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Double Jeopardy in Dual Diagnosis: Challenges in Patients with Traumatic Spinal Cord Injury and Brain Injury

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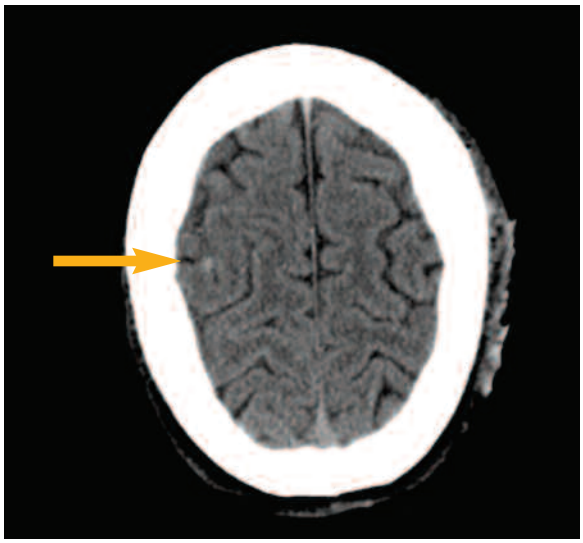
Case Vignette

DJ is a 24-year-old, right-handed male who was an unrestrained driver in a motor vehicle accident. At the time of the accident, he sustained brief loss of consciousness with an initial GCS of 13. After neck immobilization, DJ was sent to a Level I trauma facility where spine imaging revealed a C6 burst fracture with instability. His head CT and other routine skeletal survey imaging were unremarkable.

DJ underwent emergent cervical spinal decompression and repair. ASIA examination post-operatively revealed a C6 ASIA C spinal cord injury. He did not have any post-operative complications and was successfully extubated. When medically stable, DJ was transferred to the UPMC spinal cord inpatient rehabilitation unit for further care.

Several days into his rehabilitative course, DJ appeared to have difficulty participating in therapies. He was withdrawn, emotionally labile, and had difficulty retaining new skills. These impairments impeded his ability to benefit from rehabilitative therapies. Nursing records revealed insomnia with daytime somnolence. DJ also complained of moderate frontal headaches. His physical examination disclosed flat affect, poor attention, memory retention deficits, and word-finding difficulty.

Follow-up imaging of his brain *(see scan on Page 2)* showed small, micro-hemorrhages in the frontal lobe, frontal sulcus, and falx cerebri. These micro-hemorrhages were not appreciated in the initial scans performed in the trauma bay. The treating physicians asked for consultation regarding traumatic brain injury treatments in the setting of acute spinal injury.



This axial image of the patient's head CT shows micro-hemorrhages as depicted by the arrow.

Introduction

Individuals who suffer simultaneous spinal cord injury and brain injury after a traumatic event are defined as having a dual diagnosis.[1] Dual diagnosis is commonly seen in high kinetic abrupt impacts at high velocity.[3, 7] Motor vehicle accidents, falls, acts of violence, and sports-related activities, respectively, account for the mechanisms leading to dual diagnosis. These causes are ordered from highest to lowest incidence. Of these mechanisms, motor vehicle accidents account for a 50% rate of dual diagnosis compared to other causes.[8]

The incidence of dual diagnosis is higher when spinal cord injury is identified as the primary injury. Data from the Spinal Cord Injury (SCI) Model Systems indicate that 28.2% of all SCI cases have evidence of at least mild traumatic brain injury (TBI). Furthermore, 11.5% have severe brain injury with significant cognitive deficits.[2] Tolonen et al. conducted a study in 2007 in Finland showing a 74% rate of co-occurrence of TBI and SCI.[3] The incidence of SCI when brain injury is the primary diagnosis is less common, estimated at 1.2% to 6%.[1]

Younger males are at greatest risk for dual diagnosis. Additionally, there may be a correlation between dual diagnosis and substance abuse.[4] A study conducted by Kolakowsky-Hayner et al. identified a high incidence of prior substance abuse in traumatic brain injury patients (81%) and spinal cord injury patients (96%).[5]

There appears to be a trend between cervical traumatic spinal cord injuries and co-occurring brain injuries.[10] This relationship is directly related to the mechanism of head trauma leading to indirect forces on the cervical spine.[6] Mahmoud et al. described the biomechanics of trauma to include compressive and distractive forces on the skull producing spinal cord injuries. These forces produce injuries such as teardrop fractures, burst fractures, and ligamentous instability in the cervical spine.

