When a vascular disease is suspected, the focus is usually on morphologic features seen at contrast material–enhanced multidetector computed tomography (CT). However, unenhanced CT also plays an important role in revealing so-called hyperattenuating signs, which represent a slight increase in the focal attenuation of a vessel. Hyperattenuating signs are occasionally observed when an acute clot has formed in a vessel and can be seen in various vascular diseases, including acute arterial occlusion, acute arterial dissection, aneurysm rupture, and acute venous thrombosis. The attenuation of these signs tends to increase because the concentration of hemoglobin increases as water content decreases. Hyperattenuating signs are a transient phenomenon, as the attenuation gradually decreases. Therefore, they can serve as unique findings indicating an acute state. Although hyperattenuating signs are not well understood, recognition of these signs is important because they can help reveal serious acute vascular diseases even at unenhanced CT.
Introduction

The development of multidetector computed tomography (CT) has allowed detailed evaluation of vascular diseases. Contrast material enhancement plays a particularly important role in revealing morphologic abnormalities associated with these diseases. However, unenhanced CT also plays an important role in some vascular diseases. For instance, it can help detect an increase in the attenuation of the middle cerebral artery (MCA) caused by acute cerebral infarction of the MCA region (Fig 1) (1,2). This increased attenuation reflects the formation of fresh clots and is one of the first early CT signs of acute cerebral infarction (1,2). Although this finding is well known as the hyperattenuating MCA sign, similar findings can be observed in other vessels throughout the body and are generically known as “hyperattenuating signs.” The recognition of hyperattenuating signs is important because they allow serious acute vascular diseases to be identified even at unenhanced CT and can represent a unique finding that indicates an acute state. However, this fact is not well known.

At some or even many institutions, unenhanced CT might not be performed when contrast materials are used. Nevertheless, unenhanced CT plays an important role in many situations, including (a) the detection of hemorrhage and calcification; (b) the clarification of whether an object has an enhancement effect, since this is obscure if only contrast material–enhanced CT is performed (3–5); and (c) the detection of hyperattenuating signs as described in this article. On the other hand, in certain cases only unenhanced CT is performed, such as (a) in patients with a history of allergy to iodinated contrast material, renal dysfunction, or ambiguous symptoms without suspicion for vascular diseases, and (b) in extreme emergency situations (6).

In this article, we discuss and illustrate various hyperattenuating signs seen in a variety of vascular diseases at unenhanced CT, including acute arterial occlusion, acute arterial dissection, aneurysm rupture, and acute venous thrombosis. In addition, we address potential mimics of hyperattenuating signs. We also discuss the mechanism responsible for hyperattenuating signs and the preferred method for evaluating these signs at CT.

Mechanism of a Hyperattenuating Sign

Hyperattenuating signs indicate fresh clots caused by vessel occlusion, mainly thrombi and emboli (6). The CT attenuation value of normal blood is hematocrit dependent and ranges from 20 to 30 HU (7). As a thrombus retracts, its water content decreases, increasing the concentration of hemoglobin and subsequently raising the attenuation value of the thrombus to 50–80 HU (7,8). The attenuation of clots decreases gradually over about 7 days, at which point they cannot be differentiated from blood or may even be hypattenuating relative to blood. This phenomenon is similar to that observed in extraluminal hematoma such as a hemorrhage in the brain parenchyma and an extraaxial space (6). However, hyperattenuating signs are less distinct than hemorrhages, and the duration of the increased attenuation of a hyperattenuating sign tends to be shorter than that of a hemorrhage.

CT Evaluation

When contrast-enhanced CT is performed, unenhanced CT is sometimes omitted so as to (for example) save time or avoid radiation exposure and is perceived as providing only limited information. In terms of saving time, the inclusion of unenhanced CT is now less problematic because of the dramatic reduction in acquisition time with multidetector CT. As mentioned earlier, unenhanced CT is useful for many purposes, including the detection of hyperattenuating signs. Therefore, unenhanced CT is especially recommended for emergent diseases.

