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Catatonia Eight Years after Head Injury

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Authors' contributions

This work was carried out in collaboration between all authors. Author LG redesigned and rewrote parts of the paper and formulated the strategies of interventions. Authors KN, NV, ER, JC, JK, EAO and CC wrote parts of the manuscript, did the literature search, prepared the tables and contributed significantly through the process. Author RGB designed the study, coordinated the process and wrote parts of the manuscript. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

We present the case of a male patient with traumatic brain injury leading to gradual deterioration in functioning over several years, culminating in a state of prolonged treatment-resistant catatonia complicated by an inability to perform activities of daily living and necessitating total care by nursing staff. Extensive diagnostic evaluation did not reveal a convincing etiology of patient's catatonia. Despite several empiric treatment modalities administered early in the course of catatonia, patient's condition continued to decline. We did not observe sustained changes in response to high dose lorazepam, selegiline/minocycline coadministration, or ECT alone, but rather exponential improvement from the combination of medications and ECT.

Keywords: Catatonia; traumatic brain injury; ECT; monoamine oxidaze.

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1. INTRODUCTION

Traumatic brain injury (TBI) is a common condition and a significant cause of morbidity and mortality. The Centers for Disease Control and Prevention (CDC) has recognized the need to reduce the burden of unintentional injuries and their long term consequences [1]. The CDC initiatives focus on multiple aspects ranging from identification and prevention of TBI, recognition and management and interventions to improve long term health outcomes [1].

Catatonia is a less studied consequence of TBI [2]. The paucity of reports of TBI related catatonia could be due to a multitude of medical problems from initial trauma and significant anatomical variability of injuries and diversity of clinical presentations masking the syndrome.

However, TBI patients exhibited EEG patterns suggestive of diminished inter-hemispheric coordination. These abnormal findings were associated with weakened structural integrity of white matter tracts [3]. In Franke et al., blast-related mild traumatic brain injury (mTBI) was associated with increases in low frequency power on EEG [4]. Through an EEG index, it was found that EEG was able to distinguish between severities of mTBI and was sensitive to detect injury effects six months after their mTBI, notable for an increase in slower frequency activity revealing information to abnormalities in the blood brain barrier [5]. In Sponheim et al., two and a half years after injury there was reported decreased frontal phase synchrony on EEG [6].

Slow oscillations have been positively correlated with increased frontal white matter T2 relaxation time in moderate to severe TBI, possibly suggesting reduced white matter excitatory cortical input [4]. In severe TBI patients who completed rehabilitation and made cognitive gains as in Castellanos et al., there was a decrease in low-frequency connectivity [7]. Overall as in Franke et al., EEG changes are appreciable in the chronic period and can be useful for tracking the course of recovery and enduring susceptibilities to injury [4].

The dynamic characteristics of the EEG allow one to characterize the severity of the patient's current state, the ability to identify brain structures, and to define cortical lesions [8]. As in Sharova et al., the most important prognostic factors were found to be the baseline and reactive frequency characteristics of the EEG power spectrum, particularly when assessed two to three months after a trauma [8].

2. CASE REPORT

Mr A is a 26-year-old Caucasian male who presented to the emergency room at the University of California, Irvine (UCI) with symptoms of mutism, rigidity, withdrawal, negativism, and waxy flexibility.

Eight years prior to the admission he had a traumatic brain injury from a motor vehicle accident. After recovery from his injuries, he was able to complete high school and a four-year college. During this time, his family noticed a progressive deterioration in functioning, bursts of irritability and anger. After graduation from college, the patient was not able to find employment for 2-3 years. He became increasingly withdrawn; at times he would not talk for days. The duration and frequency of episodes of mutism increased in the following months, worsening to the point that he would not speak at all and required prompting for all activities. At this juncture, he was admitted for investigations at a large private hospital (see Table 1 for list of workup completed). Shortly after, he was again hospitalized for an extensive three-week workup at another private hospital (see Table 2 for the list of workup done). The following tests were notable: low IgA, positive streptococcus antibody screen with elevated ASO titers, positive CSF anti-neuronal antibodies, and mildly elevated alpha-1 and -2 on serum protein electrophoresis. Magnetic resonance imaging (MRI) showed mild cerebellar atrophy not different than the MRI one year before and continuous EEG showed the presence of very mild slowing. He was subjected to the following treatments: amoxicillin-clavulanate, tinidazole, a course of intravenous immunoglobulin (IVIG), lorazepam 1 mg every four hours via intravenously, olanzapine, mirtazapine, and methylprednisolone, with no clinical effect.

