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Emerg Med Clin N Am ■ (2006) ■–■

EMERGENCY
MEDICINE
CLINICS OF
NORTH AMERICA

Geriatric Neurologic Emergencies

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The aging of the United States population is well publicized. Projections indicate that by 2030, 55 to 65 million people over the age of 65 years will live in this country, approximately 20% of the total population [1]. Elderly patients are more likely to require emergency care and have different disease patterns with increased risk for morbidity and mortality. Familiarity with geriatrics is becoming an increasingly important component of emergency practice. Yet surveys suggest that many emergency physicians are uneasy assessing and managing geriatric patients [2].

Elderly patients are particularly prone to serious neurologic problems. Chronic diseases common in this group, such as hypertension, diabetes, atherosclerosis, and obstructive sleep apnea, increase the risk for stroke. Older patients have an increased incidence of same-level falls and pedestrian accidents, mechanisms more likely to cause head and neck injuries. Aging patients have decreased innate and specific immunity, increasing the likelihood of infectious disease.

Clinical assessment of geriatric patients can be difficult. Such patients often have complicated medical histories that they may not be able to relate secondary to dementia or acute illness. Mental status changes may be missed or underestimated in patients who have underlying cognitive dysfunction. In addition, vital signs and examination findings are less reliable in predicting the severity of illness, resulting in diagnostic delays and misdiagnoses.

Aneurysmal subarachnoid hemorrhage

The Framingham data indicate that the incidence of aneurysmal subarachnoid hemorrhage (SAH) increases from 15 per 100,000 among people from 30 to 59 years of age to approximately 78 per 100,000 among those

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0733-8627/06/$ - see front matter
doi:10.1016/j.emc.2006.01.013 *emed.theclinics.com*

aged 70 to 88 years [3]. Data from the International Cooperative Study on the Timing of Aneurysm Surgery, however, which enrolled 3,521 patients who had SAH, indicate the average age at presentation is 50 years [4].

Advanced age is an independent risk factor for death and severe disability after aneurysmal SAH. Lanzino and colleagues examined data on 906 patients from 21 neurosurgical centers to define the relationship between age, presentation, clinical course, and prognosis in SAH. Five age groups were compared: younger than 40 years, 41 to 50 years, 51 to 60 years, 61 to 70 years, and older than 70 years. Mortality rates increased from only 12% in the youngest patients to 35% in the oldest. Good outcomes defined by Glasgow Outcome Scores (GOS) [5] at 3 months were significantly less likely in aged patients, decreasing steadily from 73% in patients younger than 40 years to 25% in those older than 70 years (Fig. 1) [6]. This relationship between age and morbidity and mortality persisted even when outcomes were controlled for the severity of bleed and presence of pre-existing comorbidities. Although there is no correlation between aneurysm size or location with age, it is clear that elderly patients who have large aneurysms (>9 mm) are more likely to be disabled and dependent [7].

Clear differences in presentation and hospital course appear in geriatric patients who have SAH. Elderly patients more often present with thick subarachnoid clot and a profoundly depressed level of consciousness. The percentage of obtunded or comatose patients steadily increased from 12% in those younger than 40 years to 27% in those older than age 70 years. Elderly patients were more likely to re-bleed; rates of recurrent hemorrhage ranged

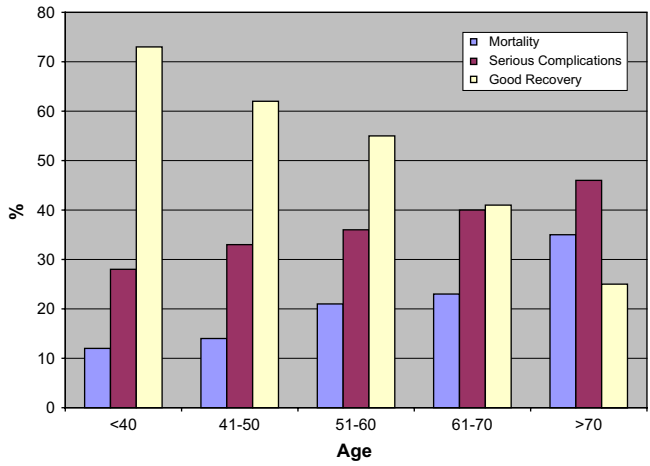

Fig. 1. Poor outcomes in patients who have aneurysmal subarachnoid hemorrhage (SAH) are related to advanced age. The rates of mortality and life-threatening complications and the likelihood of a good functional recovery by Glasgow Outcome Scale (GOS) are depicted for each age group [6].

from 4.5% in the youngest age group to 16.4% in patients older than age 70 years. The oldest patients were also more likely to develop intraventricular hemorrhage, hydrocephalus, and symptomatic vasospasm. Rates of life-threatening complications steadily increased with each decade [6].

Cardiac abnormalities, including electrocardiogram (ECG) changes, cardiac enzyme elevations, and regional wall motion abnormalities, are common in SAH. ECG abnormalities may include sinus tachycardia, ST segment elevation or depression, inverted T waves, or prolongation of the QT interval. Transient arrhythmias are common after acute bleeds, appearing in up to 9 out of 10 patients [8]. Approximately 20% to 30% of patients who have nontraumatic SAH display CK-MB and troponin spikes, usually within the first 24 hours [9]. Those patients often have some degree of left ventricular (LV) dysfunction, as evidenced by wall motion abnormalities on echocardiography.

Interpreting the clinical significance of new cardiac abnormalities in the setting of acute neurologic disease can be challenging in elderly patients who already may have underlying cardiac pathology. Ventricular dysfunction associated with SAH, although incompletely understood, does not arise from coronary occlusion. Pathology from animal models shows myofibrillar degeneration similar to that seen in states of catecholamine excess. The vast majority of patients exhibiting these deficits revert to their previous level of cardiac function within a few weeks [9]. Emergency physicians must be aware that ECG changes and cardiac enzyme elevations in such patients do not necessarily equate with ischemia; these patients are not de facto poor surgical candidates and still warrant aggressive treatment.

Advances in neurosurgical management continue to improve the overall outcome for these critically ill patients. Operative mortality has decreased drastically from more than 50% in the first Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage in 1966 to only 20% in the Cooperative Study on the Timing of Aneurysm Surgery published in 1990 [10,11]. Inagawa studied 503 SAH patients and compared outcomes for those treated in the early versus the late 1980s. He showed a twofold increase in the number of elderly patients (from 17% to 34%) and in the percentage of good functional outcomes among this patient population (from 18% to 41%) [12].