The use of contrast-enhanced CT alone to detect hyperattenuating signs is problematic because a slight increase in attenuation is obscure relative to the normal strong intraluminal enhancement. As a result, it becomes difficult to distinguish hyperattenuating findings from chronic clots, which typically show low attenuation. In contrast, in
some cases in which enhancement is poor and scanning is ill timed, filling defects caused by acute clots are indistinct at contrast-enhanced CT because the slightly increased attenuation appears similar to normal weak intraluminal enhancement. If a hyperattenuating sign is suspected at unenhanced CT, contrast-enhanced CT is needed to determine whether a clot is present. Conversely, if a clot is detected at contrast-enhanced CT, unenhanced CT findings should be reviewed to confirm whether it is an acute clot by checking for the presence of hyperattenuating signs.

The magnitude of the attenuation increase in hyperattenuating signs is usually small. The development of hyperattenuating signs depends on the age and size of the clot, which in turn reflects the size of the vessel itself. For these reasons, it is often difficult to recognize hyperattenuating signs. Therefore, comparison with the contralateral side and adjacent regions is recommended. Use of thin sections and multiplanar reformatted images, or of a narrower window width to emphasize the contrast of the subtle increase in attenuation, is also useful in identifying hyperattenuating signs.
Acute Arterial Occlusion

Cerebral Infarction
As stated earlier, the hyperattenuating MCA sign is one of the earliest CT signs of acute cerebral infarction (Fig 1) (1,2). The hyperattenuating component of this sign represents occlusion of the MCA by an intraluminal clot from a thrombus or embolus (1,2). The hyperattenuating MCA sign can theoretically be seen at the time of vascular occlusion, before other changes associated with acute stroke (eg, loss of the insular ribbon, hemispheric sulcal effacement) are manifested (Fig 1a) (1,2). The hyperattenuating MCA sign is well known because most strokes occur in this region, and a sign in the proximal MCA is more easily detected because of the location and size of this vessel. However, similar hyperattenuating signs can be observed, albeit infrequently, in other cerebral arteries. For example, occlusion of the peripheral MCA causes an increase in the spotted attenuation in the sylvian fissure (“hyperattenuating MCA dot sign”) (Fig 2) (9,10). Occlusion of the basilar artery or posterior cerebral artery can also cause what are known as the “hyperattenuating basilar artery sign” and the “hyperattenuating posterior cerebral artery sign,” respectively (Fig 3) (9,11).
include increased attenuation of the SMA at unenhanced CT (Fig 4a) and increased attenuation of the fat surrounding the SMA. Decreased bowel wall enhancement, bowel wall thickening, and bowel dilatation represent ischemic changes in the intestine (12).

**Superior Mesenteric Artery Occlusion**

Acute mesenteric artery occlusion can cause severe mesenteric ischemia (12). The etiology of acute mesenteric artery occlusion consists of embolism and thrombosis (12). Acute emboli generally lodge at the bifurcation of the middle colic artery and the superior mesenteric artery (SMA) (12). Thrombosis more often involves the proximal SMA. Findings at contrast-enhanced CT that indicate occlusion of the SMA are a contrast material filling defect or ringlike enhancement of the SMA (Fig 4) (12). Important findings related to acute SMA occlusion include increased attenuation of the SMA at unenhanced CT (Fig 4a) and increased attenuation of the fat surrounding the SMA. Decreased bowel wall enhancement, bowel wall thickening, and bowel dilatation represent ischemic changes in the intestine (12).

**Peripheral Artery Occlusion**

The etiology of acute peripheral artery occlusion also consists of embolism and thrombosis (13). Embolism is more likely to produce sudden symptoms and severe, limb-threatening ischemia owing to the lack of preexisting collateral circulation that is often produced in thrombosis (13). In neither case does the occluded region enhance at contrast-enhanced CT (Fig 5). However, it is difficult to
Figure 7. Aortic dissection with an early thrombosed false lumen in a 75-year-old man with lumbar back pain. (a) Unenhanced CT scan obtained 1 day after onset shows a thrombosed false lumen with increased attenuation (arrowhead). (b) On a contrast-enhanced CT scan, the increased attenuation is obscure (arrowhead).

distinguish between acute and chronic occlusion when only contrast-enhanced CT is performed. An increase in the attenuation of the occluded region is sometimes observed at unenhanced CT and indicates acute occlusion (Fig 5a).