Diagnostic Challenges

While it is clear there is a co-occurrence of TBI and SCI, the incidence is variable because of the high occurrence of missed diagnosis of TBI in the setting of SCI. There exist several diagnostic pitfalls in dual diagnosis when spinal cord injury is the primary injury. These include unidentified concurrent brain injury in mild cases, lack of universal standardized criteria in defining closed head injury, poor documentation of cognitive status in the acute care setting, limited use of neuropsychological testing in the acute care setting, and greater emphasis on recovery from the spinal cord injury.[6, 9] *(see Table 1)*

TABLE 1: Dual Diagnosis Diagnostic Pitfalls

- **Mild traumatic brain injury**
- **Lack of universal standardized criteria for defining brain injury**
- **Poor documentation of cognitive impairments in acute care**
- **Lack of neuropsychological testing**
- **Emphasis on the primary spinal cord injury recovery**

In dual diagnosis, mild traumatic brain injury (GCS of 13-15) is often unrecognized due to lack of radiographic evidence in the initial trauma screen (i.e. no pathology seen on head CT or MRI). Normal radiographic imaging in the acute stages can occur in diffuse axonal injury (DAI). DAI is a brain injury that results from rotational shearing forces resulting in lesions at the gray/white matter interfaces. Affected sites include subcortical white matter, corpus callosum, caudate nucleus, internal capsule, and the brainstem. Several studies have shown the potential for mild traumatic brain injury to cause major cognitive deficits. Thornhill et al. reported that 30% of mild traumatic brain injury cases have severe cognitive impairments one year after injury.[11] Furthermore, Hsiang et al. showed that the GCS rating does not always correlate with the severity of brain injury.[10, 12]

A recent study by Wei et al. in 2008 used diffusion-tensor imaging in seven patients having dual diagnosis. Diffusion-tensor imaging is a MRI technique that examines the diffusion of water in tissues, including the brain. In this study, functional anisotropy was used as an index, observing specific white-matter tracts. These data were analyzed between dual diagnosis patients, SCI only subjects, and a healthy control group. The results showed significantly decreased functional anisotropy in the genu and splenium of the corpus callosum as well as the forceps minor of the dual diagnosis group. It was concluded that diffusion-tensor imaging is a useful modality to diagnose brain injury in suspected dual diagnosis cases.[13] While further research is necessary, this technique may be most beneficial in suspected mild brain injury cases.

Cognitive and behavioral changes exist as a result of brain injury sequele. Issues of arousal, alertness, disorientation, processing, concentration, distractibility, and reasoning are often encountered. Behavioral changes can be a result of medical causes, sleep disregulation, or a direct injury to the frontal lobe.[7, 14, 15] Unfortunately, cognitive changes are often overlooked or misinterpreted as an adjustment to the injury. These erroneous conclusions may lead to a missed diagnosis.

Another diagnostic pitfall is the lack of validated criteria to establish a diagnosis of TBI. For example, the Traumatic Brain Injury Model Systems of the National Institute on Disability and Rehabilitation Research collect data on loss of consciousness, GCS scores, posttraumatic amnesia, and neuroimaging. However, not all institutions utilize these criteria to define a co-occurring brain injury. Additionally, there is poor or inconsistent documentation of cognitive status following acute trauma and clinicians often overlook the cognitive exam once patients are extubated and alert.

