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Original Contributions

THE CLINICAL PRESENTATION AND IMPACT OF DIAGNOSTIC DELAYS ON EMERGENCY DEPARTMENT PATIENTS WITH SPINAL EPIDURAL ABSCESS

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□ **Abstract—Previous reports have recommended the use of a "classic triad" of fever, spine pain, and neurologic deficits to diagnose spinal epidural abscess (SEA); however, the prognosis for complete recovery is poor once these deficits are present. This retrospective case-control study investigates the impact of diagnostic delays on outcome and explores the use of risk factor screening for early identification of SEA in a population of ED patients. Inpatients with a discharge diagnosis of SEA and a related ED visit before the admission were identified over a 10-year time period. In addition, a pool of ED patients presenting with a chief complaint of spine pain was generated; controls were hand-matched 2:1 to each SEA patient based on age and gender. Data regarding demographics, presence of risk factors, physical examination findings, laboratory and radiographic results, and clinical outcome were abstracted from medical records and entered into a database for further analysis. Patients with SEA were compared to matched controls with regard to the prevalence of risk factors and the "classic triad." We also explored the impact on outcome of diagnostic delays, defined as either: 1) multiple ED visits before diagnosis, or 2) admission without a diagnosis of SEA and >24 h to a definitive study. A total of 63 SEA patients were hand-matched to 126 controls with spine pain. Diagnostic delays were present in 75% of SEA patients. Residual motor weakness was present in 45% of these patients vs. only 13% of patients without diagnostic delays (odds ratio 5.65, 95% C.I. 1.15–27.71, $p < 0.05$). The "classic triad" of spine pain, fever, and neurologic abnormalities was present in 13% of SEA patients and 1% of controls during the initial visit ($p < 0.01$); one or more risk factors were present in 98% of SEA patients and 21% of controls ($p < 0.01$). The erythrocyte sedimentation rate (ESR) was more sensitive and specific than total white blood cell (WBC) count as a screen for SEA. In conclusion, diagnostic delays are common in patients with SEA, often leading to irreversible neurologic deficits. The use of risk factor assessment is more sensitive than the use of the classic diagnostic triad to screen ED patients with spine pain for SEA. The ESR may be a useful screening test before magnetic resonance imaging in selected patients. © 2004 Elsevier Inc.**

□ **Keywords—epidural abscess; spinal infection; cauda equina; myelopathy; diagnosis**

INTRODUCTION

Spinal epidural abscess (SEA) is a relatively uncommon but potentially devastating disease due to the high potential for permanent neurologic disability. Despite advances in diagnostic imaging modalities, antibiotic therapy, and surgical techniques, almost half of survivors are left with neurologic deficits, including 15% with paresis or complete paralysis (1). The most critical factor in preventing permanent disability is early recognition and

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treatment, as almost 90% of SEA patients are left with residual weakness when motor deficits are present at the time of diagnosis (1–3). Unfortunately, the early identification of SEA is difficult given its infrequency and the non-specific nature of early symptoms (1–8). Furthermore, the definitive diagnosis is usually made using magnetic resonance imaging (MRI) or computed tomogram (CT) myelography, both of which may be difficult to obtain on an emergent basis (1,9,10). Thus, the classic diagnostic triad of spine pain, fever, and neurologic deficits, defined with the initial descriptions of SEA, is still considered the hallmark of this disease (5,11,12).

This presents the Emergency Physician (EP) with a dilemma, as use of this "classic triad" relies on the presence of potentially irreversible neurologic deficits (5). Although previous studies have documented initial misdiagnoses in patients with SEA, there have been no previous attempts to systematically investigate the early presentation of this disease, the consequences of diagnostic delays, or the use of alternative screening strategies to achieve earlier diagnosis (2,5–8,11). This retrospective case-control study investigates the early presentation and impact of diagnostic delays on SEA and explores the use of risk factor assessment as a screening strategy in a population of Emergency Department (ED) patients presenting with spine pain.

