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

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Purpose: To determine whether certain patients with epidural hematomas would benefit from conservative treatment and to assess the neuroradiologist's role in decision-making. **Methods:** We reviewed the CT scan findings, clinical presentation and outcome of 48 consecutive patients with epidural hematoma managed at our institution within the past 5 years. In 18 patients, initial management was nonsurgical, and only one of these went on to require surgery due to clinical deterioration and evidence of enlargement of hematoma on CT. The remainder of these 18 did well without surgery. **Observations and Conclusions:** Clinical indicators of neurologic dysfunction (decrease in Glasgow coma scale score, pupillary dilatation, and hemiparesis) in the presence of even small epidural hematomas usually dictates the need for surgical management. The role of the neuroradiologist is most important when the patient presents in a good clinical state, when identification of both favorable and unfavorable prognostic factors on CT is essential. The initial diameter of nonsurgically managed epidural hematomas generally must be small (mean, 1.26 cm in our series, all under 1.5 cm), and midline shift should be minimal (mean, 1.8 mm in our series). The identification of lucent areas within the epidural hematoma (suggesting active bleeding), or CT evidence of uncal herniation, can be ominous and the neurosurgeon must be alerted to their presence. Even in the presence of a favorable clinical status, presence of a larger epidural hematoma with significant mass effect or central lucent areas should alert the neuroradiologist and neurosurgeon to the strong possibility of sudden neurologic deterioration, and indicate the probable need for surgical management.

Index terms: Hematoma, epidural; Hematoma, computed tomography; Neurosurgeons and neurosurgery

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Epidural hematoma (EDH) is a potentially serious, but treatable sequela of head injury. These lesions usually present with acute neurologic deterioration and require prompt surgical intervention. However, the recent neurosurgical literature indicates that selected patients with a high Glasgow coma scale score and a small hematoma may respond well to closely supervised conservative management. We present and compare 18 cases of nonsurgically managed and 30 cases of surgically treated EDH. The clinical and computed tomography (CT) criteria predicting a good outcome with nonsurgical management are discussed.

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Materials and Methods

The charts of 48 consecutive patients with CT evidence of EDH treated in our hospital over the past 5 years were reviewed, and 18 patients were found in which initial management was nonsurgical. The remainder underwent surgery as primary management. All patients were admitted to the neurosurgical service. Patients with depressed skull fracture and a tiny (<0.3 cm) associated EDH were excluded. All CT scans were completed within 24 hours of injury.

A review of initial and follow-up CTs was performed, and lesion diameter, location, mass effect as determined by midline shift, and presence of other intracranial injuries were documented. For 36 patients, the initial diagnostic CT scan was performed in our hospital on a Picker 600 or 1200 CT scanner; for 12 patients, the initial scan was performed in another hospital in our municipality. All were

of good diagnostic quality. All CT scans were initially reviewed by a neuroradiologist in consultation with the attending neurosurgeon and neurosurgical resident(s). In this retrospective review, measurements were performed using calipers and a centimeter scale devised from the scale printed on the CT images. The presence or absence of lucent areas within the EDH, and presence and location of associated skull fracture, were also noted. The total number of CT scans performed in each patient was recorded; for each subsequent scan performed on the patients managed nonoperatively, the date postinjury, lesion diameter, and midline shift were documented.

A chart review was performed and the following information obtained: patient age and sex; type of injury; initial clinical status as estimated by Glasgow coma scale (1) at time of presentation—recorded by medical or nursing staff in the chart in 24 patients (12 operated, 12 not operated), estimated from physicians' and nurses' initial notes in 24 patients (18 operated, six not operated); length of hospital stay; and long-term outcome as defined by the Glasgow outcome scale (2). Long-term patient outcome was determined whenever possible by contacting the attending physician's office, or from the discharge notes in the hospital chart if no outpatient follow-up was available. The Student's *t*-test was employed for statistical analysis.