Although no randomized controlled trial (RCT) specifically addresses the benefit of definitive management over medical therapy in elderly patients who have SAH, several lines of evidence argue that outcomes with surgical or endovascular treatment are superior. First, the prognosis in conservative management is bleak, with a 5-year survival of only 20% [13]. Also, geriatric patients are far more likely to re-bleed and suffer acute complications amenable to surgical correction, such as hydrocephalus. Also, some medical management strategies, such as Triple H therapy (hypertensive, hypervolemic, and hemodilution therapy), can be pursued more safely once the aneurysm has been obliterated.

The emergency physician's role is in the rapid detection and stabilization of patients who have ruptured aneurysms. Once computed tomography (CT) scans or cerebrospinal fluid (CSF) analysis reveal the presence of subarachnoid blood, the care pathways for elderly patients are similar to those for younger patients. All require careful monitoring, assessment of the airway, seizure prophylaxis, and control of pain and nausea. Coagulopathy, frequently present in elderly patients secondary to warfarin use, should be reversed. Although severe hypertension is associated with more dire outcomes, acute lowering of blood pressure has not proven to improve consistently the clinical course of aneurysmal bleeds [14].

Considerable uncertainty exists with regard to optimal blood pressure parameters in patients who have SAH; some surgeons advocate tolerance of mean arterial pressures (MAP) less than 130 mm Hg, whereas others insist on tight control of systolic blood pressures (SBP) to less than 140 mm Hg. When antihypertensive agents are needed, the use of a short-acting and titratable agent, such as nicardipine or labetalol is preferable. Nitroprusside, although effective in decreasing systolic pressures, has several disadvantages in neurologic emergencies; it dilates cerebral vasculature, placing the patient at risk for elevations in intracranial pressure (ICP), impairs autoregulation, and may induce excessive hypotension in elderly patients [15].

Patients who have known SAH must be monitored carefully in the emergency department (ED) for signs of decompensation. Patients who have a changing neurologic examination or alterations in mental status warrant a repeat CT scan to look for signs of progression. Nonspecific changes, such as increased confusion, also may be a postictal change or the presenting symptom of cerebral vasospasm [16].

Early involvement of a skilled neurosurgical consultant is a critical component of management. Emergency physicians should have a low threshold for transfer of surgical candidates to centers with experience in aneurysm repair, because increased surgical volume correlates with significantly lower in-hospital mortality [17]. Centers that offer the option of traditional open surgical clipping and endovascular coiling techniques are preferable. The International Subarachnoid Aneurysm Trial (ISAT), which randomized 2,143 patients eligible for both techniques to clipping versus coiling, showed a distinct advantage to endovascular repair; coiled patients had a 22.6% relative risk reduction for death and dependency without a significant increase in rebleeding [18].

Emergency physicians also may be in the fortuitous position of diagnosing unruptured intracranial aneurysms. Such patients may present with symptoms such as headache, transient ischemic attack (TIA), seizure, third nerve palsy, or other evidence of mass effect. Patients who have unruptured aneurysms require neurosurgical evaluation, although management for asymptomatic lesions remains controversial [19,20]. Clinical data indicate that aneurysm repair can result in good functional outcomes even for elderly patients [21]. It is again critical to refer such patients to sites that offer open

and endovascular techniques, because coiling may confer particular advantage to patients older than 65 years of age and those who have significant comorbidities [22].

Traumatic brain injury

Elderly patients are at particular risk for traumatic brain injury (TBI). The overall incidence of TBI cases seen in emergency departments in the United States is 444 cases per 100,000 persons. The incidence increases in the elderly population and peaks at 1,026 cases per 100,000 in patients older than 85 years of age. Younger patients are 1.6 times more likely to be male, but this sexual disparity reverses in the elderly population [23].

Elderly patients have higher morbidity and mortality from head injury. Worse outcomes do not seem to be the result of therapeutic nihilism. In most applicable studies, similar percentages of younger and elderly patients received ICP monitoring and neurosurgical intervention [24,25]. Even accounting for differences in the premorbid state, outcomes remain worse in old age; something innate to the aging brain lends a particular vulnerability to neurologic insult.

The relative frequency of different injuries and mechanisms of injury differ in geriatric patients. Subdural hematomas (SDH) are far more common, accounting for 46% of TBI cases in elderly patients versus only 28% in younger cohorts [24]. Epidural hematomas are less common among elderly trauma victims (Fig. 2). Although younger trauma patients are more likely to be injured in motor vehicle collisions (MVC), the elderly population has more pedestrian accidents and falls [25].

Falls represent an enormous cause of morbidity and mortality in older adults. Those older than 65 years of age have an annual fall incidence of

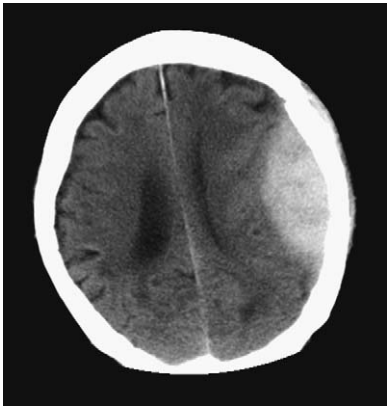

Fig. 2. Trauma is less likely to produce epidural hematomas in elderly patients.

30% and this rate increases to 50% in octogenarians [26]. This high predilection for falls is multifactorial. Normal aging adversely affects vision, joint function, and proprioception. Superimposed chronic diseases, such as diabetes, can result in autonomic dysfunction, peripheral neuropathy, and general deconditioning. Syncopal events may be associated with medication-related orthostasis or arrhythmias. Geriatric patients are not only more likely to fall, but they are also more likely to sustain serious injury when they do. Sterling noted that falls in elderly patients were seven times more likely to be the predominant etiology of injury (48% versus 7%) and seven times more likely to be the cause of death (55% versus 7.5%) than in younger patients [26].

Falls from a sitting or standing position can lead to surprisingly severe injuries in older people. Sterling and colleagues found that same-level falls resulted in serious injury in 30% of elderly persons compared with only 4% of a younger cohort. Head and neck injuries were particularly common, occurring more than twice as often (47% versus 22%) [26]. Mortality for these low falls in the elderly population approaches 15%, three times that seen in younger cohorts [27]. Paramedics and emergency physicians must be cautious with these patients and maintain a low threshold for cervical spine immobilization, imaging, and admission.

The increased number of pedestrian accidents seen in elderly patients has important implications for the nature and severity of injuries. Hui and colleagues studied elderly trauma patients admitted to the Surgical Intensive Care Unit (SICU) and compared patients injured in motor vehicle accidents with those injured in pedestrian traffic accidents. They found that pedestrian victims were significantly more likely to have subarachnoid (26% versus 9%) and subdural (29% versus 8%) bleeding. Pedestrians struck by motor vehicles had significantly greater Injury Severity Scores (ISS) [28] and higher mortality (19.6% versus 9.5%) [29].