Neuropsychological testing is useful in diagnosing the presence of concomitant brain injury. Unfortunately, the use of neuropsychological testing is limited in acute primary spinal cord injury cases due to the time required for testing, patient pain complaints, and a lack of cooperation from patients. Instead of completing a neuropsychological battery, weekly measures such as GCS scores, orientation logs, and the mini-mental exam may be helpful during the acute care episode to give clinicians objective criteria for impairments in cognition.

Therapeutic Challenges During Inpatient Rehabilitation

The rehabilitation of patients with SCI is intense for patients and families; it also requires a high-learning curve.[7] Those with dual diagnosis have significant cognitive and behavioral impairments that adversely affect the usual rehabilitation process. In this situation, studying the mechanism of trauma and utilizing good clinical judgment may help make the diagnosis. Difficulty understanding tasks, following instructions, or communicating may give rehabilitation professionals criteria to look for a simultaneous brain injury. More severely affected individuals may have a lack of orientation, profound memory deficits, and agitation.

Posttraumatic agitation, present in up to 55% of TBI patients, could be described as verbal or physical aggressiveness, emotional lability, incontinence, disinhibition, and motoric restlessness occurring in the state of posttraumatic amnesia. It is usually self-limited and treated conservatively with environmental and behavioral modification, unless it threatens safety. The pharmacological treatment of posttraumatic agitation has shifted from the judicious use of atypical antipsychotics, sedative

hypnotics, and neuroleptics to lipid soluble, seratonergic agents, and neurostimulants.[16] For example, propanolol can be prescribed at a typical starting dose of 10 to 20 mg up to four times daily. Venlafaxine, a serotonin norepinephrine reuptake inhibitor, may be prescribed up to 75 mg twice daily. Amantidine, which is a primary dopaminergic agent with NMDA antagonist properties, has been used not only in the treatment of agitation but also for improving arousal, fatigue, and attention. The starting dose is 100 mg per day, but up to 400 mg per day can be safely prescribed.[16]

Posttraumatic epilepsy is characterized by recurrent late-onset seizures that are not attributable to any other cause but TBI and therefore can be seen in dual diagnosis. Current guidelines recommend seizure prophylaxis with phenytoin or valproic acid not more than one week post-injury.[17] These antiepileptic drugs help minimize the incidence of immediate and early seizures. Late seizures in TBI patients are not prevented by using these agents more than one week post-injury, however they may be useful for those with a high seizure risk.

Several pharmocotherapeutic medications, which are commonly used in spinal cord medicine or brain injury medicine, respectively, may cause unintended adverse affects in dual diagnosis patients. *Table 2* summarizes some of the common medications used in dual diagnosis and the potential adverse effects.

TABLE 2: Common Medications Used in Dual Diagnosis: Benefits and Adverse Effects

Medication Category and Prototypical Drug	*Desired Effect*	*Adverse Effect in Dual Diagnosis Patients*
Anticonvulsants (Phenytoin and Phenobarbital)	Seizure prophylaxis	Decreased cognition
Benzodiazepines (Diazepam)	Spasticity treatment, agitation treatment	Amnesia, sedation, memory deficits
Tricyclic antidepressants (Amitriptyline)	Treat pain and depression	Anticholinergic side effects, drowsiness
Antihypertensive (Clonidine)	Decrease blood pressure and possibly reduce spasticity	Decreased cognition
Typical antipsychotic (Haloperidol)	Reduce agitation/restlessness	Slows motor recovery and prolongs posttraumatic amnesia, sleep disturbance
Neurostimulant (Methylphenidate)	Enhance cognition	Hypertension, agitation, seizure
Dopamine agonist (Amantidine, Bromocriptine)	Enhance cognition	Decrease seizure threshold
GI motility agent (Metoclopromide)	Promote gastric motility	Decrease cognition; extra pyramidal side effects
Antiemetic (Ondansetron)	Antiemetic	Decrease cognition
Anticholinergics (Oxybutynin)	Treat overactive bladder	Sedation, impair memory and attention