Results

A significant difference was noted in initial diameter of the EDH between operated and nonoperated cases, as would be expected by the selection criteria used to decide upon mode of management (Table 1). The mean diameter of the EDH in nonoperated patients was 1.26 cm on initial scan, versus 2.55 cm in operated patients ($P < .0005$). The degree of midline shift on initial scan was also significantly less in the nonoperated group—1.8 mm versus 6.6 mm in the operated group ($P < .0005$). Actual signs suggesting impending uncal herniation (obliteration of basal cisterns, particularly the suprasellar cistern, and dilatation of the contralateral lateral ventricle) were seen in 13 of 30 patients taken to surgery. One of the nonoperated cases showed signs suggestive of impending herniation, but surgery was not performed.

The presence of lucency within the EDH (Table 1), considered a sign of active bleeding, was seen in two of 18 (11.1%) of nonoperated patients, and 21 of 30 (70%) of operated patients ($P < .0005$).

It has been suggested that EDH in a low temporal location may create more symptoms of mass effect for its size, due to uncal compression (3). However, in this group there was only a slight statistically nonsignificant trend for higher rate of surgery with low temporal lesions (Table 1); four of 18 nonoperated EDHs (22.2%) had a low temporal component, versus 10 of 30 (33.3%) of those surgically removed ($P > .4$).

In our series, the number of patients with associated hemorrhagic parenchymal lesions was not significantly different between the two groups: 16 of 30 operated patients also had at least one hemorrhagic parenchymal lesion, as did 9 of 18 in the nonoperated group ($P > .8$). An attempt was not made to quantitate the degree of parenchymal injury. Significant hemorrhagic parenchymal lesions would, of course, indirectly affect several parameters which were measured, such as midline shift and Glasgow coma scale scores.

A fracture was detected radiologically in 16 of 18 nonoperated patients and in 23 of 30 operated patients (Table 2). When visualized, the fracture was felt to cross the middle meningeal artery (MMA) groove in 11 of 16 patients managed nonoperatively (68.8%), and in a nearly identical percentage (65.2% or 15 of 23) of those patients managed surgically. The fracture was over the MMA or a major venous sinus in 13 of 16 (72.2%) of nonoperative patients and in 18 of 23 (78.3%) patients taken to surgery initially, also not a significant difference. Of the nine patients (two nonoperatively managed, seven operatively) in which a fracture was not seen radiologically, only one had a skull radiograph; only two of the remaining eight had bone windows filmed at the time of the initial CT.

TABLE 1: Radiologic data

	Surgical (n = 30)	Nonsurgical (n = 18)	*P* Value
EDH diameter	2.55 cm	1.26 cm	<0.0005
Midline shift	0.66 cm	0.18 cm	<0.0005
Lucent areas in EDH	21 (70%)	2 (11%)	<0.0005
Low temporal component	10 (33%)	4 (22%)	>0.4
Uncal herniation	13	1[a]	<0.0005

[a] See text.

TABLE 2: Fracture sites

	Surgical (n = 30)	Nonsurgical (n = 18)
Fracture detected[a]	23	16
Crossing MMA	15/23 (65%)	11/16 (69%)
Crossing MMA or venous sinus	18/23 (78%)	13/16 (72%)

[a] Six cases in which a fracture was not detected had neither skull films nor CT with bone windows (see text).

Pupillary changes (Table 3) were present in 12 of 30 patients who underwent surgery, and none of the nonoperated group ($P < .0005$). Contralateral hemiparesis was present in eight of the surgical group on admission, and in none of the nonoperated cases. Admission clinical status as assessed by the Glasgow coma scale (Table 3) was also significantly poorer in the operative group (mean of 9.0 in the surgical group, 13.1 in the nonsurgical group; $P < .0005$).

