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Prospective evaluation of a clinical decision guideline to diagnose spinal epidural abscess in patients who present to the emergency department with spine pain

Clinical article

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Object. A spinal epidural abscess (SEA) is rare but potentially devastating if not diagnosed early. Unfortunately, diagnostic delays and associated neurological deficits are common. The objectives of this analysis were to explore the use of a novel clinical decision guideline to screen patients who present to the emergency department (ED) with spine pain for SEA and to determine the diagnostic test characteristics of the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) level in patients at risk for SEA.

Methods. This was a prospective, cohort analysis comparing the incidence of diagnostic delays and presence of motor deficits at the time of diagnosis before and after implementation of a novel decision guideline using risk factor assessment followed by ESR and CRP testing prior to definitive imaging. A delay was defined as either multiple ED visits or admission to a nonsurgical service without a diagnosis of SEA. A 9-month substudy was performed in all patients who presented to the ED with spine pain so that the diagnostic test characteristics of the ESR and CRP level could be defined.

Results. A total of 55 patients with an SEA in the 9-year control period and 31 patients with an SEA in the 5-year study period were identified. Diagnostic delays were observed in 46 (83.6%) of 55 patients before guideline implementation versus 3 (9.7%) of 31 after guideline implementation ($p < 0.001$). Motor deficits were present at the time of diagnosis in 45 (81.8%) of 55 patients before guideline implementation versus 6 (19.4%) of 31 after guideline implementation ($p < 0.001$). The sensitivity and specificity of ESR in patients with an SEA risk factor were 100% and 67%, respectively. The receiver operating characteristic curve analysis revealed better test characteristics for ESR (area under curve 0.96) than for CRP (area under curve 0.81).

Conclusions. A treatment guideline incorporating risk factor assessment followed by ESR and CRP testing was highly sensitive and moderately specific in identifying ED patients with SEA. A decrease in diagnostic delays and a lower incidence of motor deficits at the time of diagnosis was observed. *(DOI: 10.3171/2011.1.SPINE1091)*

Key Words • **spinal epidural abscess** • **infection** • **decision guideline** • **paraplegia** • **emergency department** • **cauda equina**

Spinal epidural abscess is uncommon but has the potential for permanent neurological deficits or even death if not diagnosed early in its course.[1–3,8,9,13,15,16] However, the early signs and symptoms are difficult to distinguish from other benign causes of back or neck pain, and waiting until manifestation of the "classic triad" of SEA—fever, spine pain, and neurological deficits—is problematic since injury to the spinal cord or nerve roots may be irreversible.[5,9,11]

A previous analysis from our institution documented diagnostic delays in three-quarters of SEA patients, and the odds of permanent neurological deficits were 6 times higher in patients with a delay to diagnosis versus those without such delays.[4] A risk factor was identified in virtually all patients with SEAs but in only one-fifth of patients who had non-SEA–related spine pain. In addition, the ESR was elevated in all cases at the time of diagnosis. These data suggest that the ESR may be a reasonable screening test prior to definitive diagnosis using MR imaging, which is expensive and may not be readily available in many EDs.

In response, we created a decision guideline to improve early diagnosis of SEA. Risk factor assessment was incorporated to increase sensitivity, while the ESR and CRP tests were included to improve specificity and avoid unnecessary diagnostic imaging. In this study, we prospectively evaluated this decision guideline in a cohort of patients who presented to the ED with spine pain. In addition, the diagnostic test characteristics of the ESR and CRP level in patients at risk for SEA were determined.

Abbreviations used in this paper: AUC = area under curve; CRP = C-reactive protein; ED = emergency department; ESR = erythrocyte sedimentation rate; ROC = receiver operating characteristic; SEA = spinal epidural abscess.

Methods

Study Design

This was a prospective, cohort analysis to evaluate

the impact of a novel clinical decision guideline. The incidence of diagnostic delays and presence of motor deficits at the time of SEA diagnosis before and after guideline implementation were determined. In addition, a 9-month substudy was performed in all patients who presented to the ED with spine pain to define the test characteristics of risk factor assessment combined with the ESR and CRP level. Approval was obtained from our institutional Human Research Protection Program.