Medical Complications Seen in Dual Diagnosis

Heterotopic ossification (HO), the formation of mature bone around joints, occurs in traumatic brain injury and spinal cord injury patients, with an estimated incidence of 11% to 76%, due to diagnostic variability. The most common clinical presentation is swelling, which can be confused with deep vein thrombosis, infection, fractures, or hematoma.[24] Other clinical symptoms are joint pain and decreased range of motion. In those with cognitive impairments and loss of sensation, complaints of pain may be minimal. The gold standard for an HO diagnosis is the triple-phase bone scan. X-rays are most helpful in the sub-acute period when bone has calcified. Health care professionals need to have good clinical suspicion of HO when there is swelling, limited range of motion, or pain surrounding a joint. Treatment consists of range of motion therapy, use of etidronate disodium, and anti-inflammatory drugs. Severe cases may require surgery.

Although central dysautonomias are observed in both TBI and SCI, their pathophysiology may differ *(see Table 3)*. In traumatic brain injury, an acute catecholamine surge or injury in the hypothalamus may affect the thermoregulatory centers. Common presenting symptoms include tachycardia, hyperthermia, diaphoresis, dystonia, and extensor posturing. This is called PAID syndrome (Paroxysmal Autonomic Instability with Dystonia), and it is usually a self-limiting process that occurs acutely, but its persistence correlates to increased injury severity and poor outcomes.[18] It is treated symptomatically with hydration, antipyretics, beta-blockers, non-steroidal anti-inflammatory drugs, and dantrolene sodium. Propanolol may be used at 10-20 mg bid with titration to qid dosing for adults. The starting dose for dantrolene sodium is 25 mg bid for a maximum total dose of 400 mg per day, monitoring liver function tests. Bromocriptine at a dose of 1.25 mg twice daily also can be used; it is postulated to stabilize dopaminergic systems that are involved in temperature regulation between the anterior and posterior hypothalamus.[18]

In spinal cord injury, autonomic dysreflexia (AD) occurs acutely in patients with injuries at or above a T6 level. Painful stimuli from peripheral nerves trigger a sympathetic response from the intero-mediolateral cell columns distal to the lesion, resulting in vasoconstriction, especially in the splanchnic vasculature, causing hypertension and increased cardiac output.[19] Unlike PAID syndrome, where tachycardia is part of the presentation, AD results in bradycardia as a compensatory reflex as the brainstem vasomotor reflex attempts to reduce blood pressure through the parasympathetic vagus nerve. Treatment focuses on addressing the noxious stimuli, such as impaction and urinary infection. Rapid-acting short duration antihypertensive agents (nifedipine and nitroglycerine paste) can be given while the offending stimulus is being investigated.[19]

TABLE 3: Characteristics of Dysautonomias in Dual Diagnosis

Paroxysmal Autonomic Instability with Dystonia (PAID)	*Autonomic Dysreflexia (AD)*
✓ Common following brain injury	✓ Common following spinal cord injury
✓ Tachycardia	✓ Bradycardia
✓ Hyperthermia	✓ Hypertension
✓ Diaphoresis	✓ Diaphoresis and facial flushing above the level of injury
✓ Dystonia and extensor posturing	✓ Headache
✓ Treat with beta-blockers, antipyretics, NSAIDS, and dantrolene sodium	✓ Treat by eliminating noxious stimuli and using short-acting antihypertensives

Several neuroendocrine abnormalities may exist in dual diagnosis. Hyperglycemia is seen acutely as a response to stress. Those who remain immobilized in intensive care settings or those with paralysis may have hypercalcemia due to increased bone resorption. Brain injury can induce hyponatremia caused by syndrome of inappropriate antidiuretic hormone (SIADH) secretion, cerebral salt wasting (defined as hypovolemic hyponatremia), or dehydration. It is recommended to check urine osmolality, serum osmolality, urine electrolytes, and daily sodium levels to follow hyponatremia. Other common electrolyte abnormalities such as hypokalemia, hypomagnesemia, and hypophospatemia may be related to poor oral intake in dual diagnosis patients.