Some researchers have argued that the threshold for scene triage and subsequent transfer to trauma centers should be lower for geriatric patients. Meldon and colleagues conducted a retrospective analysis of trauma outcomes in 455 patients older than age 80 years who were transported to a trauma center (level I or II) or an acute care hospital. Most of the deaths occurred in patients who had an ISS in the range of 21 to 45. Within that group, trauma center care conferred an enormous survival benefit (56% in trauma centers versus 8% in acute care hospitals). Using logistic regression to control for age, gender, ISS, and the presence of TBI, they demonstrated that very elderly patients are three times more likely to die at nontrauma centers [30].

Standard prehospital protocols may underestimate the severity of injury in older trauma patients. Traumatized elders are less likely to display clear hemodynamic distress. Changes in mental status may be under appreciated in patients who have underlying cognitive dysfunction. Changes in the aging brain may make standard clinical scoring mechanisms, such as the Glasgow

Coma Scale (GCS), less reliable. Low-energy mechanisms, such as same-level falls, do not commonly trigger transport to trauma centers.

Vital signs may not reveal the extent of injury in geriatric patients. In fact, up to 63% of elderly patients who have an ISS of greater than 15 and 25% who have an ISS greater than 30 did not display any of the standard hemodynamic criteria for trauma activation [31]. Tachycardia may be absent because of inherent decline in the maximum output of the cardiovascular system or secondary to cardiac medications, such as beta blockers. Blood pressure that would be considered normal in a younger person may represent significant relative hypotension in an elderly patient.

Use of GCS is a standard component of trauma evaluations but carries special import for patients who have TBI. Admission and postresuscitation GCS is correlated with survival and the ultimate degree of impairment after discharge [32], but physiologic data on the aging brain raise questions about the usefulness of GCS for elderly patients who have brain injury. Mosenthal and colleagues found significant mortality in aging adults whose sole detected injury was a minor TBI as defined by a GCS of 14 to 15 [24]. Normal age-related atrophy results in enlargement of the space between the brain and the inner table of the skull for hematoma accumulation. Significant brain injury may exist without midline shift on CT scans or clinical evidence of elevated ICP. Emergency physicians should not be falsely reassured by high GCS scores, especially in adults older than 70 years of age.

Even if prehospital protocols were optimized for geriatric trauma, several studies have shown a perplexing discrepancy in protocol compliance for elderly patients. Scheetz examined a registry of 5,712 trauma victims with an ISS $\geq$16. In that analysis, young men were most likely to be brought to a trauma center (82%), whereas older women were least likely to be transported to a trauma center (60%) [33]. Ma and colleagues conducted an analysis of 32,950 EMS transports and similarly found that a disproportionate percentage of elderly trauma victims were transported to nontrauma centers [34]. More investigation is needed to elucidate the reason for this trend.

Age itself has been suggested as a potential criterion for trauma team activation and for the use of early intensive monitoring and resuscitation. Demetriades and colleagues prospectively evaluated outcomes once age greater than 70 years was used to trigger trauma team activation. Overall mortality in the elderly population was significantly lower, decreasing from 53.8% to 34.2%, without a concomitant increase in survivors who had permanent disability (16.7% versus 12%) [35].

Most studies on TBI in the elderly population are muddied by the presence of multisystem trauma. Mosenthal and colleagues gathered retrospective data on elderly patients who had isolated TBI and found a persistent pattern of increasing mortality with each decade past 50 years. Overall in-hospital mortality for isolated TBI was twofold higher in elderly patients, 30% versus 14%, and remained significantly elevated even in mild to moderate brain injury. Even when pre-existing medical conditions and

complications were removed from the equation by logistic regression, age remained an independent risk factor for death despite that similar percentages received invasive neurosurgical interventions [24].

Geriatric TBI patients who survive to discharge exhibit poorer cognitive and functional outcomes when compared with younger cohorts. As expected, more elderly patients are discharged to skilled nursing facilities, require longer periods of rehabilitation, and display less rapid clinical improvement [36].

Chronic subdural hematoma

Among types of traumatic brain injuries, chronic subdural hematoma (SDH) bears special mention, because it can be subtle and varied in presentation and hence frequently misdiagnosed. Epidemiologic data show the annual incidence of chronic SDH is approximately 1 to 2 cases per 100,000 population, but this number increases to more than 7 cases per 100,000 among patients older than 70 years of age [37]. Most studies show a clear male predominance in all age groups [38].

Although frequently attributed purely to cerebral atrophy and concomitant stretching of the bridging veins, the vulnerability of the elderly patient to chronic SDH likely has additional contributors. As previously mentioned, aging patients are prone to falls and have an increased incidence of head trauma. They also are more likely to be on antiplatelet or anticoagulant medicines that can exacerbate bleeding from minor injury. Certain structural brain lesions, such as meningiomas and metastatic tumors, increase the likelihood of hemorrhage into the subdural space. Intracranial hypotension, such as that caused by over shunting of CSF, represents another potential etiology of chronic SDH. In fact, up to 8% of adults shunted for normal pressure hydrocephalus (NPH) develop subdural bleeding [39].

Minor trauma, often long forgotten by the time of presentation, is postulated to be the initial insult in most cases of chronic SDH. Severe head injuries with brisk bleeding are likely to present as an acute SDH. More minor injuries with slow hematoma accumulation lead to the delayed presentation and more subtle spectrum of deficits seen in chronic SDH. The seemingly benign nature of many of these injuries, the time lag before symptom onset, and the frequent presence of cognitive deficits in these patients means that 25% to 50% of them are not able to relate any history of trauma [38].

The presence of cortical atrophy in aging adults directly affects clinical presentation. A comparatively large space between the brain and inner table exists for hematoma accumulation, meaning that significant blood collections with mass effect can occur over time without causing elevations in ICP. Older patients thus tend to have an insidious onset of symptoms and are less likely to present with the classic clinical picture of ICP elevation, including headache, visual changes, and vomiting [38,40]. Instead, aged adults

are more likely to manifest seizures, focal neurologic signs, such as hemiparesis and aphasia, and subtle cognitive deficits, such as confusion, personality changes, memory loss, and impaired judgment. These changes can mimic many neurologic and psychiatric illnesses; common misdiagnoses include transient ischemic attack (TIA), stroke, vascular dementia, Alzheimer disease, and depression.

Emergency physicians must consider the diagnosis of chronic SDH when evaluating an elderly person who has mental status changes or sudden progression of known neurologic or psychiatric disease. It is diagnosed easily by CT scan (Fig. 3) and treatable by surgical intervention. Practitioners should seek a history of chronic SDH in such patients, because recurrence rates vary from 9% to 26% [41].