Pulmonary Thromboembolism
Acute pulmonary thromboembolism is a life-threatening condition. Most causes of emboli derive from deep venous thrombosis. The most reliable criterion for the diagnosis of acute pulmonary thromboembolism is the visualization of an intraluminal filling defect at contrast-enhanced CT (Fig 6). In some cases, however, especially with large emboli, unenhanced CT can depict hyperattenuating emboli at the central site of the pulmonary artery (Fig 6a) (6). Awareness of this sign is important because, even when acute pulmonary thromboembolism is not clinically suspected, it may be identified at unenhanced CT, and such detection can help guide further imaging and allow the timely initiation of appropriate therapy (6).
Acute Arterial Dissection

Aortic Dissection with an Early Thrombosed False Lumen
Controversy exists regarding the pathogenesis of intramural hematoma of the aorta, which has commonly been defined as dissection without an intimal tear (14). However, recent studies have reported intimal defects in many cases of intramural hematoma (14–16). Therefore, aortic dissection with an early thrombosed false lumen is the more appropriate term. An early thrombosed false lumen manifests at unenhanced CT as a crescent-shaped high-attenuation area in the aortic wall, a finding that reflects acute clots (Fig 7) (17) and may be the only sign to indicate an acute state. The false lumen later becomes thinner, and its attenuation gradually decreases. Some reports have indicated that this type of aortic dissection may have a better clinical outcome than the typical type (17,18).

SMA Dissection
Spontaneous dissection of a visceral artery is a very rare condition, with the SMA being the most frequently affected visceral artery (12). Cystic medial necrosis and fibromuscular dysplasia have been shown to be etiologic factors (12). Findings of SMA dissection include enlargement of the diameter of the SMA and narrowing of the true lumen, which is surrounded by the dissected false lumen at contrast-enhanced CT (Fig 8). In cases of SMA dissection with an early thrombosed false lumen, increased attenuation of the false lumen at unenhanced CT indicates an acute state (Fig 8a). This finding is similar to that seen in aortic dissection with an early thrombosed false lumen. Increased attenuation of the fat plane around the SMA also indicates an acute state.

Vertebrobasilar Artery Dissection
Vertebral artery dissection is increasingly being recognized as a cause of posterior circulation stroke in young and middle-aged adults (19). Vertebral artery dissection can be classified into aneurysmal and steno-occlusive types according to its morphologic features at angiography (20). In the steno-occlusive type, a narrowed centric or eccentric lumen surrounded by crescent-shaped mural thickening and an abrupt or tapered occlusive lumen are observed at CT angiography (19). Hyperattenuating signs are infrequently observed at unenhanced CT and are similar to those seen in aortic dissection with an early thrombosed false lumen (Fig 9). In most cases, however, any hyperattenuating sign is obscured because the diameter of the vertebral artery is thinned at the skull base, an area with extensive beam-hardening artifacts from bones (21).
Aortic Aneurysm Rupture

It is imperative that a ruptured aortic aneurysm be diagnosed promptly because it constitutes an extreme emergency and has a high mortality rate. The CT findings are straightforward and include extraluminal hematoma and extravasation of contrast material (22). There is usually a delay of several hours between the initial intramural hemorrhage and frank extravasation (22). Imaging features suggestive of instability or impending rupture include a low thrombus-to-lumen size ratio and an increased aneurysm size (22). Another notable sign is the “hyperattenuating crescent sign,” which consists of a well-defined peripheral crescent of increased attenuation within the thrombus of an aneurysm at unenhanced CT (Fig 10) (22–24). This finding represents an internal dissection of blood into either the peripheral thrombus or the aneurysm wall, a process that causes or perhaps results from a loss of the ability of the thrombus to protect the aneurysm from rupture (22,24,25). Although the hyperat-
The hyperattenuating crescent sign is not always predictive of rupture, it is one of the earliest and most specific imaging manifestations of the rupture process (22–25). Such findings indicate that the aneurysm is at risk for rupture, and prompt surgical consultation should be sought (23). The hyperattenuating crescent sign is most common in the abdominal aorta but can also be observed in the thoracic aorta and other arteries.