METHODS

This was a retrospective case-control study conducted in a large, urban university hospital with approximately 45,000 ED visits each year. Waiver of consent was granted by our Investigational Review Board. Patients with SEA were identified from a computerized database maintained by our medical records department using ICD-9 discharge codes (intra-spinal abscess, 324.1) over a 10-year period from April 1992 to March 2002. We also examined medical records of patients with a diagnosis of vertebral osteomyelitis or discitis to screen for concurrent SEA. The final diagnosis of SEA was confirmed by operative reports or final MRI or CT interpretations. Patients were excluded if they had not been seen in the ED for symptoms related to SEA before the hospitalization, although admission from the ED was not required. Any ED evaluation subsequent to the onset of symptoms as defined by the patient was considered. Our primary interest was in the clinical presentation of bacterial epidural abscess. Thus, patients with fungal and tuberculous abscesses, who were anticipated to have a more indolent course, were excluded from this analysis. A pool of ED patients presenting with back or neck pain was generated using our computerized ED medical record database. Each SEA patient was hand-matched to two controls from this pool using age and gender. Data were collected in an identical manner for both the SEA cohort and the hand-matched controls.

Data were abstracted from medical records onto a standardized form by one of four investigators. The first 15 charts were reviewed by all four to establish consistency with regard to data collection and to identify data points missing from the standardized abstraction form; for the remainder of the charts, any questions regarding abstracted data were referred to the principal investigator to maintain consistency. Data were ultimately entered into an Excel® (Microsoft, Redmond, WA) spreadsheet for further analysis. Collected data included: demographics, history of present illness (presenting complaints, duration of symptoms, and number of ED and MD visits), past medical history with emphasis on a priori risk factors, physical examination (vital signs, back examination, and neurologic examination), post-void residual, laboratory results (CBC, ESR, blood and wound cultures), radiographic findings, hospital course, and neurologic status at the time of discharge. Risk factors described previously included: diabetes, intravenous drug use, liver disease, renal failure, indwelling catheter, immunocompromised status, recent invasive spinal procedure, vertebral fracture, and distant site of infection (2,4,8,12,13). The "classic triad" for SEA was defined as fever (temperature $\geq$ 38°C, 100.4°F), spine pain, and a neurologic deficit documented during the ED visit. Data were abstracted from attending and house officer ED notes and the admission note written by the inpatient service. A risk factor or neurologic abnormality was considered present if it was included on any of the notes; the attending EP note was used with any inconsistencies in documented data.

Patients with SEA were compared to controls with regard to the prevalence of risk factors as defined above and the presence of the "classic triad" for SEA. The sensitivity, specificity, positive and negative predictive value, and the likelihood ratios were also calculated. In addition, SEA patients were stratified into those with and without a diagnostic delay, defined as either: 1) multiple ED visits without a diagnosis of SEA, or 2) admission to the hospital without mention of SEA and greater than 24 h to a diagnostic study (inpatient delay), accounting for patients admitted specifically to obtain a diagnostic study on the following day. Due to the possibility that septic embolization to the spine could occur during hospitalization in a patient with endocarditis or a distant site of infection, signs or symptoms consistent with SEA (spine pain, radicular pain, or neurologic deficits) were required during the ED visit for inclusion in the inpatient delay to diagnosis group. Elopement or discharge against medical advice with subsequent presentation to the ED was not considered a diagnostic delay. For patients trans-

Table 1. Sensitivity, Specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV), and Likelihood Ratios for a Positive (LR+) and Negative (LR−) Test for the Incidence of the "Classic Triad" Versus Risk Factor Assessment in Screening ED Patients with Spine Pain for SEA

	Sensitivity	Specificity	PPV	NPV	LR+	LR−
Classic triad	7.9	99.2	83.3	68.3	10.0	0.93
Risk factors	98.4	78.6	69.7	99.0	4.6	0.02

ferred from other facilities or admitted from clinic, a diagnostic delay was defined as prior discharge from an ED without diagnosis of SEA or transfer after an ED visit without mention of SEA and a delay greater than 24 h to a diagnostic study.

Patients with and without diagnostic delays were compared with regard to residual motor weakness, defined as strength deficits documented in physician or physical therapy notes at the time of discharge that were not present before onset of symptoms, and the presence of the "classic triad." Chi-square and odds ratio calculations were used to compare SEA patients to controls with regard to the prevalence of risk factors and the presence of the "classic triad" and to compare patients with and without diagnostic delays with regard to the incidence of residual neurologic deficits. In addition, patients with a history of intravenous drug abuse were analyzed separately to investigate possible biases favoring timely vs. delayed diagnosis in this group. Descriptive statistics using range, median (25^{th}–75^{th} quartiles), and mean (95% confidence intervals) were used to report other results.

