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Sigmoid Micro-Perforation and Pelvic Abscess Masquerading as Hyperactive Delirium

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Abstract

Diagnoses Background: Delirium is a common finding in elderly patients with sepsis. Early identification of the cause of delirium and treatment is important to avoid any worsening of mental or physical status. Sepsis secondary to colonic micro-perforation (CMP) in a patient with a history of diverticulosis should be high on the list of differential diagnosis. **Case Report:** We present a case of a patient who presented with hyperactive sepsis-associated delirium (SAD). Six days after the presentation, the patient started complaining of abdominal pain. An abdominal and pelvic computed tomography (CT) scan showed free air in the abdomen. The patient underwent surgical intervention and treatment with intravenous antibiotics. Pathological examination showed CMP connected to the patient's history of diverticulosis. Delirium superimposed on dementia (DSD) resulted in the worsening of both the mental and physical status of our patient with the need for placement in a nursing home.

Keywords

Delirium, Dementia, Colon Perforation, Sepsis, Diverticulosis

1. Introduction

Delirium is a common finding in elderly patients with sepsis. Sepsis secondary to CMP should be highly suspected as a cause of delirium in patients with diverticulosis. The duration between the initial presentation and the discovery of CMP may be a few days. Electroencephalogram (EEG), and neuroimaging studies should be utilized for early detection of delirium. Delirium superimposed on dementia (DSD) is a growing emerging clinical challenge, and dementia is a risk

factor for delirium.

2. Case

An 89-year-old woman presented to the emergency department, with acute hyperactive delirium. There were no other symptoms. Past medical history includes hypertension, dementia, hyperlipidemia, and diverticulosis. Past surgical history includes hysterectomy and appendectomy. There was no history of cigarette smoking, alcohol abuse, allergies, and no family history of note. Medications were simvastatin, donepezil, sertraline, lisinopril, clonazepam, clonidine, and amlodipine.

Physical examination showed a temperature of 36.8˚C, pulse of 88/minute, respiration of 16/minute, and blood pressure of 168/97 mmHg. The rest of the physical examination was unremarkable except for signs of hyperactive delirium.

Complete blood count was normal. Lyme screening test was negative. Serum glucose 94 mg/dL (70 - 100), blood urea nitrogen 22 mg/dL (7 - 25), creatinine 1.0 mg/dL (0.6 - 1.2), sodium 140 mmol/L (136 - 145), potassium 4.6 mmol/L (3.5 - 5.1), chloride 102 mmol/L (100 - 108), calcium 9.6 mg/dL (8.6 - 10.2), phosphorus 2.9 mg/dL (2.5 - 5), magnesium 1.5 m Eq/L (1.3 - 1.9), folate 19.8 ng/mL (5.9 - 24.8), vitamin B12 level 532 pg/mL (180 - 914), thyroid stimulating hormone 0.372 IU/mL (0.34 - 5.6), free T_4 0.83 ng/dL (0.78 - 1.64), T_3 0.53 ng/mL (0.87 - 1.78). None of the laboratory results revealed the cause of the delirium including renal, hepatic, electrolytes, thyroid function tests, or vitamin abnormalities.

Chest radiograph was normal, and no free air was seen under the diaphragm. Electrocardiogram showed atrial fibrillation. Head CT scan showed age-related atrophy, without acute findings. No clear cause of delirium was identified. Although no clear reason was identified for the delirium, treatment was started with haloperidol and lorazepam, to prevent self-harm and to treat agitation. Six days after the presentation the patient started complaining of abdominal pain. Abdominal examination revealed lower abdominal tenderness. Abdominal and pelvic CT scans showed pneumoperitoneum, a collection of fluid in the rectosigmoid junction, and a presacral pelvic abscess.

Exploratory laparotomy showed purulent peritonitis and an abscess in the cul-de-sac. Pelvis abscess drainage and sigmoidectomy were performed. Examination of surgical specimen revealed acute diverticulitis with CMP. The peritoneal samples grew *Klebsiella* and *Escherichia coli*. Therapy with intravenous Piperacillin-Tazobactam was given for ten days. Delirium resolved and the patient was discharged to a nursing home because of worsening dementia and physical status.

3. Discussion

Delirium is an acute or subacute onset of reversible neuropsychiatric syndrome which is caused by multiple factors. With the increase in the aged population, further increases in delirium prevalence seem likely [1]. Delirium is associated

with many adverse outcomes including cognitive impairment, functional decline, prolonged hospitalization, and increased need for nursing services [1].

4. Risk Factors

With advanced age, the accumulation of neuronal and cerebral microvascular damage disrupts the energy uptake pathway leading to inadequate energy uptake in the brain, resulting in the development of delirium in elderly patients [1]. A pivotal factor is diminished connectivity, micro-angiopathy with impaired blood flow autoregulation, increased blood/brain barrier permeability, changes in cerebrospinal fluid dynamics, and weakened mitochondrial performance a pro-inflammatory involution of the immune system may also affect neurons, and even cause the progression of delirium to dementia [2]. DSD is a growing emerging clinical challenge, and dementia is a risk factor for delirium [3].

