AJNR

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AJNR Am J Neuroradiol 1992, 13 (3) 860-862
http://www.ajnr.org/content/13/3/860.citation

Commentary

Nonoperative Management of Acute Epidural Hematoma Diagnosed by CT: The Neuroradiologist's Role

Oren Sagher,[1] Guilherme C. Ribas,[1] and John A. Jane[1]

As computerized tomography (CT) becomes commonplace in the management of head injuries, clinically silent or unsuspected intracranial pathology is recognized with increasing frequency. Contusions, subarachnoid hemorrhage, and small subdural hematomata in patients who have no neurologic deficits are not uncommon. These lesions do not usually dictate operative intervention unless clinical deterioration occurs, so that their discovery serves mainly to make the already watchful eye more vigilant. Considerably less agreement exists among neurosurgeons regarding the treatment of asymptomatic or mildly symptomatic epidural hematomas (EDH). A number of reports of conservatively managed EDH, suggests that some of these lesions may resolve spontaneously and without sequelae. On the other hand, since the surgical management of EDH is relatively simple and has a very low morbidity, many neurosurgeons recommend immediate evacuation rather than placing patients at risk for deterioration. In the present paper, Hamilton and Wallace describe and analyze their experience with EDHs in order to delineate clinical and CT criteria for conservative management of these lesions (1).

The authors conduct a retrospective analysis of 48 patients with EDH, seen at their center over the past 5 years. Eighteen of these patients were managed conservatively; the remainder underwent surgery at presentation. The two groups were compared with respect to clinical condition, size, and location of the EDH, radiographic characteristics, and the outcome of the treatment each received.

The natural history of epidural hematomas is at the center of this discussion. Epidural hematomas can be caused by a variety of mechanisms (laceration of the middle meningeal artery, venous oozing from overlying fracture, laceration of dural venous sinus), and their natural history is likely to be equally varied. When reporting the history of a conservatively managed case, therefore, it is important to have an accurate assessment of the patient's condition at presentation, the location (and proposed mechanism) of the EDH, as well as its size and the time of its discovery.

The authors present a number of interesting findings in their study; still, their conclusions can be considered only preliminary, as not all facets of the natural history are addressed. Also, the retrospective nature of this study introduces an element of bias to its conclusions. Hamilton and Wallace carefully analyze the existence and location of skull fractures in their cases, in an effort to compare the probable mechanisms of EDH formation. They find no statistical difference between the groups, suggesting that hematomas caused by laceration of the middle meningeal artery or a major venous sinus are no more likely to produce rapid deterioration than those caused by venous oozing. This finding may be a function of the retrospective nature of this study; it has not been the experience in prospective series (2).

The timing of EDH discovery is crucial, since the likelihood that a hematoma will remain silent increases the longer the lesion has been asymptomatic. The authors do not address this issue in the current series. Also, if we assume that patients were seen shortly after injury, no statement can be made regarding the natural history of subacute or chronic hematomas.

Hamilton and Wallace compare the average sizes of the EDHs carefully and, not surprisingly, find that the operated patients had larger hematomas. Since such measurements do not necessarily reflect the brain's compliance, their use in conjunction with midline shift is more informative, as in this paper. The fact that the "presence of lucent areas within the hematoma likely indicates active bleeding and suggests the need for

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Index terms: Hematoma, epidural; Neurosurgeons and neurosurgery; Commentaries

AJNR 13:860–862, May/ June, 1992 0195-6108/92/1303-0860 © American Society of Neuroradiology

rapid surgical intervention" is also very well established by the authors.

They analyze and discuss the EDH location briefly, mentioning the published trend to operate on low temporal lesions (3, 4). Hamilton and Wallace find in their study "a slightly statistically nonsignificant trend for higher rate of surgery with low temporal lesions." Any statistical analysis has to take into consideration that this finding comes from a retrospective analysis.

The higher risk of low temporal lesions has to be emphasized, as their topography in relation to the brain stem increases the danger of uncal herniation. This is stressed in series analyzing temporal lobe contusions (5–7). The fact that EDHs in this region are usually due to meningeal arterial bleeding magnifies the risk, in our opinion. Unfortunately, temporal lesions can cause brain stem compression without significant elevations in intracranial pressure (8, 9), which makes monitoring unreliable in such cases.

Although good results have been obtained and reported in a few cases of temporal EDH managed nonsurgically (10), most of the studies pertinent to this issue suggest operating on EDHs located in the temporal fossa (3, 4, 11).

Some hematomas outside the temporal fossa are also considered dangerous. Large frontobasal hematomas, for example, can be initially asymptomatic, and are particularly hazardous when they are bilateral (12). Posterior fossa EDHs are also frequently evacuated because of the risk of deterioration (11).

The only prospective study of the management of small EDHs is that of Knuckey et al (2). In this paper, a group of 22 patients with "asymptomatic," small (mean volume 12–14 cc) EDHs were managed expectantly. Seven of these patients (32%) subsequently deteriorated and required evacuation, and two patients had mild to moderate deficits following surgery, whereas all of the unoperated patients were normal at follow-up. The rather high incidence of deterioration in this study is alarming, and makes routine conservative management hard to justify.

In the present paper, one of the conservatively managed patients later deteriorated and required surgery. His EDH was smaller than average and there were no other features placing him at increased risk for deterioration. Nevertheless, 48 hours later, this patient required emergency surgery and suffered long-term disability. This poor outcome may have been avoided with early surgery, and is quite unnerving to those of us considering nonoperative management.

In analyzing the cost of the two treatment groups, one has to consider the total cost of serial CT scans, the hospitalization costs, and the length of time these patients are kept from resuming a normal lifestyle. One avoidable bad result with life-long loss of income and institutional care could make serial scans and operative treatment seem a bargain. In this paper, the relatively low number of CT scans in the conservative group (2.67) may be a function of the retrospective nature of the study. Also, the fact that surgically treated patients were hospitalized almost three times as along as observed patients (27.6 days vs 11.2 days) is largely a function of their poorer condition at presentation. It would be interesting to compare the hospitalization stays among neurologically comparable patients in both groups.

Considering previously published data as well as the present paper, we believe that the EDHs that should be considered for observation are those high-convexity hematomas that cause no significant midline shift. Those EDHs incidentally discovered 48 hours or later after head injury should also be considered for nonsurgical management.

Hamilton and Wallace stress that favorable and unfavorable prognostic factors by CT have to be analyzed both by the neuroradiologist and by the neurosurgeon. Unfortunately, however, the study also suggests that there is danger in observing even those patients with no worrisome clinical or radiologic features.

The notion that surgery is too aggressive and dangerous is no longer a reason not to operate, since advances in neurosurgery and neuroanesthesia have resulted in extremely low morbidity. Also, patients fare far better if operated on prior to the onset of significant brain compression and secondary ischemic damage. Considering the safety of EDH surgery, we strongly suggest that the doubtful cases continue to undergo evacuation until we have very well-defined criteria for nonoperative management. Such guidelines can only be established through prospective, well-controlled studies.

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