Spinal injury

Studies on cervical spine trauma show that elderly patients have different predominant mechanisms and patterns of injury. Older patients are more likely to be injured in falls and have increased likelihood of upper cervical injuries, particularly of the odontoid. Lomoschitz and colleagues conducted a retrospective analysis of 149 patients older than the age of 65 years with a total of 225 cervical spine injuries. C2 was the cervical bone injured most commonly, accounting for 40% of all fractures (Fig. 4). In the lower

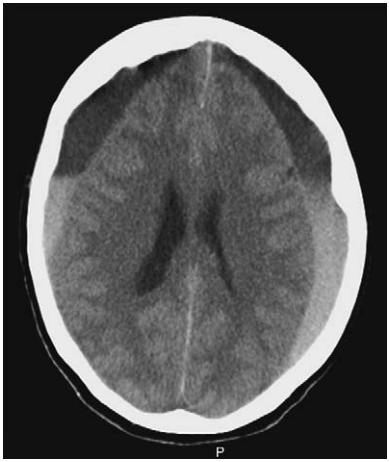

Fig. 3. The computed tomographic characteristics of subdural hematomas (SDH) change over time. Acute subdural blood usually appears hyperdense to brain parenchyma. After approximately 1 week, the hematoma becomes isodense. By 3 weeks, most SDHs appear hypodense to adjacent parenchyma [102]. Mixed density blood collections, as seen in this noncontrast CT, usually represent acute on chronic bleeding. This elderly woman was found down and had a recent history notable for 3 weeks of gait instability and confusion.

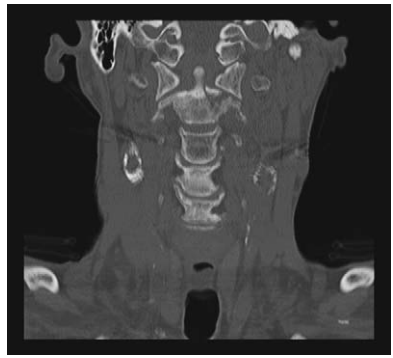

Fig. 4. Elderly patients are at particular risk for high cervical spine fractures, particularly of the dens. These injuries can be difficult to detect, because elderly patients are likely to present without signs of neurologic injury. This man suffered a comminuted C2 fracture during a fall from standing.

cervical spine, C5 and C6 were the most likely levels of injury, accounting for 12% each. Four in 10 elders sustained multilevel cervical injuries, most commonly at C1/2 or C5/6. The investigators specifically examined the differences in injury pattern, mechanism, and initial clinical presentation of those aged 65 to 75 years with those older than 75 years of age. The eldest patients (>75 years) were significantly more likely to have upper cervical injuries regardless of mechanism [42].

Degenerative changes of the spine may result in increased risk for spinal fracture and specifically atlantoaxial injury in older persons. Osteopenia places bones at greater risk from what would otherwise be trivial trauma. In young patients, C4 to 7 is the most flexible portion of the cervical spine and the most likely to fracture. The presence of senile degenerative disease alters spinal mechanics, making the upper cervical levels comparatively more mobile and vulnerable to blunt trauma.

Cervical spine plain film interpretation in very elderly patients is complicated by relative osteopenia, the presence of degenerative changes, and unreliable markers of soft tissue injury. Cervical radiographs lack obvious prevertebral soft tissue swelling in 17% of upper cervical and 40% of lower cervical spine injuries [42]. Given the enhanced sensitivity of CT scans and the high incidence of pathology, some physicians have advocated bypassing plain films in this population.

It may be safe in certain clinical situations to forego spine imaging in elderly patients. Touger and colleagues conducted a subpopulation analysis of geriatric patients in the National Emergency X-radiography Use Study (NEXUS) database to determine if NEXUS criteria can identify safely low-risk patients who do not need cervical spine imaging. The database included information on 2,943 patients 65 years of age or older (8.6% of the total sample). Of these, 14% failed to meet the five criteria for imaging and

qualified as low risk. Only two of these patients later proved to have cervical injury, specifically two cases of C2 lateral mass avulsion that the investigators classified as clinically insignificant. Based on this analysis, the sensitivity of NEXUS criteria for significant injury in patients older than 65 years of age was 100% (95% CI, 97.1%–100%) [43].

Many studies note the unnerving tendency for elders to suffer significant head and neck injuries from low-energy mechanisms, particularly same-level falls [26,27]. To further complicate clinical assessment, three in four elderly patients who have cervical spinal injury have normal neurologic examinations [42]. Aged patients are in fact less likely to exhibit paralysis at every level of spinal injury (Fig. 5) [44], despite their much higher risk for mortality [45]. Emergency physicians must be wary of such patients and have a low threshold for cervical immobilization and imaging.

It is not uncommon for geriatric patients to present to the ED with clinical evidence of vertebral or spinal cord injury in the absence of known trauma. The increased incidence of osteoporosis and cancer in this population substantially raises the risk for pathologic fracture. Degenerative spine disease may lead to canal or neural foraminal stenosis with subsequent motor and sensory findings. Unremitting back pain may be the herald of vertebral fracture, spinal metastasis, epidural hematoma, or abscess. The threshold for imaging the spine, whether by plain radiograph, CT, or magnetic resonance imaging (MRI) should be lower for geriatric patients. MRI is the modality of choice when there is clinical suspicion of spinal cord compression.

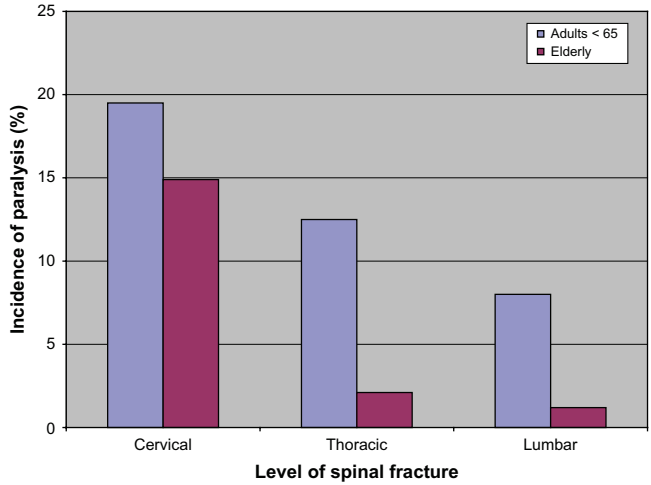

Fig. 5. Geriatric patients have a lower incidence of paralysis at every level of spinal injury [44].