Patients with emotional disturbances, Alzheimer's disease, childhood neurodevelopmental delays, history of alcohol abuse, opioid and benzodiazepine use, malnutrition, brain function impairment, dementia, acute somatic diseases, infections, sleep disorders, electrolytes imbalance, pain, surgical anesthesia, being a patient in ICU, and visual and auditory impairment have a high risk of developing delirium [1].

5. The Role of Inflammatory Cytokines

Delirium is associated with inflammatory cytokines [4]. There is also some evidence that frailty is a state of chronic low-grade inflammation [4]. Tokuda *et al* studied SAD. Their findings clarified the role of inflammatory cytokines and mediators (e.g., C-reactive protein (CRP), interleukin (IL)-6, tumor necrosis factor alpha, IL-1RA, IL-10, and IL-8) in contributing to the subsequent neurobehavioral and cognitive symptoms that are characteristic of delirium [5]. Noah *et al* assessed the role of preoperative inflammatory mediators and postoperative delirium. Higher preoperative IL-6, CRP, and neopterin levels were associated with postoperative delirium [4], which supports the role of inflammatory cytokines in the development of delirium [4]. Finally, evidence of the unique vulnerability of the brain of certain patients has been described [4]. The interplay between cytokine surge and innate brain vulnerability is fertile ground for future exploration [4].

6. Assessment

DSD, especially hypoactive, is often under-recognized because of its overlap with dementia [1]. The diagnosis of delirium is primarily clinical. Evaluation should include a meticulous history and physical examination [1], with the utilization of neuropsychological assessment tool scales [6].

Ancillary investigations may include routine blood parameters, EEG, and radiographic studies. Hanna *et al* reviewed 1,516 EEG records of patients with delirium. Delirium was found consistently associated with global decreased alpha

band connectivity [7]. Their findings highlighted the potential for network dysconnectivity as a possible pathophysiologic mechanism to explain EEG findings in delirium. The pathophysiology of network dysregulation, in delirium, is not purely electrical, because of the presence of metabolic and chemical signatures [7]. Hanna *et al* suggest using EEG to detect delirium, especially in cases of DSD [7].

7. The Roles of Neuroimaging Studies

Akhtar *et al* found that the use of head CT to diagnose the etiology of delirium varied widely and yield has declined [8]. The presence of focal neurological deficits was a consistent factor that increased the diagnostic yield of head CT scans in patients with delirium [8].

Song *et al* evaluated the utilization of brain functional magnetic resonance imaging (fMRI) in ICU patients who develop delirium [9]. Abnormal resting-state brain activity in the left superior frontal gyrus and structural changes in the frontal lobe, temporal lobe, corpus callosum, hippocampus, and cerebellum were observed in ICU patients who developed delirium. Brain fMRI study by van Montfort *et al* has yielded similar results to the EEG findings. The fMRI showed decreased functional connectivity and overall network disintegration during delirium [10]. This is in accordance with the EEG-measured functional connectivity by Hanna *et al* [7]. Brain fMRI examination is recommended for early detection of delirium which facilitates early intervention for ICU patients, reduces the length of hospital stay, and improves patients' prognosis [9]. Although imaging in delirious patients, who may not be able to cooperate or remain still, presents significant challenges.

Nitchingham *et al* documented regional cerebral hypometabolism on ^{18}F-fluorodeoxglucose positron emission tomography scan in patients with delirium, and found that thalamic hypometabolism is unique to delirium [11].

8. Treatment

Treatment should focus on treating the precipitating illnesses [1]. Interventions are divided into non-pharmacological, and pharmacological measures [1].

Non-pharmacological measures include orientation, cognitive stimulation, early activity, sleep improvement, sensory impairment correction, pain management, supplemental nutrition, and enhancing oxygen delivery [1].

Pharmacological agents that may be used include antiepileptic drugs such as valproic acid, alpha-2 agonists like dexmedetomidine [12], melatonin to enhance the sleep-wake cycle, vitamin B1 replacement in cases of deficiencies [1], and antipsychotic medications like quetiapine, haloperidol, olanzapine, or risperidone [1]. Antipsychotics should only be used in patients with severe distressing symptoms, or safety concerns, due to the potential harm in patients with pre-existing dementia [3].

9. Outcome and Prognosis

Patients usually recover within 7 - 10 days, rarely developing into chronic delir-

ium, or have long-term effects on cognition [1]. There is a close interaction between delirium and dementia. Dementia is a risk factor for delirium and delirium is a known risk factor for newly developed dementia or worsening of dementia [3]. The risk of developing post-delirium dementia in elderlies is eight times higher than in normal older adults [1]. This emphasizes the importance of delirium prevention to avoid any post-delirium cognitive decline [13].

10. Conclusions

Sepsis is one of the most important and strongest factors for delirium. In a patient with diverticulosis, CMP should be included in the differential diagnosis of delirium. The duration between the initial presentation and the discovery of CMP may be a few days.

DSD is a growing emerging clinical challenge, and dementia is a risk factor for delirium [3]. Like the case of our patient, delirium may result in worsening mental and functional status.

Since sepsis and delirium are both closely associated with increased morbidity and mortality, it is important not only to prevent but also to promptly diagnose and treat SAD [5].

Given the importance of DSD, it is essential to educate healthcare providers on the best approach for delirium management and treatment [3].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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