Utility of dual-energy CT in differentiating contrast extravasation from intracranial hematoma

TO THE EDITOR: We read with great interest the article by Zamora and Lin (Zamora CA, Lin DD: Enhancing subdural effusions mimicking acute subdural hematomas following angiography and endovascular procedures: report of 2 cases. J Neurosurg 123:1184–1187, November 2015). The authors reported 2 interesting cases of iodine contrast extravasation into the subdural space mimicking acute subdural hematoma after intracranial neuroendovascular procedures. We applaud the authors for bringing attention to this important phenomenon and appreciate their literature review to help readers better understand the pathophysiology of contrast extravasation.

We have encountered similar cases and found that dual-energy CT (DECT) has significant diagnostic value in differentiating between contrast and blood in the subdural space. Dual-energy CT is a relatively new imaging technology that first became commercially available in 2006. It is based on the principle that materials have different attenuations at varying energy levels, which means that materials with similar Hounsfield units at one energy level will have different Hounsfield units at another energy level. Given this intrinsic property of materials, CT images obtained at 80 and 140 kV can be processed by computer algorithms, and each voxel can be separated into brain parenchyma, iodine, and hemorrhage components. Given these components for each voxel, computer software can then generate a set of images including a single-source image (equivalent to conventional CT), a virtual nonenhanced image, and an iodine overlay image. Hemorrhage would only appear hyperdense on a virtual nonenhanced image, while contrast extravasation would only appear hyperdense on an iodine overlay image (Table 1). Multiple recent studies have demonstrated that DECT can readily differentiate hematoma from contrast extravasation with high sensitivity, specificity, and positive predictive value.

We recently encountered a case that illustrates this point nicely. Our patient presented with subarachnoid hemorrhage and had external ventricular drain placement for hydrocephalus. Subsequently, the patient underwent endovascular treatment of a left posterior communicating artery aneurysm, and postoperative CT demonstrated a

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left-sided hyperdense mass in the subdural space concerning for subdural hematoma (Fig. 1A). However, the subdural collection appeared hyperdense on an iodine overlay image (Fig. 1B) and was not hyperdense on a virtual nonenhanced image (Fig. 1C). Given these imaging characteristics on DECT, the diagnosis of contrast extravasation was made. Indeed, a follow-up CT scan the next day showed near-complete washout of the contrast material (Fig. 1D).

Dual-energy CT is an important tool in distinguishing intracranial hemorrhage from contrast extravasation, and it can help clinicians quickly reach the correct diagnosis and avoid potentially unnecessary surgical intervention.

References


Response

We thank Tan et al. for their insightful comments and for sharing a case of subdural contrast extravasation on head CT following an endovascular procedure that was very similar to ours. Dr. Tan and colleagues underscore the clinical utility and elegance of DECT (also called dual-source CT) for discriminating between 2 high-attenuating substances, blood and extravasated contrast material, whose presence has widely different clinical implications.

Like many medical centers, ours has several scanners with this capability, and dual-source acquisitions are routinely performed for CT angiography and other applications that require bone subtraction. However, dual-source imaging is generally not applied to routine head CT and is therefore not available in many cases (including the 2 cases in our report). We agree that this technique is promising for distinguishing blood from contrast material, as well as other applications, and is certainly a tool that should be taken advantage of when available. However, we also point out that if Hounsfield units within a collection markedly exceed the attenuation expected for blood, the presence of iodine can be confidently established and further imaging may not be needed for characterization. Lastly, the paucity of reports on contrast extravasation into the subdural space suggests that this event is very rare or underrecognized or both. In making a diagnosis of subdural hematoma, it is prudent to keep in mind the possibility of extravasated contrast medium from a recent examination.

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DISCLOSURE

The authors report no conflict of interest.

Cranioplasty complications and the timing of surgery

TO THE EDITOR: It is with great interest that we have read the article by Zanaty et al.3 (Zanaty M, Chalouhi N, Starke RM, et al: Complications following cranioplasty: incidence and predictors in 348 cases. J Neurosurg 123:182–188, July 2015). Cranioplasty is a relatively simple procedure from a technical standpoint, yet it could be linked with significant morbidity and mortality. Despite the fact that there is an increase in the number of patients undergoing cranioplasty, we still have a deficiency in data regarding several aspects of this procedure that may affect the outcomes.

One of these aspects is the timing of surgery. As the authors highlighted in their paper, there are contradictory data concerning this factor in the literature. However, we believe this factor needs to be analyzed according to patient subgroups and the characteristics of the inflammatory response after the injury. For example, we have noticed that early surgery seems to be associated with more bleeding complications in patients with massive middle cerebral artery (MCA) infarction. This could be attributed to a higher degree of fragility of cerebral parenchyma in this group of patients in comparison with other groups. The primary problem in this group of patients is the severely compromised blood supply. Perhaps this leads to a suboptimal inflammatory response; thus, it may be rational to expect a delayed recovery course associated with unusual tissue fragility.

Because of this increased tissue fragility, the cerebral parenchyma can be more susceptible to injury by manipulation (such as pushing the bone flap against the brain cortex or tissue during adjustment). The increased tissue fragility and the resultant greater vulnerability could amplify the risk of acute or delayed blood oozing and the...
development of intraparenchymal or superficial cortical hemorrhage. The latter can result in (subdural) hematoma, which sometimes has special characteristics (based on some observations) by relatively following the configuration of the brain surface (Fig. 1).