Disparities exist in the rate of surgery in young and old patients who have spinal fractures. Elderly patients are significantly less likely to be selected for surgical intervention despite the clear mortality benefit in both groups [44].

Transient ischemic attacks

TIAs are common among older patients, occurring in as many as 1 in 15 adults older than the age of 65 years [46]. Up to 8.6% of patients have a stroke within 7 days of their index TIA, and more than half of these may occur within 48 hours of the initial ED visit [46,47]. Over 5 years, the stroke incidence exceeds 25% [48]. Correct diagnosis is paramount, because the institution of appropriate therapy can mitigate the risk for future stroke.

Diagnosing TIAs in the ED can be difficult. Such patients may present in a deceptively benign fashion; neurologic deficits have reversed often before physician evaluation, and brain CT is usually normal. A careful history is the key to diagnosis. Given these pitfalls, even experienced neurologists frequently disagree on the diagnosis [49].

Debate exists within the neurology community about the definition of a TIA. The classic definition of a sudden focal neurologic deficit caused by a vascular lesion that dissipates within 24 hours was framed before the availability of advanced brain imaging techniques, such as diffusion-weighted imaging (DWI) MRI. As a result, clinicians were unable to differentiate TIAs from strokes with reversible deficits. True TIAs usually resolve within 30 to 60 minutes. More than 98% of patients who do not reverse their deficit within 1 hour or rapidly improve within 3 hours are having a stroke, not a TIA [50]. These data have led some stroke experts to proffer a redefinition of TIA to include episodes that last typically less than 1 hour and are not associated with acute infarction [51]. This new proposed definition implies that an MRI has been performed.

TIA patients rarely require acute stabilization. All such patients warrant a finger stick blood glucose, a careful neurologic examination, and an ECG to look for arrhythmias. Any patient who has a persistent neurologic deficit must be treated as an acute stroke victim until proven otherwise. TIA patients whose deficits have resolved still should have some neuroimaging; at a minimum, a head CT without contrast should be performed to rule out other etiologies of neurologic dysfunction, such as hemorrhage or mass effect. No published guidelines argue for routine MRI for suspected TIA. MRI, however, detects small infarcts in up to 67% of patients who have traditionally-defined TIAs [52–54].

Further vascular assessment is indicated in patients diagnosed with TIA. Anterior circulation TIAs mandate urgent carotid evaluation, usually by ultrasonography, to look for high-grade stenosis. Two-year follow-up data from the North American Symptomatic Carotid Endarterectomy Trial (NASCET) revealed that patients who had high-grade stenosis defined as

greater than 70% occlusion achieved a 17% absolute risk reduction for ipsilateral stroke from carotid endarterectomy (CEA) [55]. Benefits were more modest (6.5% absolute risk reduction) for patients who had moderate (50%–69%) stenosis and absent for those who had only low-grade (<50%) blockage [56]. Clinical trials are in progress to determine if angioplasty with stenting may be more advantageous than CEA [57,58]. To date no published data address the optimal timing for vascular intervention; the need for carotid evaluation has not been proven to be emergent. Posterior circulation TIAs require radiologic studies of the vertebrobasilar system, such as transcranial Doppler ultrasonography (TCD) or angiography. In patients who have a possible cardioembolic source, transthoracic or transesophageal echocardiography is indicated.

Most patients who have TIA should be placed on antiplatelet therapy, because aspirin alone confers a 20% relative risk reduction for subsequent stroke [49]. Considerable variation in clinical practice, however, exists among neurologists with regard to full anticoagulation for these patients. One of the few proven indications for emergent anticoagulation is a TIA in the setting of new onset atrial fibrillation. In the European Atrial Fibrillation Trial (EAFT) study, 1,007 patients who had atrial fibrillation and a history of recent TIA or minor stroke were randomized to anticoagulation, aspirin, or placebo treatment arms. The annual rate of stroke was 8%, 15%, and 19%, respectively [59]. TIA patients who had new atrial fibrillation or flutter should be admitted, anticoagulated, and assessed for intracardiac thrombus with echocardiography. Although controversial and not based on data from RCTs, up to one in two neurologists also recommend heparin for patients who have crescendo TIAs [60].

Given the high but unpredictable risk for further ischemic events, many clinicians routinely admit TIA patients to assure an expedited work-up and close monitoring. Certain patient subsets at particular risk and who may warrant admission and urgent neurologic consultation include: (1) patients who failed first-line therapy with antiplatelet agents, such as aspirin or clopidogrel, (2) patients on full anticoagulation, such as enoxaparin or warfarin, (3) patients who have crescendo TIAs, defined as three or more events over 72 hours with escalating severity or duration, and (4) patients who have suspected cardioembolic sources of TIA, such as new onset atrial fibrillation or valvular vegetations from endocarditis.

There are few data to guide admission decisions for TIA patients. Johnston and colleagues studied patients diagnosed with TIA in the ED to isolate risk factors associated with poor short-term prognosis. Using multivariate logistic regression, Johnston found five factors associated with higher short-term risk for ischemic stroke: age greater than 60 years, a history of diabetes, symptoms lasting more than 10 minutes, and the presence of motor weakness or speech difficulties with the event. The 3-month risk for stroke ranged from 0% in patients who had none of the five criteria to 34% in those who met all five [46]. The caveat to Johnston's data is that the average

length of symptoms in the study was 207 minutes; ergo by modern definition, this was a mixed population of TIAs and strokes.

Certain factors increase the likelihood of safe outpatient work-up and management of a TIA. Benavente studied medically treated patients who had amaurosis fugax versus those who had hemispheric TIA and discovered that the 3-year risk for ipsilateral stroke was twice as great in the latter group [61]. Patients who have this form of transient monocular blindness thus may be discharged safely on aspirin if prompt carotid imaging and close physician follow-up can be ensured. Greater caution is warranted in patients who are male, older than 75 years of age, and have a past history of hemispheric TIA or stroke, because their risk for subsequent ischemic events is higher [61]. Elderly patients whose TIA occurred more than 1 week before arrival also may be safe for outpatient work-up, because the period of greatest risk has passed.

A recent study showed that nearly 30% of patients discharged from the ED with a preliminary diagnosis of TIA were not given antiplatelet agents [62]. This inconsistency creates vulnerability of the patient to further ischemic events and of the physician to litigation. If considering outpatient TIA management, it is essential for the emergency physician to discuss the risk for future stroke with the patient, explicitly describe reasons to return for emergent care, prescribe an antiplatelet agent or document its contraindication, and coordinate timely and appropriate follow-up.

Stroke

Stroke is the leading cause of disability and the third leading cause of death in the United States. Detailed descriptions of stroke syndromes are beyond the scope of this article but have been well described elsewhere [63].