It has been stated that nonoperative management of small EDHs would require intensive inpatient monitoring with repeated CT follow-up to detect deterioration early, and that this would entail greater expense and risk to the patient (4). Only one of the patients initially selected for nonoperative management went on to require surgery: at 48 hours postinjury, EDH diameter increased from 1.0 to 2.5 cm, midline shift from 0 to 1.5 cm, and clinically the patient deteriorated and required intubation. Rapid surgical evacuation was performed with the patient experiencing a moderate long-term disability. In all other nonsurgically managed patients, the maximum increase in lesion diameter was 0.3 cm, with no patient suffering deterioration in mental status during the time of inpatient observation (range, 2–41 days; mean, 11.2 days).

In contrast, the majority of patients in the surgically managed group deteriorated clinically in the brief period between admission and surgery: 11 of 30 had a normal or near normal clinical exam and then deteriorated, and four of 30 had an abnormal clinical exam with subsequent further deterioration.

Nonsurgically managed cases in fact spent a significantly *shorter* period of time in hospital (Table 4): mean for the nonsurgically managed patients was 11.2 days, versus 27.6 days for operated patients ($P < .05$). Of note, five of the patients who underwent surgery required brief readmission at a later date for cranioplasty. The average number of CTs was almost equal in the two groups: 2.67 scans per patient for nonoperated patients, 2.57 for the operated patients ($P > .8$).

Mean age did not differ significantly between the two groups (34.2 for operated group, 29.5 for nonoperated group); sex distribution also showed no significant difference (males predominated in both groups—23 of 30 surgically managed patients versus 11 of 18 in the nonoperated group, $P > 25$). The type of trauma, most frequently motor vehicle accident or fall, did not differ significantly (data not shown).

Long-term patient outcome tended to be better in patients treated nonsurgically (Table 5). Only 18 of 30 (60%) of those in whom surgery was performed as initial therapy made a good recovery, compared to 17 of 18 (94.5%) of patients treated nonoperatively on admission ($P < .01$). The one patient in the latter group who sustained a moderate long-term disability was the patient who underwent a delayed evacuation of his EDH 48 hours post-admission.

Serial CT images in a typical case of nonoperatively managed EDH are shown in Figure 1; a large lesion with central lucent "swirl" indicative of active bleeding, typical of the group requiring acute surgical intervention, is seen in Figure 2.

TABLE 3: Clinical data

	Surgical (n = 30)	Nonsurgical (n = 18)	*P* Value
Mean admission GCS[a]	9.0	13.1	<0.0005
Pupillary dilatation	12	0	<0.0005
Hemiparesis	8	0	<0.0005
Deterioration post-admission	15 (50%)	1[b] (6%)	<0.0005

[a] Glasgow coma scale score, estimate of neurologic status.
[b] Patient underwent operation 48 hours after admission.

TABLE 4: Hospital data

	Surgical (n = 30)	Nonsurgical (n = 18)	*P* Value
Mean number of CTs	2.57 (range 1–6)	2.67 (range 1–6)	>0.8
Mean days in hospital	27.60 days (range 7–210)	11.20 days (range 2–41)	<0.05

TABLE 5: Long-term clinical outcome[a]

	Surgical (n = 30)	Nonsurgical (n = 18)
Good recovery	18	17
Moderate disability	6	1
Severe disability	4	0
Vegetative	0	0
Dead	1	0

[a] Glasgow outcome scale (2).

Discussion

Acute EDH is a relatively uncommon result of head injury, comprising 1.5% of treated lesions (5). Traditionally, the detection of EDH has been considered a definite indication for surgery, and many patients do indeed present with acute neu-

