

Case Report

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Spinal Subdural Hemorrhage as a Cause of Post-Traumatic Delirium

A 64-year-old man with TBI was admitted to our institute. In following days, he showed unusual behavior of agitation, restlessness, emotional instability and inattention. Post-traumatic delirium was tentatively diagnosed, and donepezil was given for his cognitive dysfunction. Although there was partial relief of agitation, he sustained back pain despite medication. Lumbar magnetic resonance image revealed SDH along the whole lumbar spine, and surgical drainage was followed. Postoperatively, his agitation disappeared and further medication was discontinued. We report a unique case of post-traumatic delirium in a patient with concomitant TBI and spinal subdural hemorrhage (SDH) that resolved with operative drainage of spinal hemorrhage.

KEY WORDS : Back pain · Delirium · Subdural hematoma · Traumatic brain injury.

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INTRODUCTION

Traumatic brain injury (TBI) is manifested in altered consciousness, although the degree of disturbance can range from coma to transient confusion¹⁴. In most patients, acute confusion improves with time, and the agitation is attributed to a certain anatomic, physiological or neurochemical derangement within the brain. Neuropathologic concepts of the TBI, thus constitute and provide theoretical basis for behavioral and pharmacologic intervention to post-traumatic delirium¹². We had recently experienced a patient with such post-traumatic delirium that had been successfully treated by surgical intervention of lumbar subdural hemorrhage (SDH). The role of extracerebral impact, i.e. pain elsewhere in the body, in developing delirium is discussed with review of the relevant literatures.

CASE REPORT

A 64-year-old man was referred to our institute for evaluation of somnolence following vehicle collision accident. He had been drinking about 8 ml (6.4 g) of alcohol weekly for 25 years, but had no significant past medical history of admission or operation. Initial physical examination showed scalp bruises and swelling on left side of the head, but evidence of systemic injury was not disclosed. He was drowsy, and showed mild right-sided facial palsy, diplopia and strabismus. There was neither sign of sensory/motor dysfunction in the extremities, nor abnormal laboratory result. A computed tomogram (CT) showed multiple cerebral contusions and subdural clots over the underlying atrophied cortex (Fig. 1). Conservative management including NSAIDs (e.g. ibuprofen), muscle relaxant (eperisone), anti-convulsant (valproate) and benzodiazepine were prescribed with conventional dosage.

In next three days, he became disoriented to time and place, showed bizarre behavior such as yelling, agitation, cursing to the nursing staffs, labile emotion, and did not sleep at all. On sixth hospital day, minimal state examination (MMSE)² was assessed that revealed 18 points, the confusion assessment method (CAM) score⁸ to be positive 1, 2, 3 categories,



Fig. 1. Non-enhanced brain computed tomogram at admission shows multiple contusions and subdural bleeds over the atrophied cortex, predominantly on the right side.