Setting

This study was performed in an urban university ED with approximately 45,000 annual visits. Undergraduate research associates were present in the ED for up to 16 hours per day during the 9-month period of more intensive data collection. There were no institutional changes in the availability for obtaining MR imaging during the study period.

Patient Population

All patients in whom an SEA was diagnosed in the ED were identified during a 14-year time period (1992–2005) using hospital inpatient records. This included 9 years before and 5 years after implementation of a decision guideline for diagnosing an SEA. Additional data were collected on all patients in the ED with spine pain (triage chief complaint of "neck pain" or "back pain") for a 9-month period in 2003.

Intervention

A novel decision guideline was created to help identify patients with SEAs (Fig. 1). Patients in the ED with spine pain were screened for the presence of an SEA risk factor (Table 1). Urgent or emergency MR imaging was recommended for patients experiencing spine pain with either the presence of progressive neurological deficits or elevation in either the ESR or CRP level in combination with a risk factor for SEA, fever, or radicular pain.

The decision guideline was introduced to our faculty and residents through a series of education sessions. In addition, it was posted on our clinical guideline intranet website accessible from all ED computer workstations. For the 9-month substudy, a data collection tool was completed by research associates for all patients in the ED who were experiencing spine pain. This included demographic information, chief complaint, presence of SEA risk factors, vital signs, radiographic studies, and clinical course. Complete capture of eligible patients was ensured by periodic query of our electronic database of patients in the ED, searching for the chief complaints of back pain or neck pain. In addition, research associates also reminded treating physicians about the decision guideline. Although they were not regularly present during overnight shifts, the research associates performed a routine check for patients with spine pain who were treated and discharged during off hours. This included collection of all relevant study variables and interviews with treating physicians whenever possible. The vast majority of patients who presented to the ED with spine pain had an ED visit that overlapped with research associate coverage at some point.

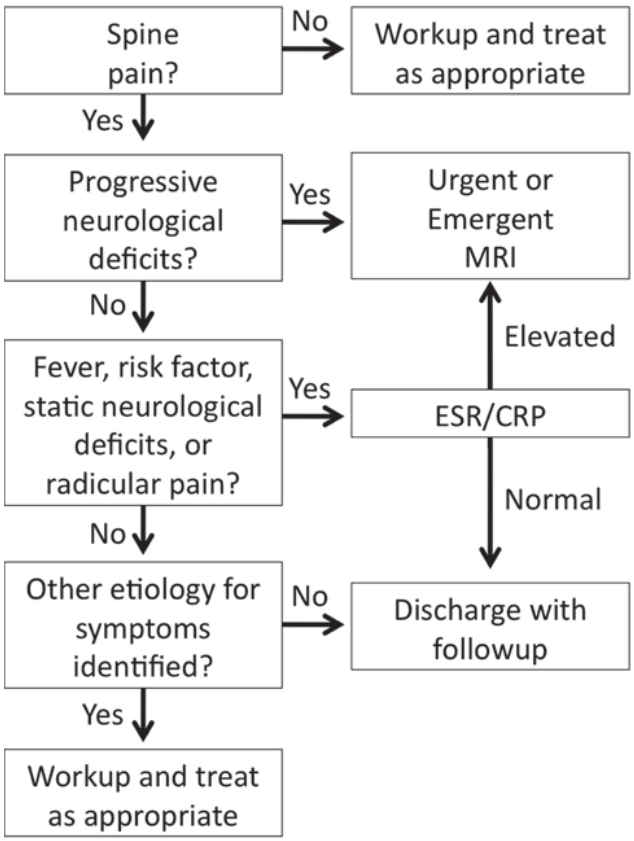

Fig. 1. Decision guideline for the ED diagnosis of spinal epidural abscess.