**Acute Venous Thrombosis**

**Superior Mesenteric Vein Thrombosis**

Superior mesenteric vein (SMV) thrombosis is an uncommon but potentially life-threatening disorder (26). There are numerous causes of SMV thrombosis, including blood dyscrasia associated with hypercoagulability, surgery, trauma, portal hypertension, infection, neoplasms, and the use of oral contraceptive pills (12,26). Peripheral thrombi are more likely than proximal thrombi to cause small bowel infarctions by preventing collateral flow (12). Filling defects in an SMV with an enlarged diameter are observed at contrast-enhanced CT (Fig 11) (12). Unenhanced CT findings indicating an acute state include swelling of the SMV and increased attenuation of the SMV (Fig 11a, 11b) and the surrounding fat (12). Gross edema of the small bowel wall and uniform homogeneous water accumulation within the mesentery may also be seen (12).

**Figure 11.** SMV thrombosis in a 61-year-old man with acute abdominal pain and back pain. (a, b) Unenhanced CT scans obtained at different levels 3 days after onset show a swollen SMV and its branches with slightly increased attenuation (arrowheads). Increased attenuation of the surrounding mesenteric fat is also observed. (c, d) Corresponding contrast-enhanced CT scans reveal filling defects (arrowheads).
Figure 12. Deep venous thrombosis in an 83-year-old woman with pain and swelling of the right lower limb. (a) Unenhanced CT scan obtained 5 days after onset shows increased attenuation of the deep vein of the right lower limb (arrowhead). (b) Contrast-enhanced CT scan reveals a filling defect, but the defect is relatively indistinct because of the increased attenuation (arrowhead).

Figure 13. Cerebral venous thrombosis in a 44-year-old man with left upper limb and neck pain. (a, b) Unenhanced CT scans obtained at different levels 1 day after onset show increased attenuation of the transverse sinus, great vein of Galen, straight sinus, and superior sagittal sinus (arrowheads). (c) Lateral venous phase angiogram obtained after injection of the left internal carotid artery reveals filling defects in the superior sagittal sinus and great vein of Galen (arrowheads).

Portal Vein Thrombosis
There are numerous causes of portal vein thrombosis, including liver cirrhosis, infections such as cholangitis and pancreatitis, neoplasms, hypercoagulable states, surgery, and SMV thrombosis (27–29). Signs and symptoms of portal vein thrombosis may be subtle or nonspecific and are often overshadowed by an underlying illness (28,29). The direct finding of portal vein thrombosis is a filling defect in the portal vein with rim enhancement of the vessel wall at contrast-enhanced CT (27,29). Indirect findings include the presence of portosystemic collateral vessels, cavernous transformation, and arterioportal shunts, all of which are usually observed in the chronic
state (27,29). The only CT finding that indicates an acute state is increased attenuation of the portal vein at unenhanced CT (27,30). However, hyperattenuating signs occur only infrequently, since CT is not usually performed during the acute state because of the subtle symptoms (30).

Deep Venous Thrombosis
Deep venous thrombosis is the major causal factor in acute pulmonary thromboembolism. The specific CT finding of deep venous thrombosis is the presence of an intravenous filling defect at contrast-enhanced CT (Fig 12) (31,32). Findings that indicate an acute state are an increase in the caliber and attenuation of the veins at unenhanced CT (Fig 12a). A possible reason that hyperattenuating signs for deep venous thrombosis are frequently found is that the symptoms of swelling and pain of the involved leg prompt CT examination during the acute phase. These findings can be observed from the inferior vena cava to the veins of the lower legs (31,32). In cases of insufficient venous contrast enhancement, inappropriate scanning timing, and heterogeneous venous flow, filling defects are likely to become indistinct during the acute state at contrast-enhanced CT because of the increased attenuation of the thrombi (31).

Cerebral Venous Thrombosis
Cerebral venous thrombosis is a relatively uncommon but serious neurologic disorder that is potentially reversible with prompt diagnosis and appropriate medical care (33). Because the possible causal factors and clinical manifestations of this disorder are many and varied, imaging plays a crucial role in the diagnosis (33,34). Contrast-enhanced CT and MR imaging can depict filling defects in an occluded sinus. Unenhanced MR imaging can help detect the absence of a flow void and the presence of altered signal intensity in the sinus (33). Unenhanced CT can also indicate the presence of cerebral venous thrombosis by showing an increase in the attenuation of the occluded sinus (Fig 13). However, hyperattenuating signs are seen in only 25% of cases of cerebral venous thrombosis (35) because the tentorium and beam-hardening artifacts from bones obscure these signs. The hyperattenuating sign caused by cerebral venous thrombosis is similar to that caused by a subjacent subarachnoid hemorrhage or a subdural hemorrhage (33).