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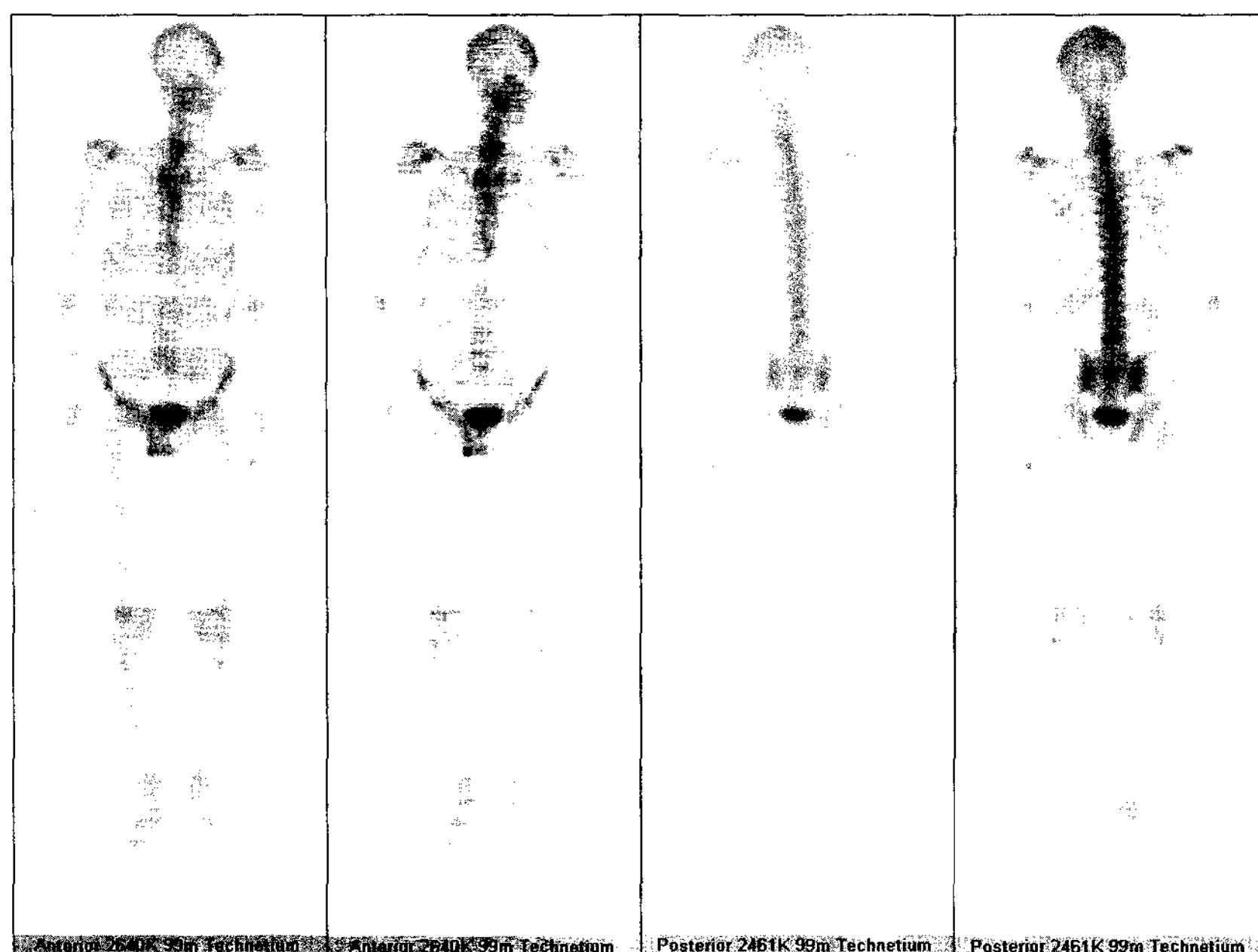


Fig. 2. Whole body bone scan using 99 m Technetium fails to show any abnormal high-uptake lesion in the body.