Data Analysis

The main objective of this analysis was to determine the effectiveness of a novel decision guideline to diagnose SEA. Patients treated in the ED who were ultimately diagnosed with an SEA during the 9-year control period were compared with those treated in the 5-year period following guideline implementation. The primary outcome measures were the incidence of diagnostic delays and the presence of motor deficits at the time of diagnosis. A diagnostic delay was defined as either multiple ED visits or admission to a nonsurgical service without a diagnosis of SEA, with subsequent diagnosis made during the admission. Physician adherence to the decision guideline was also determined.

Demographic and clinical variables were compared for patients without an SEA enrolled during the 9-month substudy and for those with an SEA over the entire 14-year study period. Differences were quantified using the t-test and ORs (95% CIs). The test characteristics of ESR and CRP level were characterized using ROC curve analysis, calculating the AUC using data from the 9-month substudy. The sensitivity of risk factor assessment combined with ESR was calculated for all patients with an SEA over the entire 14-year study period, and the specificity was determined using data from the 9-month substudy.

TABLE 1: Risk factors for SEA in patients presenting to the ED with spine pain

diabetes
intravenous drug use history
chronic liver or kidney disease
recent spine procedure or indwelling spinal hardware
recent spine fracture
indwelling vascular catheter
immunocompromised
other site of infection

Results

A total of 86 patients with an SEA were identified during the 14-year study period, including 55 patients in the 9-year control period and 31 patients in the 5-year period following guideline implementation. Diagnostic delays were observed in 46 (83.6%) of 55 patients before guideline implementation versus 3 (9.7%) of 31 after implementation of the guidelines (OR 47.7 [95% CI 11.9–191.2], $p < 0.001$). Motor deficits were present at the time of diagnosis in 45 (81.8%) of 55 patients before guideline implementation versus 6 (19.4%) of 31 after guideline implementation (OR 18.8 [95% CI 6.1–57.7], $p < 0.001$).

A total of 1019 patients who presented to the ED with back or neck pain were identified during the 9-month period of intensive data collection; 4 of these were diagnosed with SEA. Clinical and demographic data for the 1015 patients without an SEA and for all 86 patients with an SEA from the entire 14-year study period are listed in Table 2. Of note, a risk factor for SEA was identified in 86 patients (100%) with an SEA but only 237 patients (23.3%) without an SEA. Only 2% of patients with an SEA had the classic triad of fever, spine pain, and a neurological deficit at initial presentation.

Of the 1019 patients in the ED with back or neck pain identified during the 9-month substudy, the diagnostic approach in the ED was consistent with the decision guideline in 926 (90.9%). In each of the patients with an SEA risk factor in whom the ESR and CRP level were not obtained, the discharge diagnosis was "lumbar pain," "cervical pain," or "acute back pain." None of these patients returned to our ED for subsequent evaluation or were admitted to our hospital with a subsequent diagnosis of SEA. In addition, none of these patients were referred to our hospital from another facility for a missed diagnosis of SEA, which would generally occur as part of our community quality improvement mechanisms.

The ROC curve analysis revealed better test characteristics for the ESR (AUC 0.96) than for the CRP level (AUC 0.81). These data are displayed in Figs. 2 and 3. The mean ESR value for all 86 patients with an SEA was significantly higher than for those without an SEA identified during the 9-month substudy (76.5 vs 20.1 mm/hour, $p < 0.001$). The ESR was elevated (> 20 mm/hour) in 100% of patients with an SEA but in only 33% of non-SEA patients with a risk factor ($p < 0.05$). The mean CRP value for SEA patients was significantly higher than for non-SEA patients (12.4 vs 1.9, $p < 0.001$). The CRP value was elevated (> 1.0) in 87% of SEA patients but in only 50% of non-SEA patients with a risk factor (not significant). Plots of ESR and CRP values for patients with and without an SEA are displayed in Figs. 4 and 5. The sensitivity and specificity of risk factor assessment followed by ESR were 100% and 67%, respectively.

Discussion

A decision guideline incorporating risk factor assessment followed by ESR and CRP levels was sensitive and specific in identifying patients in the ED with SEA. The rate of diagnostic delays decreased from greater than 80% prior to implementation of the decision guideline to less than 10% after implementation. In addition, ESR appears to be highly sensitive and moderately specific as an intermediate screening tool in patients with spine pain who have a risk factor for SEA. The use of the CRP level, either independently or as an adjunct to the ESR, did not improve specificity.

