indicated by postcontrast attenuation values in

imaging positions 92, 96, and 100, which show little change from precontrast values.

ichobasilaris. ROFO 1979;131:255-261

- Moseley IF, Holland IM. Ectasia of the basilar artery: the breadth of the clinical spectrum and the diagnostic value of computed tomography. *Neuroradiology* **1979**;18:83–91
- 5. Kuckein D. Vaskuläre und hypoxische Gewebsläsionen im kranialen Computertomogramm und ihre Differentialdiagnose. *Computer Tomographie* **1982**;2:120–126
- New PFJ, Aronow S. Attenuation measurements of whole blood and blood fractions in computed tomography. *Radiology* 1976;121:635–640
- Norman D, Price D, Boyd D, Fishman R, Newton T. Quantitative aspects of computed tomography of the blood and cerebrospinal fluid. *Radiology* 1977;123:335–338
- 8. Yock DH Jr. CT demonstration of cerebral emboli. *J Comput* Assist Tomogr **1981**;5:190–196

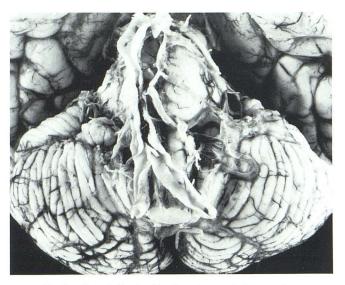


Fig. 5.—Case 8. Proximal basilar artery occlusion on autopsy.

- Buonanno FS, Moody DM, Ball MR, Laster DW. Computed cranial tomographic findings in cerebral sinovenous occlusion. *J Comput Assist Tomogr* **1978**;2:281–290
- Rao KCVG, Knipp HC, Wagner EJ. Computed tomographic findings in cerebral sinus and venous thrombosis. *Radiology* 1981;140:391-398
- Yamamoto M, Shinohara Y, Kamei T, Yoshii F. Diagnosis of internal carotid artery occlusion by dynamic computed tomography. J Comput Assist Tomogr 1981;5:637–640