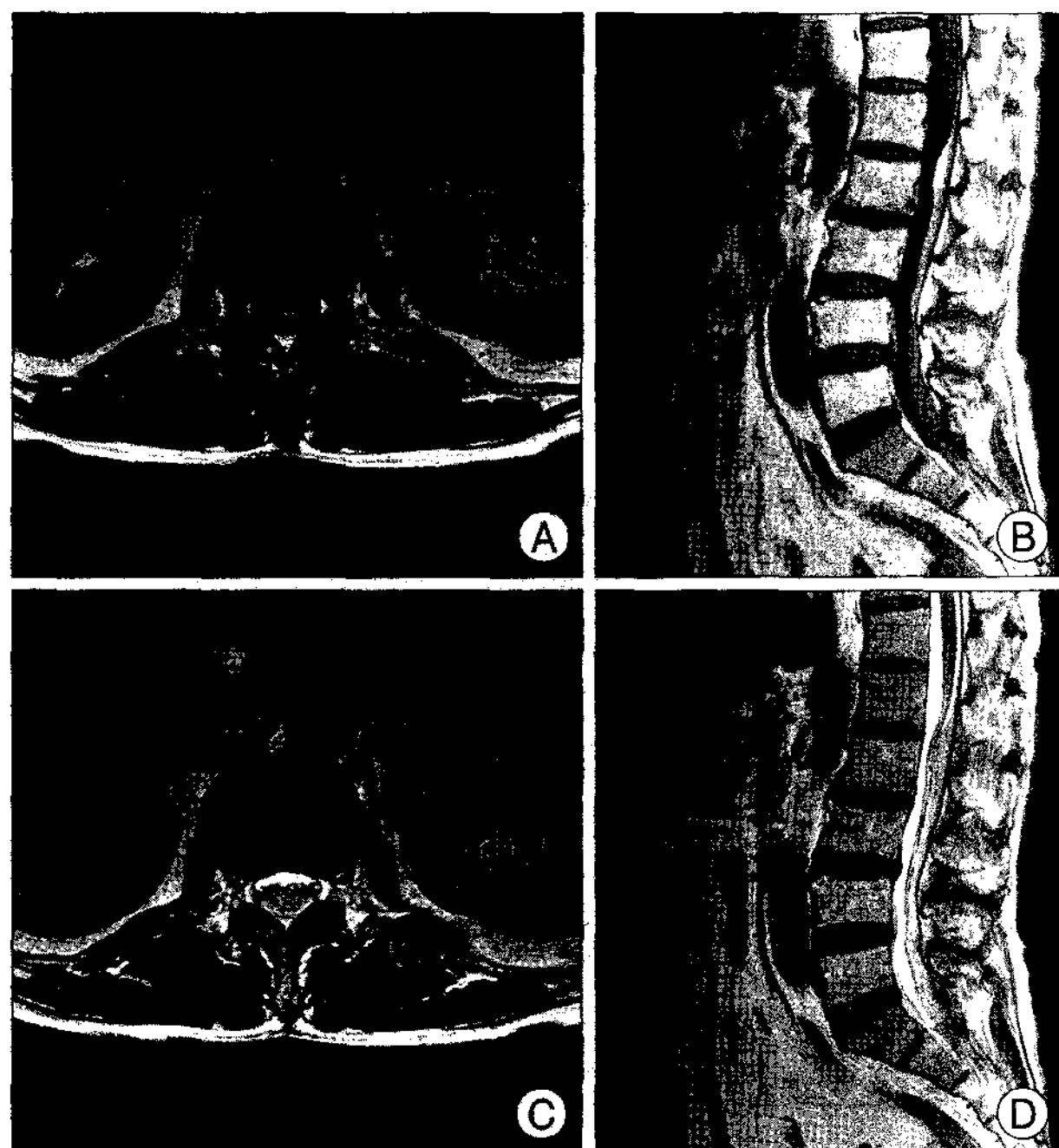


Fig. 3. Lumbar magnetic resonance image obtained at eighteenth hospital day. Non-enhanced T1-weighted axial (A) and sagittal (B) images show a crescentic high signal intensity lesion dorsal to the cauda equina. It spans from lower thoracic to lower lumbar region, and merges with subarachnoid component at the lumbo-sacral junction. T2-weighted axial (C) and sagittal (D) images also show intermediate signal lesion with crescent shape.

and global deteriorating scale (GDS) in stage 4 (moderate cognitive dysfunction), respectively. Donepezil (Aricept; Eisai, Tokyo, Japan) 5 mg was empirically administered. Following five days of prescription, his confusion and

insomnia was slightly relieved, but lower back pain that started before 3 days was persistently present. His pain with visual analogue scale (VAS) score of 8 point was sustained all-day long at rest and was not associated with movement. Repeated physical examination, lumbar X-ray, brain CT and radio-isotope bone scan were undertaken, but they failed to disclose any responsible causes (Fig. 2).

On 18th day, lumbar magnetic resonance image (MRI) was performed that revealed unexpected subdural bleeds along the whole lumbar spine that was compressing cauda equina from behind (Fig. 3). Surgical drainage was then performed for lumbar subdural hemorrhage (SDH) under a general anesthesia. Postoperatively,

he did not complain back pain anymore (VAS score 1/10). Fortuitously, his confusion was much resolved and he was able to do most of activities of daily living (ADL) by himself. On postoperative 7 days, MMSE showed 29 points, negative CAM score and GDS of stage 2. Further medication for delirium was halted and dosage of pain pills was also reduced.

DISCUSSION

Because the presentation of delirium varies and often be vague and multifaceted in most cases, only a high index of suspicion makes the patient approach, diagnosis, and ongoing management amenable. Once delirium is strongly suspected, prompt initial action should be attempted either by close patient interview with psychiatric tools (MMSE, CAM, GDS, etc), radiographic images (CT, MRI), or any other methods to confirm the diagnosis or to rule out other lesions.