Pituitary dysfunction has been reported in as much of 50% of survivors of traumatic brain injury. This is attributed to a lack of cortical or brainstem input in the hypothalamic-pituitary axis.[20] Direct injury to the pituitary-hypothalamic axis affects the release of hormones and catecholamines. The estimated incidence of hormone reduction is as follows: adrenal (15%), thyroid (5%-15%), growth hormone (18%), vasopressin (3%-37%), and gonadal (25%-80%). More than one hormone-type deficiency is present in 10%-15%[20] of all survivors. Unfortunately, the clinical symptoms of pituitary insufficiency (fatigue, apathy, depression, decreased libido, memory failure, anorexia, erectile dysfunction, weight gain) are often mistaken as effects of the TBI, leading to delayed diagnosis.

The recommended "neuroendocrine" screen for TBI patients includes morning cortisol levels, thyroxine (free T4), thyroid stimulating hormone (TSH), insulin-like growth factor (IGF-1), prolactin, follicle-stimulating hormone (FSH), and estradiol levels.[20]

Spasticity is a common complication seen in dual diagnosis, however the incidence has not been recorded. Treatment of spasticity in this group can be challenging. An agitated, brain-injured patient may not tolerate traditional therapies such as splinting, positioning, and neuromuscular blockade. Furthermore, splinting may increase the risk of skin breakdown of insensate areas. Antispasticity medications, such as diazepam have adverse side effects of amnesia and sedation. Oral dantrolene and baclofen are preferred; however intrathecal baclofen may be necessary in severe cases. A summary of major medical complications is listed in *Table 4*.

TABLE 4: Summary of Medical Complications Unique to Dual Diagnosis

✓ Heterotopic Ossification
✓ Autonomic Dysfunction
✓ DVT and PE
✓ Neuroendocrine Dysfunction
✓ Spasticity

Functional Outcomes

Overall, functional outcomes during inpatient rehabilitation for dual diagnosis patients are worse than those with SCI alone. A retrospective study in 2004 by Macciocchi et al. looked at functional independence measure (FIM) scores in dual diagnosis patients.[21] This study compared 41 subjects with SCI alone to outcomes of 41 subjects with a dual diagnosis. Both groups were matched using level of SCI, motor FIM score on admission, age, sex, and education. Compared to the SCI group alone, the dual diagnosis group had worse outcomes, including a lower admission cognitive FIM score, a lower discharge cognitive FIM score, and a smaller motor FIM change from admission to discharge.

Bradbury et al. analyzed the effect of dual diagnosis on length of stay and costs of treatment during inpatient rehabilitation. In this small study of 10 subjects, dual diagnosis patients trended toward longer hospital stays; the difference (approximately 138 days verses 100 days respectively) was not statistically significant. Cost was similar in both groups.[22]

Community reintegration is the ultimate goal for dual diagnosis patients and their families. Richards et al. compared SCI alone and dual diagnosis groups and concluded there is increased adjustment difficulties for dual diagnosis patients.[23] Furthermore, a survey-based study from Sweden by Kreuter et al. looked at SCI and TBI patient responses regarding

community integration. This study concluded that SCI and TBI groups had an overall negative quality of life and it is possible that dual diagnosis patients may have a synergistic negative effect.[25]

Case Vignette Outcome

DJ underwent a comprehensive, inpatient rehabilitation program initially with a focus on his spinal cord injury. Once cognitive issues were discovered and dual diagnosis was identified, the rehabilitation team worked with DJ in a more private setting to minimize over-stimulation until he could be reintegrated into group activities. He was started on a 100 mg daily morning dose of amantidine, which improved his concentration and wakefulness during daytime therapy sessions within one week. He also was started on a low dose of citalopram (10 mg daily) for depressed mood. He did not have any undesired side effects. Over a course of several weeks, DJ was able to learn new skills and improve his sleep hygiene. He was able to participate in group activities with other patients. At the time of discharge, he was able to use assistive devices successfully and navigate a power wheelchair. He made good functional recovery and was discharged to home with his family. He is currently continuing his rehabilitative program as an outpatient.

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