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Determining when surgery is not appropriate is just as critical as establishing when to operate for certain neurologic diseases. Since the earliest clinical description of an epidural hematoma (EDH), treatment with prompt surgical evacuation has been the standard of care for the prevention of death or neurologic morbidity. In the post-CT era, this dogma has been challenged. Recently the nonsurgical management of small asymptomatic EDH is increasingly accepted as a means of caring for patients with convexity lesions not associated with significant mass effect or midline shift. In fact, the percentage of patients managed nonsurgically in clinical series since the 1960s has progressively increased from less than 1% to more than 60% (1–5).

The rational therapeutic approach for an acute EDH is based on clinical and radiologic parameters. Clinical manifestations of neurologic deterioration, such as a decrease in the Glasgow Coma Scale score, pupilary dilatation, and hemiparesis, even in the presence of a small EDH, usually require surgical intervention. The critical role of a neuroradiologist is to guide the treatment of a patient with EDH who appears to be in good clinical condition. Favorable and unfavorable CT findings can dictate therapeutic decisions. Most studies emphasize that the thickness of the epidural hematoma, the degree of midline shift, and the presence or absence of cisternal obliteration are important prognostic factors on a CT scan (1-6). Other features erroneously associated with EDH growth include the presence of a fracture across a venous sinus or middle meningeal artery or a contralateral brain injury (5, 6).

Sullivan et al in this issue of the American Journal of Neuroradiology (page 107) explore the evolution of nonoperatively managed EDH by retrospectively reviewing 160 conservatively managed patients who were selected from a cohort of 221 patients with EDH. These selected patients were studied over a period of 5 years at a level 1 trauma center. This represents one of the largest series of patients with EDH who were managed without surgery. The investigators found that approximately one quarter of the EDHs enlarged. The mean interval between injury and EDH enlargement was 8.2 hours. In all cases the enlargement occurred within 36 hours after injury. Of the clinical parameters reviewed (revised trauma score or coagulopathy), a higher revised trauma score (based on neurologic and physiological condition) was found to predict recurrent hemorrhage. No imaging features (size, presence of fracture, contralateral brain injury, midline shift) significantly correlated with EDH enlargement. The data suggest that there was no clinically significant difference in the neurologic outcome of those patients whose hemotoma enlarged and those whose did not. While EDH enlargement was common, it did not appear to impact immediate clinical outcome when measured purely by discharge disposition. There was no long-term follow-up on the conservatively managed patients to determine late clinical deterioration.

It is noteworthy that Sullivan et al only treated patients whose EDH had an average width of less than 9 mm. This represents a very selective patient group. Bricolo et al reported no surgical mortalities and complication rates of 2% to 3% for patients in good clinical condition whose EDH was treated surgically (7). The surgical management of EDH does not specifically result in morbidity. Furthermore, Cooper et al have argued that nonsurgical management is not necessarily conservative because it requires prolonged clinical monitoring (8). Servadei et al have suggested a 15-day monitoring period for patients with EDH in an acute-care neurosurgical hospital as well as repeated follow-up CT scanning (9). The strength of the Sullivan study stems from the lack of firm consensus in the literature regarding the required length of time for conservative observation of EDH. Given that the investigators reported all recurrent hemorrhages occurred in 36 hours, conservative observation periods may certainly be shorter than those recommended in previous guidelines. Clearly, the absence of follow-up data regarding the late deterioration of patients from this study suggests it may be premature to draw conclusions about the necessary length of observation for EDH. Hamilton and Wallace described a patient who did poorly even after prompt evacuation of a delayed EDH growth (6). It is important to note that patients in good neurologic condition but with a large EDH may warrant surgery. We might question why four patients with an EDH and severe shift were managed nonoperatively in the Sullivan study.

Serial clinical examinations are extremely important in selecting patients for follow-up CT scans. Sullivan et al imply that paralyzed and sedated patients with a small EDH are less likely to incur EDH enlargement. It should be emphasized

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that the lack of an examination hampers one's ability to decide when to rescan a patient. Measurements of intracranial pressure may help guide decision making. Specific locations of EDHs, however, such as the temporal region, may be associated with a higher risk of deterioration, and a frontal lobe pressure monitor may not accurately depict elevated pressures in the middle fossa.

Sullivan et al suggest that EPH enlargement occurs frequently. When EDHs enlarge, they do so by 36 hours. Based on this information, delayed CT scans may not be necessary. Certainly, managed care organizations and insurance carriers will be delighted by the cost-effective implications of this study. This, however, is not a true cost analysis of the price one pays for missing one late deterioration and the subsequent cost to society that results. Clearly, this is inconsequential from an insurance carrier's perspective. The majority of patients with EDH will be managed appropriately. Our philosophy has been that neuroimaging is important in patient care, but we believe clinical judgment is paramount when treating asymptomatic patients who harbor a small EDH and who present within hours after their injury.

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