The cerebral infarction–induced tissue fragility can be further explained by the fact that necrotic brain injury leads to a significant inflammatory response.\textsuperscript{1,2} Initially and at the early stages of injury, the inflammatory response is destructive; later on, however, the inflammatory response becomes beneficial and critical for recovery and repair.\textsuperscript{2} It is worth mentioning that hemorrhagic transformation may indicate an even more severe inflammatory response,\textsuperscript{1,2} which could be explained by the high rate of complications observed in patients harboring this condition, as shown in the Zanaty et al. study.

It is reasonable to consider the severity of the acute inflammatory response to be proportional with the size of the infarction acutely; in the late stage, however, the size of the infarction is inversely proportional to the efficiency of the recovery phase. As a consequence, perhaps there will be a propensity in the brain parenchyma in patients with massive stroke to be more fragile than it is in other conditions treated by decompressive craniectomy because of the presence of a suboptimal and delayed recovery phase. For these reasons, we may need to consider a delay in the surgery to allow time for more recovery to take place in this group of patients. Nevertheless, getting the precise answer for this matter requires studying the difference between the patterns of the inflammatory response and the course of the recovery after different types of brain injury.

Additionally, and in the same context, we may have to re-evaluate and refine our surgical technique in performing decompressive craniectomy, particularly in patients with massive MCA infarction. For instance, there is a trend to use synthetic materials as a method to create a barrier among the muscle, dura, and brain tissue to prevent or minimize the adhesions among these structures. A lot of surgeons think that minimizing the adhesions between brain tissue and other structures (especially the temporal muscle) makes the subsequent cranioplasty procedure easier. However, this technique, at least from a theoretical point of view, could have a negative impact on the recovery process, as it may lead to further compromise of the blood supply by preventing the brain parenchyma from establishing a blood supply from the outside structures such as the temporal muscle (in the same way encountered in synangiosis procedures for moyamoya). Nonetheless, this subject deserves further investigation, as the effects of using versus not using synthetic materials as a barrier (during the decompressive craniectomy procedure) to prevent the adhesions, as well as the impact of aggressive dissection of the muscle from brain parenchyma (during the cranioplasty procedure) on the short- and long-term outcomes, are not thoroughly investigated.

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**References**

**Response**
No response was received from the authors of the original article.

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**Vascularized rotational temporal bone flap**

TO THE EDITOR: I read the article by Zeiler and Kaufmann\textsuperscript{5} with interest (Zeiler FA, Kaufmann AM: Vascularized rotational temporal bone flap for repair of anterior skull base defects: a novel operative technique. J Neurosurg 123:1312–1315, November 2015). The authors mention that they discuss a novel technique of skull base reconstruction using a vascularized flap that is based on temporalis muscle. It is surprising that the authors did not have access to standard reference databases and have ignored my several articles on the technique discussed and
on the subject. I believe such articles that do not respect published work have a negative impact on scientific literature and future publications that will be based on the study. I first reported vascularized pericranial bone flap for reconstruction of the anterior cranial fossa. In the year 1994 I reported temporalis osteomyoplastic bone flap for reconstruction of the middle fossa floor. Although posterior temporal bone flaps can be based entirely on temporalis muscle for reconstruction of anterior cranial base, stretching of the vascular pedicle due to the relatively long distance can be a deterrent. Considering this issue, we based bone flaps on pericranium. To increase the thickness of the vascular pedicle, we discussed long vascularized pedicle flaps based on temporalis muscle and its superficial and deep fascial layers. Inclusion of “subgaleal fascia” in the vascular pedicle adds to the vascularity of the flap. I also discussed deployment of multilayered temporalis muscle-based vascularized flaps for both the middle fossa and anterior cranial fossa floor. The long length, ease of harvesting and deployment, and versatility of the temporalis muscle-based flaps have not been adequately exploited. The utility of vascularized flaps in situations where the host region is of less than optimum vascularity cannot be overemphasized.

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DISCLOSURE
The author reports no conflict of interest.

References

Response
We write in response to the letter to the editor from Professor Goel regarding our recent manuscript in the Journal of Neurosurgery on the application of a rotational vascularized temporal bone flap for anterior fossa repair. Professor Goel, thank you for your comments.

We acknowledge that indeed there are contributions to the literature from Professor Goel regarding skull base repair. The articles mentioned in his letter describe a variety of different techniques, and he includes references to some review articles on the subject. Vascularized rotational bone flaps for middle fossa defects, vascularized soft-tissue flaps for a variety of skull base locations, vascularized rotational bone grafts for calvarial defects, and even anterior fossa repair via split-thickness frontal bone grafts with pericranial pedicles have been described by Professor Goel, and these works are all quoted in his letter to the editor. We do believe that these contributions are important and valuable resources for practitioners dealing with such issues surrounding skull base reconstruction. The impact of these contributions should not be minimized.

However, our particular technique, in terms of the location of the graft (posterior temporal bone), the length and method of pedicle rotation, and the location of reconstruction (anterior fossa), has not been described in the literature in this particular combination to our knowledge. We do acknowledge that descriptions from Professor Goel do include anterior fossa repair via soft-tissue flaps and split-thickness frontal bone grafts with pericranial pedicles. These accounts are, however, different from ours. Thus, we believe that our case demonstrates a novel variation that, when added to the armamentarium of techniques previously described by Professor Goel and others, will provide the practicing neurosurgeon with a skill set for the repair of a variety of skull base locations.

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