RESULTS

A total of 74 patients with SEA were identified over the 10-year study period. Nine of these had not been evaluated in the ED before diagnosis but were either admitted from an outpatient clinic or developed SEA as a complication of an orthopedic procedure before hospital discharge. A total of 65 patients were seen in the ED and ultimately diagnosed with SEA. Two charts were unavailable for review; thus, a total of 63 patients were included in this analysis. The mean duration of symptoms at the time of the first ED visit and at admission were 5 days (1–13 days) and 9 days (4–21 days), respectively. The median number of ED visits before a diagnosis of SEA was 2 (1–2 visits), with a maximum of 8 ED visits in one patient. A total of 32 patients (51%t) had two or more ED visits, and 7 patients (11%) had three or more ED visits; an additional 4 patients had visited a non-ED physician before admission. Concurrent diagnoses of osteomyelitis and discitis were made in 62% and 37% of patients, respectively. The median length of hospitalization was 21 days (9−42 days).

The 63 SEA patients were hand-matched to 126 controls based on age and gender; the median age for both groups was 46 years (41–55 years); 59% were male. A total of 98% of SEA patients had at least one of the risk factors for which we screened vs. only 21.4% of controls (odds ratio 227, 95% CI 30.1–1715, $p < 0.0001$); a total of 7.9% of SEA patients had the "classic triad" on initial presentation vs. 0.8% of controls (odds ratio 10.8, 95% CI 1.23–94.3, $p = 0.008$). Given the incidence of SEA in patients presenting to our ED with spine pain and the prevalence of risk factors in this population, approximately 50 spine pain patients with a risk factor but without SEA would need to be screened to identify one patient with SEA. The prevalence of risk factors and the "classic triad" and the calculated values for sensitivity, specificity, positive and negative predictive value, and the likelihood ratios are displayed in Table 1. The breakdown of specific risk factors for SEA patients is displayed in Table 2.

At the initial ED visit, neck or back pain was the chief complaint in 95% of SEA patients and was included in the history of present illness in all patients. In addition, 33% of patients reported a history of fever; 62% reported radicular pain; and 41% reported some neurologic deficit, including sensory loss in 25%, subjective weakness in 35%, and difficulty with urination in 22%. A history of minor trauma was offered as an explanation for the pain

Table 2. Prevalence of A Priori Risk Factors in SEA Patients

Risk Factor	Percent
Intravenous drug use	60
Immunocompromised	21
Alcohol abuse	19
Recent spine procedure	16
Distant site of infection	14
Diabetes	13
Indwelling catheter	11
Recent spine fracture	3
Chronic renal failure	3
Cancer	3
Presence of one or more of above	98

Table 3. Prevalence of Abnormal Physical Examination Findings at First ED Visit and at Time of Admission

Physical Examination Finding	First ED Visit	At Admission
Median temperature (°F)	99.0 (98.0–100.2)	98.9 (97.7–100.1)
Median heart rate (BPM)	91 (79–107)	93 (79–108)
Median systolic blood pressure (mmHg)	128 (109–145)	122 (109–145)
Fever (T ≥ 38°C, 100.4°F) at triage (% of total)	24	19
Fever (T ≥ 38°C, 100.4°F) during ED visit (% of total)	32	29
Focal spine tenderness (% of total)	52	62
Diffuse spine tenderness (% of total)	65	63
Positive straight leg raise (% of total)	13	11
Abnormal neurologic examination (% of total)	32	44
Loss of sensation (% of total)	17	27
Weakness (% of total)	29	40
Ataxia (% of total)	3	6
Abnormal reflexes (% of total)	8	17
Abnormal rectal tone (% of total)	5	10
Saddle anesthesia (% of total)	2	2
"Classic triad" w/triage temperature (% of total)	8	10
"Classic triad" w/highest ED temperature (% of total)	13	13

by 19% of patients, with 14% reporting a history of chronic back or neck pain.

