Case Report

A case of post angiography subdural collection contrast enhancement: Time course of attenuation reduction

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Abstract

The leakage of contrast material into the subdural space following intra-arterial or intravenous administration can present as hyperattenuating subdural collections on noncontrast head computed tomography (CT) scan, mimicking subdural hematomas. Such a finding can potentially initiate erroneous intervention or hinder thromboprophylaxis treatment. We report the time course of attenuation changes in enhancing subdural collections of a patient with suspected stroke following percutaneous coronary intervention. The patient had simple fluid attenuation subdural collections (hygromas) on preprocedure head CT scan, which showed gradually increasing attenuation on 2- and 10-hours post angiography CT scans. On delayed follow-up head CT scan, at 24 and 31 hours after the percutaneous coronary intervention, the subdural collection attenuation returned to preprocedural levels. In this patient, findings on an MRI obtained 9 hours after the procedure, were not in favor of a subdural hematoma. This case highlights the likelihood of contrast leakage into subdural space, mimicking extra-axial hemorrhage on head CT scans, and the time needed for normalization of subdural collection attenuation.

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Case report

A 91-year-old male with past medical history of sick sinus syndrome and left bundle branch block presented to the emergency department with syncope. An initial head computed tomography (CT) scan showed no acute intracranial hemorrhage, but the prominence of the bilateral extra-axial Cerebrospinal fluid (CSF) spaces was noted—most likely secondary to parenchymal atrophy (Fig. 1a).

The patient had increased serum troponin levels as well as reduced lateral cardiac wall mobility on echocardiography. Thus, he underwent cardiac catheterization, and a drug-eluting stent was placed in the obtuse marginal coronary artery. After the percutaneous coronary intervention (PCI), the nursing staff found the patient to be confused and dysarthric,

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Fig. 1 – Axial (left column) and coronal (right column) slices of head CT scans obtained (a) 46 hours before, and (b) 2-, (c) 10-, (d) 24-, and (e) 31- hours after the percutaneous coronary intervention. On initial preprocedural head CT (a), the iso-attenuating right frontal parafalcine subdural collection (arrow) was not conspicuously visualized and perceived as new subdural hemorrhage on postprocedural head CT (arrow, b). The subdural collection average Hounsfield Unit attenuation, and thickness are summarized in Fig. 2, and Table 1, respectively.

Table 1 – Thickness of subdural collections in head CT scans obtained before and after the percutaneous coronary intervention. The subdural collections along the cerebral convexity slightly increased in thickness after the procedure, which could be in part due to change in hydration status of the patient.

<table>
<thead>
<tr>
<th>Subdural collection thickness (mm)</th>
<th>46 h pre</th>
<th>2 h post</th>
<th>10 h post</th>
<th>24 h post</th>
<th>31 h post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Along the falx cerebri</td>
<td>5.2</td>
<td>4.8</td>
<td>5.7</td>
<td>4.0</td>
<td>3.9</td>
</tr>
<tr>
<td>Left cerebral convexity</td>
<td>5.5</td>
<td>8.0</td>
<td>6.2</td>
<td>9.4</td>
<td>10.2</td>
</tr>
<tr>
<td>Right cerebral convexity</td>
<td>5.2</td>
<td>7.1</td>
<td>6.4</td>
<td>9.0</td>
<td>8.5</td>
</tr>
</tbody>
</table>

Discussion

Contrast leakage into the subdural space and enhancing subdural collections following intra-arterial or intravenous administration of contrast have been reported before [1,2]. Such a hyperdense extra-axial collection can be mistaken for a subdural hematoma—similar to present case. Besides, the use of anticoagulants during a PCI procedure can further raise clinical suspicion of intracranial hemorrhage. It is particularly important after a PCI to rule out subdural hemorrhage as it will determine whether a patient can continue anticoagulants after the procedure [3,4].
The mean Hounsfield Unit (HU) of extravasated contrast is usually higher (mean HU of 139) than of hemorrhage (mean HU of 54) [5]. In our case, the average attenuation of the subdural space before PCI was about 13-14 HU (Fig. 2), which increased to 20 HU immediately after the PCI. The average attenuation was only normalized after 24 hours from angiography (Fig. 2), although nonhemorrhagic nature of the subdural collection was already confirmed by MRI scan. The time course of attenuation changes in patients with enhancing subdural collection is vital, as follow-up CT scans are commonly ordered for equivocal findings. Thus, a radiologist should pay attention to the possible persistence of contrast enhancement in subdural collections for as long as 24 hours.

One issue contributing to the uncertainty in the presented case was the increased thickness of subdural collections in addition to hyperattenuation. Given the time course of changes, we speculate that the changes in fluid collection diameter were likely due to hydration status of the patient and fluctuation in extra-axial space. In addition, on immediate post angiography CT scan, it was suspected that the subdural collection along the falk cerebi was new since the preprocedural head CT; however, in careful examination, it becomes evident that a smaller collection with simple fluid attenuation was present on the initial CT scan which was expanded and started to enhance on postangiography scan (Fig. 1a and b).

The distinction of contrast enhancement from hemorrhage into a subdural collection is crucial to prevent any unnecessary intervention. Also, misdiagnosis of subdural hematoma in polytrauma patients who had head CT after obtaining contrast-enhanced body CT can delay initiation of thromboprophylaxis to prevent deep vein thrombosis [6]. Similarly, enhancing subdural collection mimicking intracranial hemorrhage in patients with recent cardiac or cerebrovascular catheterization can delay initiation of antithrombotic treatment [2]. In patients with recent intra-arterial or intravenous contrast administration, radiologists should not only consider contrast leakage into a hyperattenuating subdural collection but also be aware that such enhancement may persist up to 24 hours after contrast administration (Fig. 2).

Dual-energy or spectral CT scan has shown to be helpful in distinguishing a hemorrhagic product from the iodinated contrast in different forms of intracranial hemorrhage [7,8]. A recent study by Bodanapally et al. has also demonstrated the application of dual-energy head CT in the differentiation of subdural hematoma from enhancing collections in trauma cases after receiving intravenous contrast as part of the polytrauma whole body scan [6]. However, dual-energy CT scans or MRI scans may not be (readily) available in patients with suspected subdural hematomas, especially in community hospitals [9]. In these cases, short-interval follow-up CT scans are usually considered. The time course of attenuation changes in subdural collections in our case is particularly helpful in such a clinical scenario, as the radiologist should be aware of the time interval required for subdural collection enhancement to normalize. Moreover, the resorption of iodinated contrast material can take several days; however, we have described specific time points that highlight the importance of at least 24 hours window of observation before the hyperdensity normalizes on head CT scan [1].

In summary, the patient presented in this report demonstrated increased attenuation and thickness of pre-existing subdural collections following PCI, which raised the possibility of a subdural hematoma. Multiple short-term follow-up CT scans in this patient provide the time course of atten-

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**Fig. 2** – The average Hounsfield Unit (HU) attenuation of subdural collections (depicted in Fig. 1) before and after percutaneous coronary intervention (PCI).
Fig. 3 – MRI scan was performed 9 hours after the percutaneous coronary intervention, precluding subdural hematoma on (a) T1-weighted images, (b) T2-weighted images, (c) apparent diffusion coefficient map, (d) diffusion weighted images, (e) susceptibility weighted images, and (f) fluid attenuation inversion recovery (FLAIR) images. The subdural collections in this patient demonstrate the CSF signal characteristics; whereas acute phase hemorrhagic products are expected to demonstrate T1 and FLAIR hyperintensity or reduced diffusion—and blooming artifact on susceptibility weighted images in more chronic phase.