These data are consistent with our previous retrospective analysis and support the concept that a risk factor for SEA is present in virtually all SEA patients but in less than one-quarter of non-SEA patients who present to the ED with spine pain.[4] While more patients with an SEA presented with fever or an abnormal neurological examination, the "classic triad" of fever, spine pain, and neurological deficits was rarely present in patients regardless of whether they had an SEA.

The use of risk factor assessment rather than reliance on the presence of fever or neurological deficits is more consistent with the underlying pathophysiology of SEA.[4,6,7] Bacterial infection of the vertebral column usually occurs as a result of bacteremia with "seeding" of the intervertebral disc or vertebrae, likely due to stagnation of vascular flow in these structures. Thus, a risk for bacteremia is the primary factor to consider in raising suspicion for SEA in the presence of spine pain. Alternatively, direct inoculation of the spine can occur following invasive procedures or with indwelling hardware. The development of clinical symptoms is highly variable, with an initial stage characterized by vague pain localized to the spine that can last for days to weeks. If the disease remains undiagnosed, symptoms can progress to include fever, radicular pain, neurological deficits, and complete paralysis. These latter stages appear to occur rapidly, often over a period of hours, making any decision guideline that mandates the presence of fever and neurological deficits physiologically inadvisable.[4,6,7]

While overall adherence to the decision guideline was high, it is notable that treating physicians did not follow the guideline in about one-third of patients with a risk factor for SEA. This may reflect the lack of specificity with regard to our triage complaints of back pain or neck pain. Discomfort away from midline may suggest a diagnosis unrelated to spinal pathology, although radicular pain has been described with SEA. This may also suggest that physicians use other factors in their decision to pursue additional diagnostic studies to evaluate for SEA. A history of trauma appears to dissuade physicians from or-

TABLE 2: Characteristics of all patients with spine pain who presented to the ED*

Parameter	SEA Patients	Non-SEA Patients
no. of patients	86	1015
mean age	44.8 (41.7–48.0)	44.1 (43.2–44.9)
% male	60.4 (49.9–70.3)	51.1 (48.0–51.2)
clinical		
mean temperature (°F)	98.9 (98.5–99.3)	98.1 (98.0–98.1)†
% w/ temperature ≥100.4°F	7.3 (3.0–14.4)	2.0 (1.2–3.0)†
mean SBP (mm Hg)	128 (122–134)	130 (128–131)
% w/ SBP <90 mm Hg	6.3 (1.8–15.5)	0.7 (0.3–1.4)†
HR (bpm)	94 (90–99)	85 (84–86)†
% w/ HR >100 bpm	37.5 (25.7–50.5)	16.4 (14.1–18.8)†
% w/ abnormal neurological exam	24.0 (15.8–33.7)	9.2 (7.5–11.1)†
% w/ classic triad‡	2.3 (0.3–8.1)	0.4 (0.1–1.0)
% w/ any risk factor	100.0 (96.2–100.0)	23.3 (20.7–26.0)†
DM	16.7 (9.8–25.6)	7.5 (5.9–9.3)†
IVDA	60.4 (49.9–70.3)	4.4 (3.3–5.9)†
liver disease	13.5 (7.4–22.0)	4.5 (3.3–6.0)†
renal disease	2.1 (0.3–7.3)	1.0 (0.5–1.8)
recent spine procedure/hardware	17.7 (10.7–26.8)	4.8 (3.6–6.3)†
recent spine fracture	10.4 (5.1–18.3)	1.1 (0.5–1.9)†
indwelling vascular catheter	9.4 (4.4–17.1)	0.6 (0.2–1.3)†
immunocompromised	17.7 (10.7–26.8)	3.5 (2.5–4.9)†
other site of infection	26.0 (17.6–36.0)	1.9 (1.1–2.9)†

* Data are expressed as mean values or percentages with the 95% CIs in parentheses. Abbreviations: DM = diabetes mellitus; HR = heart rate; IVDA = intravenous drug abuse; SBP = systolic blood pressure.
† $p < 0.01$.
‡ Fever (temperature ≥ 100.4°F), spine pain, and neurological deficit (%).

dering additional laboratory or radiographic studies, even in the presence of a risk factor for SEA, as evidenced by the predominance of a discharge diagnosis of lumbar or cervical pain in these patients. This may or may not be appropriate, as our previous analysis documented a history of minor trauma in 19% of patients with an SEA.