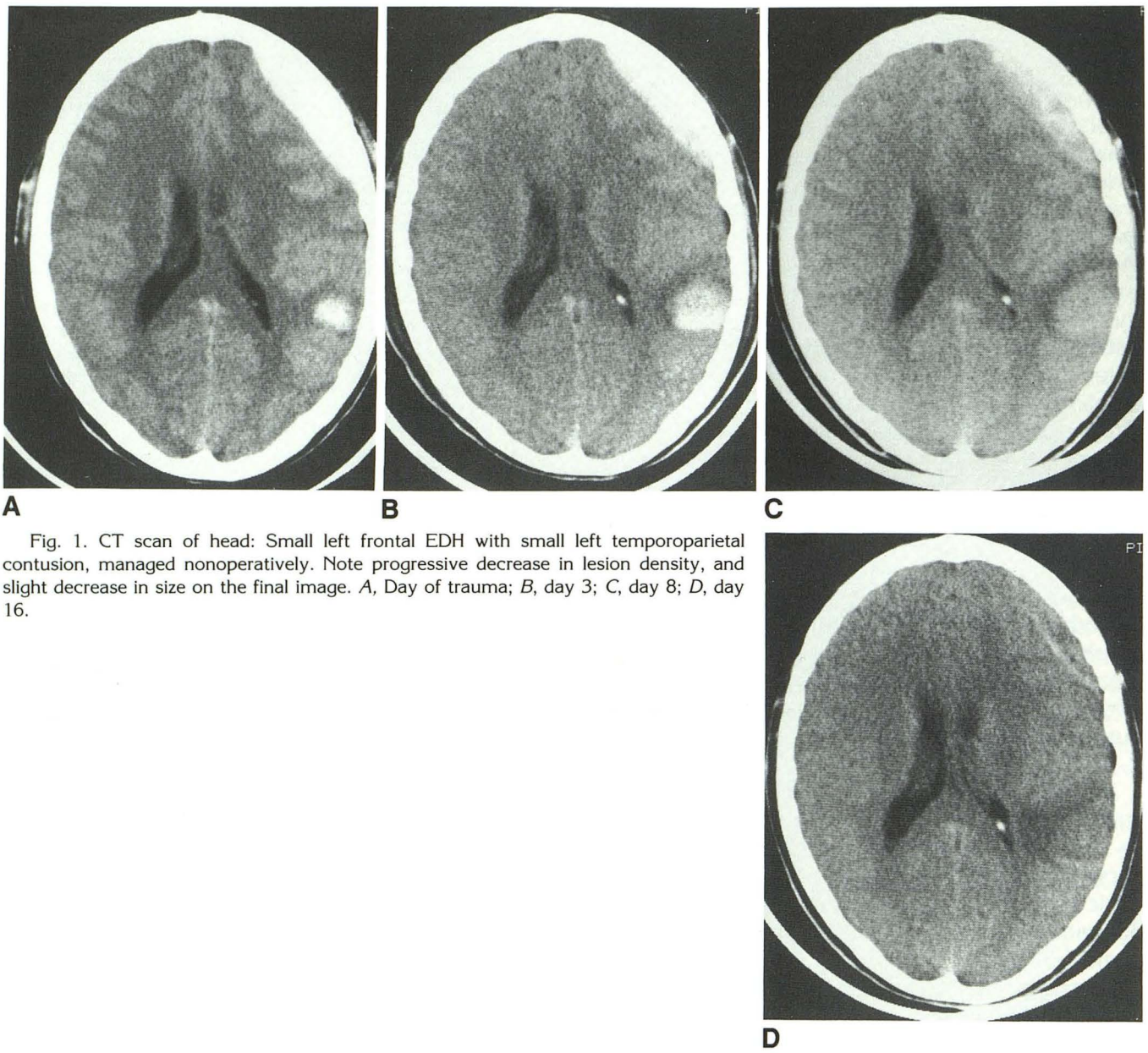

Fig. 1. CT scan of head: Small left frontal EDH with small left temporoparietal contusion, managed nonoperatively. Note progressive decrease in lesion density, and slight decrease in size on the final image. *A*, Day of trauma; *B*, day 3; *C*, day 8; *D*, day 16.

rologic deterioration requiring urgent decompression.