One of the first steps in the evaluation of suspected stroke is to check a blood glucose level, because hypoglycemia can mimic an infarct from any vascular territory. Practitioners must keep in mind that such neurologic deficits occasionally take several hours to reverse even after the restoration of normal serum glucose levels. Although ongoing deficits persist, emergency physicians must continue to evaluate the patient for the possibility of hemorrhagic or ischemic disease.

Many other clinical entities besides hypoglycemia can masquerade as stroke. Other mimics include seizures with postictal Todd paralysis, CNS infections, toxic-metabolic defects, and intracranial mass lesions, such as chronic SDH [64,65]. Libman and colleagues examined more than 400 patients diagnosed with acute stroke in the ED after the completion of a history and physical examination and discovered that nearly 20% had an entirely different source of CNS pathology [66]. The percentage of misdiagnosed strokes decreases substantially to less than 5% once historical and physical examination data are combined with brain imaging (Fig. 6) [64]. The potential for misdiagnosis bears special importance for patients who arrive shortly

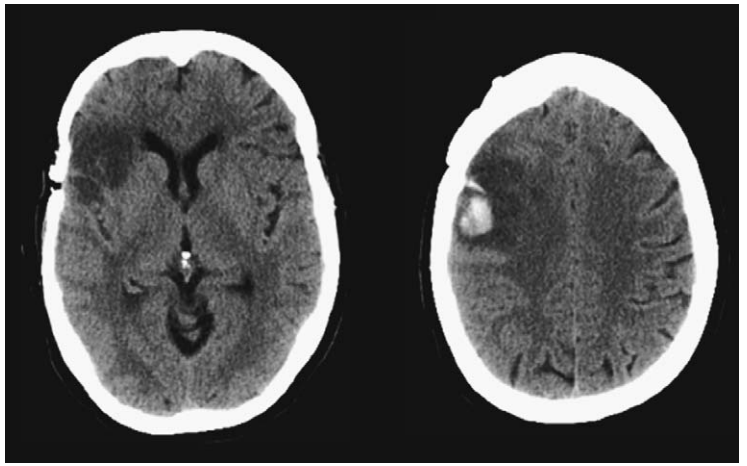

Fig. 6. These axial images from a noncontrast head CT scan reveal a wedge-shaped region of low attenuation in the right frontal lobe with a focal intraparenchymal hemorrhage at the superior margin. These images, consistent with a hemorrhagic transformation of a subacute infarct, were taken from a 65-year-old woman who had a history of TIAs and who presented with a right facial droop and slurred speech.

after the onset of symptoms. Although no clinician would intentionally treat a stroke mimic with thrombolytics, a recent small observational study showed no hemorrhagic complications in these patients [67].

The urgency of assessment and treatment implicit in the golden hour model of trauma care has been adapted successfully to stroke. Guidelines for optimal speed in treatment were established by the National Institute for Neurological Disorders and Stroke (NINDS) Study Group in 1997. The recommendations suggest an initial physician evaluation within 10 minutes of ED arrival, noncontrast CT scan within 25 minutes, radiologist interpretation within 45 minutes, and the administration of thrombolytics, if appropriate, within 1 hour [68]. Such a stringent timeline is only feasible with firm commitment from the departments of emergency medicine, radiology, and neurology within a given institution.

The incidence of spontaneous intracerebral hemorrhage (ICH) ranges from 10 to 20 cases per 100,000 population and is associated directly with advancing age [69]. Chronic hypertension and amyloid angiopathy, both more common in the elderly population, increase the likelihood of bleeding. Hemorrhagic strokes, although less frequent than ischemic events, are more deadly and have fewer effective therapeutic options. One-month mortality ranges from 35% to 52% [70].

Early progression of hemorrhage is common and linked with abrupt clinical decline. In a prospective study, Brott and colleagues found that 38% of ICH patients imaged within 3 hours of symptom onset developed significant hematoma expansion within 20 hours. Nearly 70% of these patients (26% of

the ICH study population) progressed within 1 hour of the initial CT scan [71]. Because hematoma size has proven a powerful predictor of mortality and functional outcome in ICH, some researchers have focused on the therapeutic role of early hemostatic therapy. Activated factor VII is a candidate hemostatic agent that may have clinical benefit even for patients who do not have coagulopathy [72,73].

Acute medical management of geriatric patients who have ICH is the same as in younger patients and includes airway management, blood pressure and ICP monitoring, reversal of coagulopathy, and seizure prophylaxis. Patients who have evidence of elevated ICP may benefit from osmotherapy with mannitol and cautious hyperventilation. Because ICH patients are at risk for hematoma expansion and subsequent neurologic decline, they often benefit from admission to an intensive care unit.

The role for surgical drainage in ICH is still an area of active clinical investigation. Hankey and colleagues examined data on 249 ICH patients from three surgical trials and documented increased rates of death and dependency in the subset of patients randomized to craniotomy and hematoma evacuation (83% versus 70%) [74]. In 2005 the International Surgical Trial in Intracerebral Hemorrhage (STITCH) published data on 1,033 ICH patients from 83 medical centers and demonstrated that early surgery provided no clinical benefit for patients who had supratentorial ICH [75]. Craniotomy sometimes is recommended in cerebellar hematomas [69], but further study is needed to determine the clinical benefits of intervention for these patients.

Ischemia accounts for most strokes, with an incidence of 300 to 500 events per 100,000 population [76]. Acute medical management includes blood pressure monitoring, fever control, blood glucose regulation, and, if appropriate, thrombolysis. Hypotension is disastrous in patients who have stroke; given the loss of normal autoregulation and the marginal perfusion of the penumbra, mild to moderate hypertension should remain untreated. Antihypertensive medications should be administered only for specific indications, such as concurrent acute coronary syndrome (ACS), aortic dissection, malignant hypertension, or severe hypertension in a thrombolytic candidate.

Given the enormous potential for functional impairment and mortality in ischemic stroke, there is considerable interest in thrombolytic agents. The first published studies were not encouraging, leading to controversy within the medical community about continued clinical exploration. In 1995 the NINDS study was published [50]. In part one of the study, 291 ischemic stroke patients presenting within 180 minutes were randomized to treatment with intravenous tissue plasminogen activator (rt-PA) or to placebo. Researchers found no clinically significant improvement at 24 hours among patients in the treatment arm. In part two of NINDS, 333 patients were randomized to rt-PA versus placebo and patients in the rt-PA arm showed improved functional outcomes at 3 months. The data showed that eight to nine

patients would require thrombolysis to have one patient recover with minimal or no deficit. The downside of thrombolysis was evident; patients treated with rt-PA were far more likely to have a symptomatic ICH (6.4% versus 0.6%). The subsequent FDA approval for thrombolytic agents later that year rested largely on the NINDS data.