It is worth pointing out that our decision guideline does not mandate immediate MR imaging in all patients with a risk factor for SEA and an elevated ESR and CRP level. Instead, the option for emergency versus urgent MR imaging is offered. This allows the "definitive study" to be performed the following day in patients without rapid progression of symptoms or concerning neurological deficits, which may offer greater flexibility and improve the

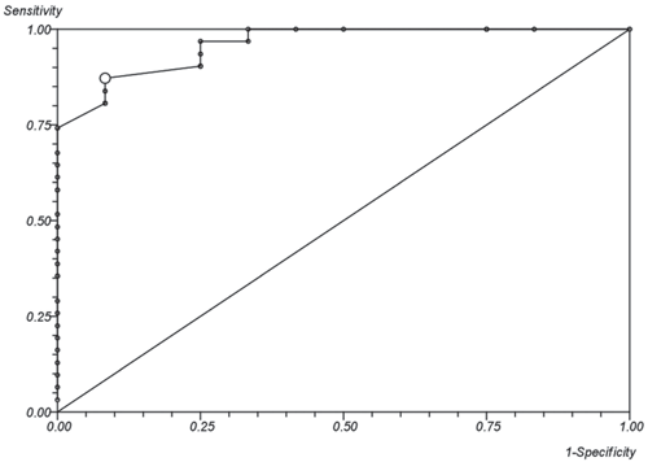

Fig. 2. Receiver operating characteristic curve to predict the presence of spinal epidural abscess for ESR. The AUC was 0.96.

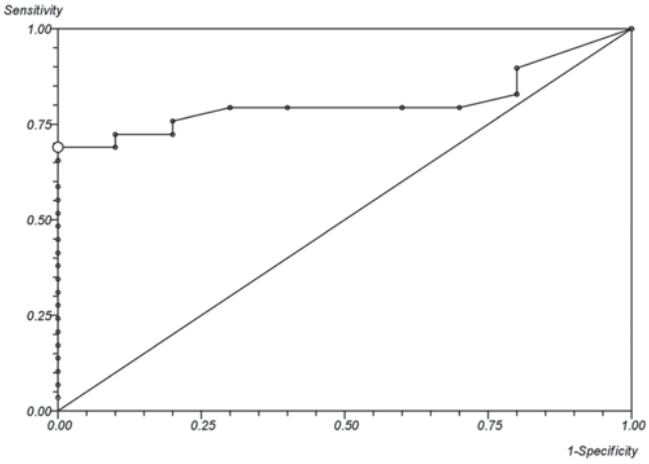

Fig. 3. Receiver operating characteristic curve to predict the presence of spinal epidural abscess for CRP. The AUC was 0.81.

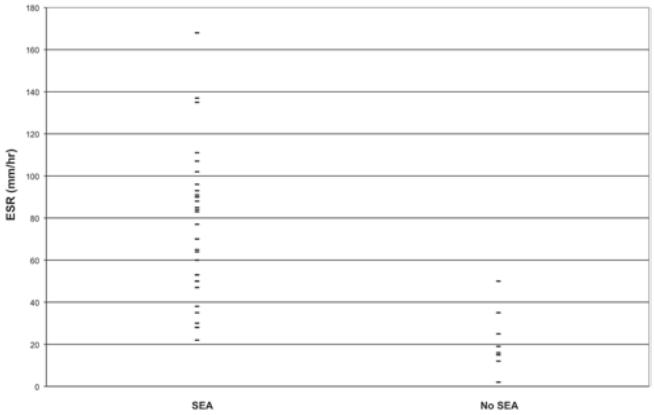

Fig. 4. Erythrocyte sedimentation rate values for patients with and those without an SEA.