Bowel Obstruction and Volvulus
In cases of small bowel obstruction and colon volvulus, the “whirl sign,” which is created by swirling strands within the mesenteric fat caused by twisted bowel loops and branching mesenteric vessels, is observed along with bowel enlargement (36). In cases of closed-loop obstruction with intestinal ischemia, absence of bowel wall enhancement is seen in the affected bowel loops (36). It can be accompanied by enlargement of the small mesenteric veins in the whirl sign caused by congestion (37). If the bowel loops are tightly twisted, venous congestion can progress to a venous thrombus (Fig 14). Furthermore, if arterial flow is interrupted, a thrombus can form in the involved arteries (Fig 15). These events can cause hyperattenuating signs within the whirl sign (Figs 14a, 15a).
**Mimics of a Hyperattenuating Sign**

**Stones and Calcification**

Stones and mural or perivascular calcification can mimic hyperattenuating signs (Fig 16) but can usually be differentiated from a hyperattenuating sign in that its attenuation is typically higher than that of a thrombus.

**Beam-hardening Artifacts**

Beam-hardening artifacts from bones, such as those at the skull base, can increase the attenuation of the surrounding tissues, including vertebrobasilar arteries and the venous sinuses (21), thereby mimicking hyperattenuating signs (Fig 17). In such cases, one should be aware of the potential for misdiagnosis.

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**Figure 15.** Volvulus of the sigmoid colon in a 76-year-old man with abdominal pain. (a) Unenhanced CT scan obtained 3 hours after onset shows slightly increased attenuation of the artery and vein in the sigmoid colon mesentery (arrowheads). (b) On a contrast-enhanced CT scan, the artery and vein are not enhanced (arrowheads), unlike the other vessels. The patient underwent surgical resection of the infarcted sigmoid colon.

**Figure 16.** Calcification mimicking a hyperattenuating sign in a 72-year-old man. Unenhanced CT scan shows calcification mimicking a hyperattenuating sign (arrowhead). Calcification can be differentiated from a hyperattenuating sign in that its attenuation is typically higher than that of a thrombus.

**Figure 17.** Beam-hardening artifacts mimicking hyperattenuating signs in an 81-year-old man. Unenhanced CT scan shows beam-hardening artifacts mimicking hyperattenuating signs (arrowhead). The attenuation of the vertebral arteries is increased by beam-hardening artifacts from the surrounding thick bones in the skull base. In such areas, one should be aware of the potential for misdiagnosis.
Aortic Pulsation

In the thoracic region, artifacts from cardiac and aortic pulsation can also mimic hyperattenuating signs. Aortic pulsation due to aortic wall motion is often observed at the proximal ascending aorta, especially at the left anterior and right posterior aspects of the aortic circumference (38). Aortic pulsation occasionally manifests with high attenuation and appears similar to hyperattenuating signs (Fig 18), although it usually manifests as low-attenuation curvilinear areas. It can be recognized on the basis of its characteristic location and its restriction to only one or two adjacent sections (38). This artifact can be reduced by shortening the scanning time or implementing electrocardiographic triggering.

Hemorrhage

Hemorrhagic attenuation is similar to a hyperattenuating sign. Thus, for instance, subarachnoid hemorrhage sometimes has the appearance of a hyperattenuating sign because of its similar location relative to the cerebral vessels.

Contrast Enhancement

After contrast material injection, there is a transient increase in the attenuation of vessels. In particular, the attenuation of the cerebral vessels can be relatively high compared with that of brain tissue, which shows little enhancement because of the blood-brain barrier (Fig 19).

Low Hematocrit

The attenuation of blood is decreased in patients with a low hematocrit (39). Accordingly, the attenuation of the arterial wall appears to be high compared with that of the blood, thereby mimicking a hyperattenuating sign (Fig 20). This
condition can be differentiated from a hyperattenuating sign in that the relatively hyperattenuating walls are very thin, homogeneous, and circumferential and are observed throughout the body.

Conclusions

Hyperattenuating signs can be observed at unenhanced CT in various vessels when an acute clot has formed in the vessel. Recognition of these signs is important because it can help identify serious acute vascular diseases even at unenhanced CT.

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References

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