However, several lines of evidence suggest that once formed an EDH does not necessarily expand relentlessly. Some authors have suggested that by the time of presentation, EDH is a relatively stable lesion (3, 6, 7) having attained maximum size within minutes of injury. Clinical deterioration, when it does occur in patients managed conservatively, usually happens early (mean, 2.7 days); close observation, therefore, may be most crucial only during the first week (3). Proponents of conservative management also cite Marshall's study of 34 patients who "talked and deteriorated" after head trauma: in only four of these patients was EDH responsible (8). Since the onset of the routine use of CT for evaluation of patients with head trauma, many with EDH have been found not to exhibit the classic presentation of lucid interval followed by rapid deterioration (3, 9). Even prior to the CT era, it was recognized that some cases of acute EDH went undetected and progressed to a chronic stage. A single reported case of conservatively managed chronic EDH was reported by Brodin in 1952 (10). Several cases of chronic EDH (11, 12) were followed to resolution without surgical intervention in the late 1970s and early 1980s.

Subsequent case reports (13, 14) and patient series (3, 9, 15–18) demonstrated that selected

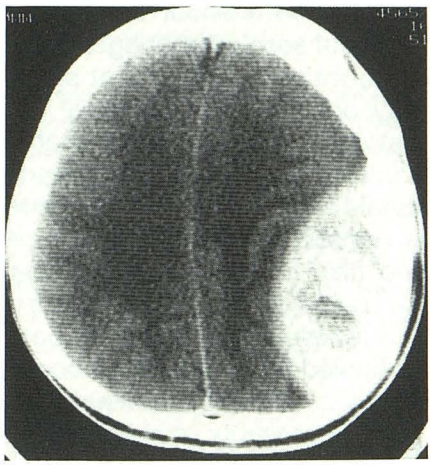

Fig. 2. CT scan of head: Large left posterior parietal EDH that required urgent evacuation. Note central lucencies in the posterior portion suggesting active bleeding.

cases of acute EDH could be safely followed to complete recovery. In Knuckey's series (17), seven of 22 patients (32%) were taken to surgery 1 to 10 days following the acute event, but only two of Pang's (15) 11 patients and none of the cases in Pozzati's (9), Servadei's (16) or Bullock's (3) series (22, 42, and 12 patients, respectively) went on to surgery. Our series also shows minimal "crossover," with only one of the 18 patients assigned initially to nonoperative management requiring subsequent surgery.

Reports of nonoperative management have, however, elicited criticism. Several authors (15–17) documented increase in size of EDHs on serial CTs, both in the first 24 hours and more gradually, up to 16 days postinjury. A case report of even greater delay (4 months) from trauma to rebleeding has been documented (19). While minor delayed expansion did not require operative intervention, the results of these series did imply that extended follow-up and inpatient monitoring is required if the nonoperative approach is chosen. This may involve both greater risk and greater expense. Several studies performed in both the pre- and post-CT era (5, 20–22) suggest that rapid evacuation of all EDHs is the only way to obtain the best possible patient outcomes; patient outcome may be compromised if surgery is delayed until the onset of clinical deterioration (23). Certainly CT has played a role in decreasing morbidity and mortality by facilitating rapid detection and evacuation of EDH (23–25), but it has also resulted in increasing detection of small, possibly asymptomatic, epidural bleeds.

The arguments outlined above have been conducted almost entirely in the neurosurgical literature, with only isolated case reports in the radiologic journals. However, the detection of small, minimally symptomatic EDH is largely due to the development of high-resolution CT, and neuroradiologists should be made aware of the controversy that exists regarding management of these lesions, and of the role of neuroradiologic findings in decision-making.

Which CT criteria are employed in making the decision to operate or observe? Clinical criteria are, of course, the most important treatment decision variables; only patients with very favorable status as rated by clinical examination are considered suitable for nonoperative management.

In our group the initial Glascow coma scale was much higher (mean, 13.1) in the nonoperated than in the operated group, which had a mean score of 9 (normal = 15). A review of the literature indicates that CT also plays an important role, not only in the initial selection of cases for nonoperative management, but also in the close monitoring of patients for early detection of increasing lesion size and mass effect.