The physical examination findings for SEA patients on the initial ED visit and at admission are displayed in Table 3. As noted above, the "classic triad" of spine pain, fever (temperature ≥ 38°C, 100.4°F), and neurologic deficits was present in only 8% of patients on the initial visit and 10% at admission; using the highest recorded temperature in the ED increased the prevalence of the "classic triad" to 13%. Anti-pyretic use was reported by 43% of patients. Fever was documented at triage on the first visit in 17% of these patients vs. 33% of patients not reporting anti-pyretic use ($p = 0.192$); fever was documented at some time during the initial ED visit in 35% of patients reporting anti-pyretic use vs. 63% of those not taking anti-pyretics ($p = 0.039$). The specific neurologic abnormalities and the frequency with which each component of the neurologic examination was reported for SEA patients at the first ED visit are displayed in Table 4. Laboratory and radiographic data for SEA patients are displayed in Table 5. Of note, the WBC count was elevated above 10,000 in only 60% of patients at the time of admission. Conversely, the erythrocyte sedimentation rate (ESR) was elevated above 20 mm/h in 98% of patients, but was obtained during the hospitalization once the diagnosis of SEA was considered rather than at admission in several patients. Plain radiographs were obtained in 59% of patients, with degenerative disease noted in 25%, osteomyelitis in 25%, and fracture in 5%; the identified fractures involved L5 in two patients and T11 in one patient. The definitive diagnosis was made on MRI in 76% and CT scan in 19%. The causative organisms are also displayed in Table 5. Urine cultures were positive in five patients. All five grew *Staphylococcus* species from the blood; three of these grew the same organism from a urine specimen, with *E. coli* and Klebsiella identified in the other two.

A total of 47 SEA patients (75%) met criteria for a diagnostic delay. Of these, 32 patients (68%) had multiple ED visits and 31 patients (66%) met criteria for an inpatient delay to diagnosis. Of the 31 patients with an inpatient delay to diagnosis, 22 were admitted to the Medicine service for non-specific fever or concern for endocarditis, four to the Spine service for back pain, two to the Surgery service for psoas abscess, one to Otolaryngology for retropharyngeal abscess, and one to Cardi-

Table 4. Abnormal Neurologic Examination Findings and the Incidence of Reporting in the Medical Record

Neurologic Examination Finding	% Documented in Chart	% Reported as Normal	% Reported as Abnormal
General neurologic examination	98	68	32
Sensation	67	74	26
Motor	75	62	38
Reflexes	59	86	14
Coordination	14	78	22
Rectal tone	48	90	10
Rectal sensation	19	92	8

Table 5. Laboratory and Radiographic Findings in Patients with SEA

Parameter	Result
WBC (n = 63)	
Median (K)	12.5 (8.6–16.6)
Range (K)	1.9–31.3
> 10K (%)	60
Median neutrophils (%)	77 (63–86)
Median bands (%)	2 (0–8)
ESR (n = 57)	
Median (mm/h)	77 (54–95)
Range (mm/h)	5–145
>20 mm/h (%)	98
Positive blood cultures (%) (n = 54)	57
Staphylococcus aureus (%)	50
Staphylococcus epidermidis (%)	13
Streptococcus species (%)	2
GNR (%)	6
Positive wound cultures (%) (n = 50)	98
Staphylococcus aureus (%)	78
Staphylococcus epidermidis (%)	14
Streptococcus species (%)	8
GNR (%)	4
Plain radiographs positive (%) (n = 37)	59
Osteomyelitis (%)	24
Degenerative disk disease (%)	24
Fracture (%)	8
MRI obtained (%)	76
CT scan obtained (%)	40
Diagnostic study > 24 h post-admission (%)	49

ology for chest pain. The remaining patient had been discharged from the Trauma Service after a motor vehicle collision and presented to the ED a week later with chest and back pain, leading to a diagnosis of traumatic rupture of the esophagus and mediastinitis; an epidural fluid collection was discovered during the subsequent operation several days later. Of the patients with a delay to diagnosis, 45% had residual weakness at discharge vs. only 13% of patients without a delay (odds ratio 5.7, 95% CI 1.2–27.7, $p = 0.037$).