Despite the volume of research published since NINDS, the use of thrombolytics remains a subject of academic discussion and dispute. The controversy surrounding rt-PA use culminated in the 2002 release of a position statement by the American Academy of Emergency Medicine (AAEM) stating that insufficient evidence existed on the efficacy of thrombolytics to support their inclusion in the standard of care for ischemic stroke [77]. In response to continued questions regarding the safety and usefulness of thrombolytics and methodologic concerns about the original study, the NINDS researchers commissioned an independent committee to reanalyze the data from the landmark 1995 article. This group confirmed the beneficial effect of rt-PA with an adjusted odds ratio (OR) for a favorable outcome of 2.1 (95% CI, 1.5–2.9) [78]. Meta-analysis has bolstered further the case for thrombolytics. In 2003 Wardlaw and colleagues compiled data from 14 RCTs investigating the use of thrombolytics within 6 hours of symptom onset for acute ischemic stroke. The data suggested that approximately 55 more patients would survive and live independently for every 1,000 treated. This number includes the approximately 20 deaths per 1,000 caused by ICH [79].

ICH is the immediate and often disastrous risk for thrombolytic treatment. Among the studies in Wardlaw's meta-analysis, symptomatic ICH occurred in 10% (153 of 1,496) of rt-PA patients and only 3% (46 of 1,459) of control subjects [79]. The risk for hemorrhage may be higher in geriatric patients; Heuschmann and colleagues found the rate of ICH was twice as high in patients older than 75 years of age as in those younger than 55 years of age (10.3% versus 4.9%) [80]. Attention to applicable contraindications is critical, because unacceptably high levels of ICH are associated with protocol violations. A study of Cleveland area hospitals reported a 15.7% incidence of symptomatic ICH, more than twice the 6.4% reported in NINDS [81]. Approximately 50% of those patients were treated outside of existing administration guidelines, leading some academicians to argue that thrombolytic agents could not be administered safely in the community setting. The rebuttal to this argument arrived 3 years later, when Katzan demonstrated that local quality improvement initiatives could increase overall use of rt-PA, decrease protocol violations, and result in a rate of hemorrhagic complications comparable to the original NINDS data [82]. Other investigators have confirmed that rt-PA can be administered successfully in community hospitals [83].

It is difficult to assess the safety of thrombolytics for ischemic stroke in very elderly patients given the paucity of data. Octogenarians are approximately 60% less likely and nonagenarians 85% less likely to be given the

option of thrombolytic agents when compared with those younger than 60 years of age [79,84]. NINDS was perhaps the only published RCT that included patients older than age 80 years; data from only 42 such patients exists, making it impossible to form solid conclusions about the risk-to-benefit ratio of thrombolytics in the very elderly [79]. One recent observational study of 1,658 ischemic stroke patients treated with rt-PA showed that age increased in-hospital mortality with an adjusted OR of 1.6 for each additional decade in patient age. The caveat to those data is that older age also predicted in-hospital mortality in patients not treated with rt-PA, and the observational design of that study did not allow for direct comparison of the two groups [80].

Practitioners need to remember that once thrombolytics are administered, patients should be admitted to the intensive care unit or to an acute stroke unit. In addition, no invasive access, including arterial lines, central lines, or Foley catheters, should be placed for at least 2 hours following the completion of the thrombolytic dose.

Current thrombolytic guidelines stem from the original NINDS study and do not account for subsequent advances in imaging techniques, the variety of causative vascular lesions, and the judgment and experience of individual clinicians [85]. As knowledge and experience with particular vascular lesions increases, the accepted indications for thrombolysis will likely evolve. Clinical trials investigating longer time windows and the use of intra-arterial administration of thrombolytics are in progress at stroke centers around the country. Given the abysmal prognosis for basilar occlusion, some investigators use thrombolytics up to 48 hours after symptom onset [86]. Emergency physicians practicing in the community setting are well advised to develop protocols and working agreements with stroke centers to maximize the therapeutic options for these patients.

Dizziness

Dizziness is a common symptom in all age groups but is particularly prevalent in the elderly population. Approximately 50% of geriatric patients experience dizziness, and it is one of the most common presenting complaints in adults older than age 75 years [87,88]. The evaluation of the patient experiencing dizziness can be difficult, because patients use that word to describe a myriad of sensations, including fatigue, near-syncope, disequilibrium, and vertigo. Many elderly patients present with a mixed picture in which two or more forms of dizziness exist. The astute clinician must perform a careful history and physical examination to determine the source of symptoms and institute proper therapy. Although a complete review of the assessment of the patient experiencing dizziness goes beyond the scope of this article, it is well covered elsewhere [87,89,90]. This section focuses primarily on vertigo, because this includes most of the serious neurologic causes of dizziness.

Vertigo, or the illusory sense of motion, is usually peripheral, even in older patients. Peripheral vertigo is associated with acute onset of episodic, severe vertigo frequently associated with nausea, vomiting, tinnitus, and hearing loss. Common causes of peripheral vertigo include motion sickness, benign paroxysmal positional vertigo (BPPV), otitis media, vestibular neuronitis, Ménière disease, and toxic labyrinthitis from ototoxic medications. Vertigo that is positional and lasts for less than 30 seconds is almost always caused by BPPV. This diagnosis can be made by performing the Dix-Hallpike maneuver. Patients who have central vertigo experience less intense symptoms of longer duration. The differential diagnosis of central vertigo includes alcohol intoxication, temporal lobe seizures, migraine, head trauma, vertebrobasilar insufficiency, and posterior fossa masses or ischemia.

Although peripheral causes of vertigo tend to be ''benign'', the symptoms are, at a minimum, annoying and at times incapacitating to patients. The attendant nausea and vomiting may lead to dehydration that results in further morbidity. Simple treatments initiated in the ED can lead to significant clinical benefits. Recent data suggest that steroids improve outcomes in patients who have vestibular neuronitis [91]. Also, 80% to 85% of patients who have BPPV can be cured in the ED with a simple bedside maneuver, the modified Epley [92].

Distinguishing between peripheral and central causes of vertigo is critical [90], because many central etiologies require emergent treatment. Hearing loss strongly suggests a peripheral cause, because the colocation of hearing and balance occurs only in the peripheral nervous system. The presence of vascular risk factors (hypertension, diabetes, smoking) and abrupt onset of severe headache increase the likelihood of stroke. The presence of other neighborhood signs and symptoms of posterior circulation deficits (diplopia, dysarthria, ataxia, long tract problems) is another clue to serious central disease. Although patients who have peripheral vertigo may have difficulty walking, patients who have cerebellar stroke often cannot walk at all. Gait testing is therefore mandatory in all such patients. Nystagmus, when present, can help distinguish central from peripheral vertigo. Patients who have pure vertical or direction-changing nystagmus should be assumed to have a central cause until proven otherwise.