Acute confusion following TBI, a.k.a. *after-effects of concussion, acute traumatic psychosis, delirium or post-traumatic confusional state (PTCS)*, refers early phase of recovery and includes rapid-onset symptoms of agitation and irritability caused by attention abnormality, disorientation including cognitive or perceptual disturbance, decreased judgement and arousal, disinhibition, inappropriate mood and disrupted sleep/wake cycle^{14,15}. As their names imply, they arise from disorganized brain structures including the parietal lobe, the frontostriatal regions, the middle temporal gyrus, the hippocampal formation, and the fusiform gyri of the medial temporal lobe¹¹.

As for the pathophysiology of delirium, reversible neuronal dysfunction is likely the cause. Systemic inflammatory response due to some vasoactive substances and resulting perivascular edema present as a conduction disability of the nerve and decreased cerebral perfusion in certain brain areas. Acetylcholine also seems to play a crucial role in the development of delirium due to its various roles in the regulation of cerebral functions. Thus, the lack of acetylcholine or relative excess of dopaminergic transmission seems to be connected to the development of delirium. The age-dependent decrease of acetylcholine transmission is a physiologic process, and reduced "cholinergic reserve" may be responsible for the significantly higher incidence of delirium among elderly patients^{3,6,9}.

To attain satisfactory relief of post-traumatic delirium, target behaviors should be divided into those that are amenable to nonpharmacologic (behavioral) intervention versus those that require psychopharmacologic intervention, only after thorough systemic evaluation¹². Meticulous examination is thus, of utmost importance to search for underlying cause and factors that easily unrecognized. If behavioral interventions are ineffective, psychotropic medication may be needed, including neuroleptics to control symptoms like agitation, restlessness, and altered perception. Physostigmine and metrifonate are also considered breakthrough pharmaceuticals for treating behavioral problems associated with delirium by ameliorating cholinesterase activity^{1,4}. Successful use of donepezil, an effective regimen against dementia that lowers serum anticholinergic activity, has been recently reported in a delirious patient¹⁷.

In the present case, this patient has multiple TBIs within the brain including sites previously addressed. Interestingly enough, degree of confusion and agitation of the patient was reduced with the aid of donepezil in some extent, but with limited and temporary efficacy. Because majority of delirious patients seem to have such cerebral lesions pertinent to specific disorder or clinical manifestation, treatment is generally aimed to correct these anatomical or chemical disturbances. We provided relevant medication for the patient but only to fail relieving delirium. Instead, we paid close attention to his complaint of back pain and voiding difficulty. Initial radiographs failed to disclose any spine lesions, nevertheless, with the help of a lumbar MRI scan, we found unexpected spinal SDH along the whole lumbar spine. We did not find any predisposing conditions for development of spinal SDH, therefore, the etiology is believed to traumatic, although very unusual. When we consider concomitant TBI and spinal SDH, increased intracranial pressure might also increase shearing force between weak inner spinal subdural layer and subarachnoid

membrane, as proposed previously. Paucity of neurologic signs, lumbar location and symptom duration less than 3 months are favorable prognostic factors explaining this case^{5,7}.

Poorly controlled pain has been reported to have close relationship with delirium in hospitalized elderly¹³. As shown in the current case, increased pain at rest is the only causative factor associated with developing delirium. Although pain with movement may occur more severely in an acute physiologic stress, patients experience pain at rest for more hours. Therefore, pain at rest is more likely to affect their sleep-wake cycle and hormonal balance. Sleep disturbance may contribute to and warn of imminent delirium. Sleep may be disturbed by worry, depression, noise, excessive heat or cold, discomfort, pain, itching, shortness of breath and a full bladder or bowel, among other causes, some of which are easily preventable¹⁰.

Oxidative stress, like severe pain and fever, affects basal forebrain cholinergic centers and is associated with decreased synthesis or release of acetylcholine¹⁶.

CONCLUSION

We recommend the need for a head-to-toe examination in a patient with acute confusion or agitation following TBI. This is particularly true when the patient ceaselessly complain the pain elsewhere in the body. Careful examination with radiographic investigation and appropriate management can prevent a delirious patient from delayed diagnosis of hidden disease and unnecessarily lengthy hospital stay.

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