Factors determined to be important on CT are listed in Table 1. Some authors (3, 16) consider that initial lesion size and degree of midline shift are most important in determining mode of management. Bullock et al (3) used estimates of total lesion volume (rather cumbersome for rapid assessment of images in the acute situation) to determine suitability for nonoperative management. His group recommended exclusion of lesions over 40 mL total estimated volume, causing more than 1.5 cm midline shift. In Servadei's series (16), the recommendations were that EDH diameter should not exceed 2.0 cm and that shift should be less than 0.5 mm if nonoperative management was planned. Knuckey et al (17), on the other hand, did not consider EDH size one of the most important factors, citing clinical criteria and location of the fracture in relation to the bleeding vessel as the most important prognostic factors.

Location of EDH or associated fracture is important in that it may indicate the bleeding vessel responsible. It has been suggested that EDHs with associated fracture over the middle meningeal artery or vein, or over a major sinus, may have a higher incidence of increase in size following detection (9, 17). Our results, however, do not support this (see Table 1). The one patient in our series who showed a significant increase in lesion size, requiring delayed operation, did have a fracture over the MMA; however, 10 of 18 patients

in this group demonstrated fractures over the MMA but did not deteriorate radiologically or clinically.

EDH location was also considered important by Bullock (3) and Pozzati (9), who contended that distortion of the upper brain stem with aqueductal occlusion may occur early with low temporal lesions, which, therefore, should be managed surgically. However, our results do not show a significant difference between the two groups for this factor.

Presence of lucent areas within the hematoma likely indicates active bleeding (26) and suggests the need for rapid surgical intervention. This finding was present in 23 of our cases, 21 of which were managed surgically; in both cases managed nonoperatively, the lucent area was small and poorly defined and other factors did not indicate a need for surgery. Associated lesions, particularly contusions, may contraindicate nonoperative management (15), but some authors (3, 9) consider that small contusions are not a contraindication to the conservative approach. We did not identify a significant difference in the presence of hemorrhagic parenchymal lesions between the two groups, but no attempt was made to quantitate the degree of hemorrhagic parenchymal injury apart from indirect features including its contribution to midline shift and to clinical status.

How do nonoperatively managed EDHs evolve? Follow-up CTs in several series (15, 18) have shown a slight delayed increase in lesion size, sometimes without clinical deterioration, at 5 to 16 days postinjury. This is a common finding and in fact may occur in the majority of cases (18), and should not necessarily act as an indication for surgical intervention in the absence of clinical deterioration or progressive increase in intracranial pressure. This slight delayed form of growth appears to occur because of an "expansile phase" (15) in the evolution of the clot, as seen with subdural hematomas, related to osmotic factors rather than slow rebleeding. This is always associated with a gradual decrease in density, rather than increase, as would be expected with rebleeding. Pang (15) discusses the probable mechanisms of evolution and resorption of EDH in some detail, with resorption attributed mainly to formation of neomembrane. Another potential mode of clot resorption is suggested by Kaufman (27), who suggested that some degree of decompression and clot resorption could be attributed to leakage through an overlying fracture line.

Alert patients with small EDHs generally do very well without surgical management; however, the physician must be aware that sudden deterioration may occur if a nonsurgical approach is chosen. Servadei has suggested that prolonged clinical monitoring over 15 days in an acute care neurosurgical hospital (16), with repeated follow-up CT, is required if surgery is not performed. There is no firm consensus in the literature regarding the required length of conservative observation; our results suggest that patients may safely be followed for a somewhat shorter period. As stated by Cooper (28), nonsurgical management is not necessarily conservative management.

In summary, the role of the neuroradiologist in management of EDH is maximal when the patient is minimally symptomatic. The neuroradiologist can identify both favorable prognostic features (small diameter of EDH, minimal midline shift) and unfavorable features (large EDH, central lucent areas, and evidence of significant mass effect). This information may significantly influence the neurosurgeon's treatment plan.

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