Residual motor weakness was present in 37% of SEA patients; 91% of these had a diagnostic delay, with 57% having multiple ED visits and 61% experiencing an inpatient delay. One patient died during the admission; he was admitted on his first ED visit with a diagnosis of fever and pleurisy and suffered neurologic deterioration during the admission, leading to a definitive diagnosis by MRI. Residual left leg weakness was documented despite operative decompression. This patient ultimately died several days after his operation due to a cardiac dysrhythmia and arrest. Table 6 displays clinical and outcome data comparing SEA patients to those without a diagnostic delay. A total of 71% of all SEA patients underwent operative management. Of these, 49% had residual motor deficits; all of these had weakness documented before the operation. None of the 29% of patients managed non-operatively had residual neurologic deficits.

Patients with a history of intravenous drug abuse comprised 68% of the delay-to-diagnosis group (65% of the group with multiple ED visits, and 56% of admission delays) vs. 44% of the non-delay group (odds ratio 1.7, 95% CI 0.6–4.5, $p = 0.281$).

DISCUSSION

This study represents one of the largest case series of patients diagnosed with SEA and is unique in several aspects. First, previous investigators have not focused on the ED presentation of SEA patients. Second, the incidence and impact of diagnostic delays has not been documented previously. Lastly, an alternative strategy for screening ED patients with spine pain is explored. Although more SEA patients had the "classic triad" of spine pain, fever, and neurologic deficits than the control group, the low sensitivity and requirement for potentially irreversible neurologic deficits limits the clinical utility of this approach. Conversely, risk factor assessment offers a high sensitivity and negative predictive value and may offer a better screening strategy for identifying SEA patients before the onset of neurologic symptoms. Nevertheless, the prevalence of risk factors in the general

Table 6. Comparison Between Patients with and without Diagnostic Delay with Regard to Clinical Presentation and Neurologic Outcome

Parameter	Patients with Diagnostic Delay (n = 47)	Patient without Diagnostic Delay (n = 16)	Odds Ratio
% of all patients	75	25	N/A
Multiple ED visits (%)	68	N/A	N/A
Admission delay (%)	66	N/A	N/A
Neurologic deterioration during "delay" (%)	57	N/A	N/A
"Classic triad" present at admission (%)	9	13	0.65 (0.11–3.95)
Residual weakness at discharge (%)	45	13	5.7* (1.2–27.7)

* $p < 0.05$.

population of ED patients with spine pain and the low incidence of SEA mean that only one out of every 50 patients with a risk factor would be expected to have SEA, and performing MRI on each patient with a risk factor for SEA would not be practical.

The high incidence of diagnostic delays in SEA patients is concerning and illustrates the need for alternative screening strategies. Multiple ED visits and inpatient delays were both relatively common, and neurologic deterioration was observed in over half of these patients after the initial presentation. Furthermore, there was a significantly higher incidence of residual motor weakness in patients with diagnostic delays, including those admitted to the hospital with alternative diagnoses. Although we cannot exclude the possibility that septic embolization might have occurred during the hospitalization, our definition for diagnostic delay required that signs and symptoms of SEA be documented during the initial ED visit, even if not initially recognized as such. These data suggest that misdiagnosis of SEA is the rule rather than the exception, and that neurologic deterioration often results from these delays, including those occurring during hospitalization. This underscores the importance of considering SEA in high-risk patients and either initiating a workup in the ED or suggesting the diagnosis to the inpatient team to avoid potentially catastrophic delays.

Physical examination was not sensitive for detecting SEA in this study. Fever was present in a low percentage of patients, although repeat temperatures may increase the sensitivity. The use of anti-pyretics before the ED visit seemed to limit the use of fever in the diagnostic criteria for SEA. Interestingly, the neurologic examination was documented as normal in more than two-thirds of patients on the initial ED visit. This is consistent with our current understanding of the pathophysiology of SEA, with neurological abnormalities developing late in the course of the disease. This may also represent the performance of a cursory or incomplete physical examination, as reflected by the low percentage of charts documenting particular components of the neurological examination. This was particularly true of the digital rectal examination, which was performed on fewer than half of all patients. A more complete neurological assessment may increase the sensitivity of physical examination in detecting SEA and avoid potential delays to diagnosis.