Vertigo is a concerning symptom in elderly patients, because they are at greater risk for serious CNS pathology. History and physical examination are not infallible in distinguishing central from peripheral disease. For example, infarction of vestibular nuclei from basilar artery branch occlusion can be indistinguishable from vestibular neuronitis by examination. Norrving conducted a small prospective study of 24 patients aged 50 to 75 years who presented with isolated acute vertigo and discovered that 6 of the 24 (25%) were having cerebellar ischemia [93]. Some of these events were cardioembolic; diagnosing such events provides physicians with the opportunity to initiate anticoagulation and prevent subsequent strokes. Unless the

cause of vertigo is clearly benign, physicians must maintain a lower threshold for imaging and neurologic consultation in vertiginous elderly patients (Fig. 7).

Central nervous system infection

Meningitis and epidural abscess

Physicians must be alert to the higher likelihood and subtler presentation of infectious disease in the geriatric population [94]. Functional decline of immune cells associated with normal aging, termed immunosenescence, and other contributors such as malnutrition, result in increased susceptibility to infection. Presenting complaints in elderly patients are often nonspecific, such as confusion or frequent falls. In addition, older patients have a blunted fever response and may be normo- or even hypothermic.

Meningitis can occur at any age, but its largest spikes in incidence occur in infants and in people older than age 60 years. Geriatric incidence is estimated to be two to nine cases per 100,000 population [95]. Diagnosing meningitis in the geriatric population carries special clinical urgency, given the increased rate of serious complications and in-hospital mortality [96].

Unfortunately diagnostic and treatment delays are common in part because elderly patients are likely to present with an array of subtle, nonspecific signs and symptoms. The clinical triad of fever, nuchal rigidity, and altered mental status is rarely present in its entirety and has an abysmal 46% sensitivity among older patients. More than 99% have at least one of these findings, however, making the absence of all three useful in ruling out the diagnosis [97].

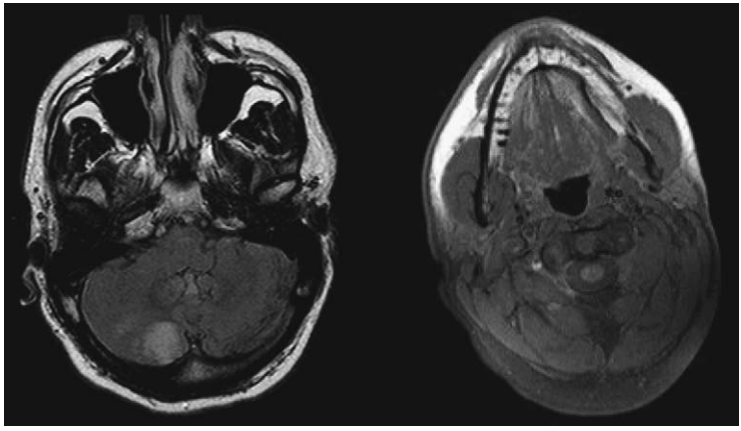

Fig. 7. The axial MRI on the left shows small areas of restricted diffusion in the right inferior cerebellum, indicating an acute right anterior inferior cerebellar artery (AICA) territory infarct. Signal changes in the right vertebral artery seen in the second image are consistent with thrombosis and dissection. This elderly patient presented with acute onset vertigo and ataxia.

Signs and symptoms of meningeal irritation are particularly unhelpful in aging patients. Nuchal rigidity, found in 92% of young patients who have meningitis, is less common and less specific in the elderly population. Neck stiffness is found in 57% of elderly patients who have meningitis [98], but in 35% of those without any evidence of CNS infection [99]. Rigidity may represent multiple other conditions in this age group, including Parkinson disease, osteoarthritis, or cervical spondylosis. Meningeal signs are also less reliable; 12% of healthy elderly people display a positive Kernig sign and 18% have a positive Brudzinski [99].

Further complicating clinical assessment is that 40% to 58% of elderly patients who have meningitis present with concomitant infections, such as pneumonias or urinary tract infections [100]. The discovery of a large infiltrate on a chest radiograph can distract clinicians from the presence of CNS infection and lead to premature closure of the diagnostic work-up.

Spinal epidural abscess is another dangerous infection more common in older patients. Any delay in diagnosis can be disastrous, because emergent surgical debridement in combination with antibiotics can prevent permanent paralysis and death. It is a difficult diagnosis to make in elderly patients, who frequently present to the ED with back pain from degenerative disease. In fact, diagnostic delays occur in up to 75% of patients, in part because more than 85% do not have the classic triad of spinal pain, fever, and neurologic deficits [101]. A lack of fever and leukocytosis does not rule out the diagnosis. Although MRI is the gold standard test (Fig. 8), some have advocated the use of inflammatory markers, such as erythrocyte sedimentation rate (ESR), as a screening tool for patients who have a lower pretest probability [101].

Patients who have suspected epidural abscess must be transferred to a center that offers MRI imaging and spinal surgery consultants. Antibiotic therapy should be administered before transfer. Patients who have suspected cervical abscesses should be monitored carefully in the ED and sent by ALS

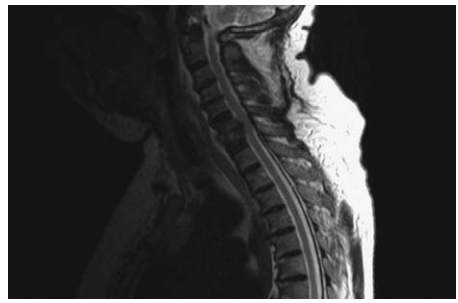

Fig. 8. Central nervous system infections can present without fever or neurologic signs in elderly patients. This is a sagittal MRI image of the spine in a 73-year-old woman who presented to the emergency department with a sole complaint of neck pain. This image reveals C6/7 osteomyelitis and discitis with epidural extension resulting in spinal cord compression.

if transferred, because they are at risk for respiratory decompensation. For safety, some patients may require intubation to undergo MRI or transfer.

Summary

Geriatrics is an important subspecialty within the field of emergency medicine and represents a burgeoning area of practice. The special vulnerability of the elderly population to neurologic disease and injury and the comparative subtlety of clinical presentation mean that physicians should have a lower threshold for laboratory studies, radiologic imaging, consultation, and admission. Transferring appropriate patients to tertiary centers that offer specialized trauma, neurologic, and neurosurgical care, greatly enhances survival and functional outcomes.

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