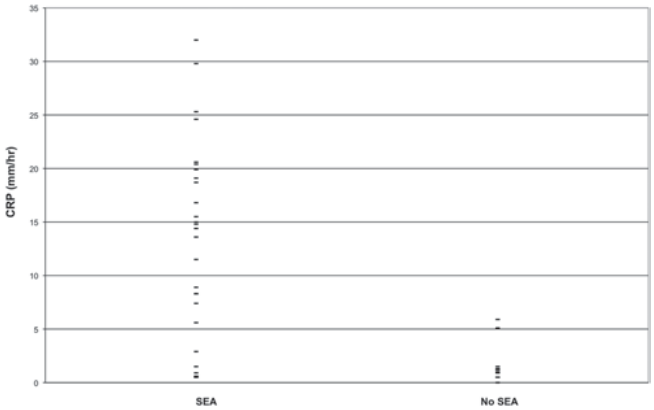

Fig. 5. C-reactive protein values for patients with and those without an SEA.

feasibility of such a protocol. Of course, before a patient can be discharged from the ED with instructions to return for MR imaging, the social situation must be considered and strict return precautions explained.

There are several limitations to this analysis that must be considered when interpreting these results. Use of historical controls raises concerns about equivalence and selection bias. We used the same data collection tool for the prospective and retrospective portions of the study to minimize missing data. In addition, the prevalence of SEA in our ED population did not change between the historical controls and the prospective group, suggesting that complete case capture had occurred.

It is notable that the algorithm was applied in 90% of patients in the ED who were experiencing spine pain. This suggests that our physicians applied additional clinical judgment beyond the decisions outlined by our guideline, which may have increased the specificities of ESR and CRP by increasing pretest probability. The most likely factor in this regard is a history of pain away from the midline or antecedent trauma, although it appears from our previous work that some patients with SEA ascribe their pain to a minor traumatic event. In addition, the presence of acute fracture with hematoma formation may increase the risk of SEA. It is also possible that the study itself or presence of research associates increased compliance with the guideline over baseline as part of a Hawthorne Effect. If the approach outlined here gains acceptance as a viable approach to patients in the ED with a potential SEA, then future efforts will appropriately focus on implementation and utilization issues.

Our patient population may not reflect that of other EDs, potentially changing the association between various risk factors and a diagnosis of SEA. However, an increased suspicion for unexplained spine pain in a patient at risk for bacteremia or direct vertebral column inoculation should become a standard consideration for emergency physicians. It is also possible that MR imaging utilization rates changed during the course of the study period, although there were no changes with regard to MR imaging availability during this time. We did not attempt to define the baseline MR imaging utilization rate during the various study periods. In addition, the study was intended to lead to more expeditious diagnosis of SEA in at-risk patients, with the anticipation that the emergency MR imaging acquisition rate would increase among patients with potential SEA.

We were also unable to definitively determine whether patients not undergoing imaging of the spine were correctly classified as patients without an SEA. Several patients with spine pain and an elevated ESR were admitted with a diagnosis of subacute bacterial endocarditis, which is an important risk factor for SEA. It is possible that the extended course of parenteral antibiotics received by these patients also treated an undiagnosed SEA.[2,10,12,14,17] Similarly, we cannot be positive that patients discharged without additional imaging were not later diagnosed with SEA at another facility. Our hospital admission database and ED electronic database of patient encounters was screened for return visits, and our quality assurance program typically would have identified a missed diagnosis of SEA.

Conclusions

A treatment guideline incorporating risk factor assessment followed by ESR and CRP testing was highly sensitive and moderately specific in identifying ED patients with an SEA. Earlier diagnosis resulted in a lower incidence of motor deficits at the time of diagnosis.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Davis, Chan, Vilke. Acquisition of data: Davis, Salazar. Analysis and interpretation of data: all authors. Drafting the article: Davis. Critically revising the article: all authors. Reviewed final version of the manuscript and approved it for submission: all authors. Statistical analysis: Davis. Study supervision: Davis.

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