The progression of SEA in our population was consistent with previously published reports (4,8,11,14,15). Historically, the early stages (Phases I and II) of SEA are characterized by spine pain, alone or in the presence of referred or radicular pain (8,11). After these "quiescent" phases, an "acute" stage of the disease is entered, often heralded by a rise in temperature and acceleration of pain, ultimately resulting in weakness or paralysis (Phases III and IV). Our data support the concept of SEA as a progressive disease, although the time course of symptoms is highly variable. This classification scheme has limited clinical utility, as the symptoms present in Phases I and II are relatively non-specific, with the diagnosis made most often in Phases III and IV, resulting in poor neurologic outcomes (2,8,11).

These data illustrate the need for an intermediate test to screen patients with SEA risk factors. Neither the total WBC count nor the percentage of neutrophils or bands was adequately sensitive to detect SEA. The ESR, however, was elevated in all but one SEA patient but was not obtained in many patients until the diagnosis of SEA was suspected; thus, the sensitivity of ESR early in the course of SEA is unknown. The potential utility of ESR in the diagnosis of SEA has been documented previously, and the ESR may be useful as a screening tool for ED patients with spine pain and a risk factor for SEA but without neurological deficits (2,12). Plain radiographs were not adequately sensitive to detect SEA but may still be useful due to the potential for detecting a co-existing osteomyelitis or fracture (16).

The bacteriology we observed is similar to previous studies (1). *Staphylococcus aureus* was the causative agent in the majority of patients with positive wound cultures. Other skin flora were also observed, with a low incidence of Gram-negative rods. In several patients, wound cultures were negative; however, these patients had all started empiric antibiotics for possible endocarditis, potentially sterilizing the cultures. The treatment patterns documented here reflect the trend toward non-surgical management in non-critically ill patients with normal neurologic examinations. Several recent studies have documented the safety of this approach; however, close monitoring via MRI and surgical consultation is mandatory in this select group of patients (13,17–20). This study was designed to evaluate the presentation of acute bacterial SEA and specifically excluded tuberculous and fungal abscesses. The differences between bacterial and these "atypical" infections with regard to presenting signs and symptoms and ultimate prognosis have not been defined.

There are several limitations to this analysis. The risk factor profile of our patient population affects the prevalence of disease and may not be generalizable to other locations. Nevertheless, a risk factor for bacteremia or direct seeding of the epidural space should remain the most important consideration in screening patients for SEA, whether related to intravenous drug use, a compromised immune system, or a recent spinal procedure. The presence of a distant site of infection can lead to bacteremia and seeding of the epidural space but may affect the specificity of fever or an elevated ESR in the evalu-

ation of SEA. There were no patients with a distant site of infection as the only risk factor for SEA; nevertheless, the presence of spine pain or neurologic abnormalities in a patient with another site of infection should raise concern for SEA and mandate close monitoring or further diagnostic workup.

The retrospective nature of the study also creates several limitations to this analysis. Complete data were not available for all patients, especially with regard to documentation of the neurologic examination and in the laboratory and radiographic evaluation on the initial ED visit. In each case, some minimal notation as to a "nonfocal" or "normal" neurologic examination was documented, but specific details regarding sensation, strength, reflexes, and cerebellar function were often absent; in addition, a rectal examination was not performed in all patients. The initial laboratory and radiographic evaluation was often limited until clinical suspicion for SEA was present, initiating a workup that included an ESR and MRI. In addition, this study may have underestimated the true incidence of SEA in our patient population, as patients initially evaluated in our ED may have been discharged and later admitted to another facility. Finally, we chose to focus on motor weakness at discharge as evidence of residual neurologic compromise. This does not include radicular pain or paresthesias, potentially under-representing the overall incidence of residual neurologic deficits. We anticipated that a motor examination would be present in either physician or physical therapy records in most patients during hospitalization and represented the most objective assessment of neurologic status. We were unable to determine the final degree of neurologic compromise for these patients, because post-admission documentation was often absent or incomplete.

CONCLUSIONS

The early diagnosis of SEA is important to avoid progression of disease and the development of potentially irreversible neurologic deficits. Unfortunately, diagnostic delays are common and often lead to neurologic deterioration in patients with SEA. Risk factor assessment is far more sensitive than screening for the presence of the "classic triad" of fever, spine pain, and neurologic deficits. The ESR warrants additional investigation as a potential intermediate screening tool before MRI in ED patients with spine pain and a risk factor for SEA.

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