At the time of admission to UCI, there were several failed the attempts to find a definitive diagnosis. The patient showed progressive deterioration despite treatment. As he had no oral intake for days prior to the admission, the patient was started on lorazepam, titrated to 10 mg a day, and showed improvement in oral intake. However, the patient would not take per os medications requiring him to receive the

Lorazepam intramuscularly. Subsequently, the patient was transferred to the intensive care unit for continuous monitoring. Lorazepam was increased to at a total dose of 72 mg a day with debatable further improvement in symptoms. Trials of Olanzapine or lithium did not show significant benefit.

After transfer back to medical psychiatry unit selegiline was started and titrated up to 12 mg a day, along with a lorazepam dose of 4 mg a day. ECT was started and by the second treatment, the Bush-Francis score decreased from 34 to 24. However, medications had to be stopped as his legal status changed requiring the team to apply for another permission from the court to give medications. During those four days, the patient continued to receive ECT; however his Bush-Francis catatonia score returned to the previous level. After we restarted his medications, we also added minocycline 100 mg PO twice a day, and continued the ECT as scheduled, at three times a week. His catatonia score decreased to 23, 14 and then 0, with these scores determined within a two-day interval. Four days later he began speaking in full sentences, became cooperative with staff and no longer required a sitter. For the following week, he maintained his significant clinical improvement with continued gradual improvement. He received a total of nine bilateral ECT treatments. Due to his significant improvement, the patient and the family declined further ECT treatment.

He was discharged with follow-up at a partial hospitalization program (PHP). From this point on, the Montreal Cognitive Assessment remained at 30. Patient continued to do well for one month, at which time he started to again display catatonic symptoms. At a follow-up appointment, it was discovered that the patient had decreased medication compliance in the week prior. Fortunately, the symptoms resolved in two days, but just weeks later reemerged- only to identify that the cause was likely decrease in the frequency of PHP program from five days to three days. The increase in days led to the symptom resolution. A few weeks later the frequency was decreased to three days a week uneventfully.

3. DISCUSSION

The brain is an electro-chemical organ. The implication of this is multifaceted. Understanding the TBI related brain changes is paramount. The clinical course over the eight years starting with severe TBI and progressing to treatment-resistant catatonia is just an example of often subtle but pervasive long term effects of brain injury. We believe that catatonia secondary to TBI should be considered in similar patient population if no other plausible causes are identified.

There are anecdotal reports of catatonia following TBI [9-11], including one case in which a 15 year-old patient made a full recovery only to develop catatonia and psychosis ten months later [12]. Another published case described a patient who sustained a head injury followed by a gradual decline in academic performance [13]. Two years after the injury, the patient developed an abrupt onset of irrational speech, irritability, and negativism. Over the next few years he developed signs of catatonia; resistant to benzodiazepines but successfully treated with risperidone. Another publication described a patient with a TBI who developed atypical catatonia consisting of primarily language processing and social interaction deficits that responded well to lorazepam [14]. A related syndrome known as lethal catatonia, in which fever and hyperactivity progress to stuporous exhaustion, has also been found to follow TBI [11]. In a retrospective study assessing the etiologies of catatonia in 75 consecutive cases, Smith, et al., identified six patients who had a history of traumatic brain injury [10]. Similarly, Wilcox, J.A. found catatonic patients were more likely to have had a prior history of brain injury when compared to manic, depressed or surgical patients used as controls [15]. Ahuja N. proposed that a common theme in the origin of organic catatonia was lesions in and around the third ventricle [16].

There is no consensus on how to treat catatonia in persons with brain injuries; however, clozapine and risperidone have been found to be effective in some cases where benzodiazepines failed [9 17]. For example, Rommel and colleagues described a woman involved in a major motor vehicle accident that caused frontal lobe injury and subsequently catatonia one month later who was effectively treated with clozapine [9]. Minimally-responsive head injury survivors may have chronic catatonia reversed years after an accident [18].

At the climax of his catatonia, Mr. A did not respond to single treatment modalities, e.g., medication, ECT or behavioral interventions, but a combination of all.

Table 1. Diagnostic testing at the first private hospital

Total hospital days: 3

Date	Diagnostic tests	Results	Normal range
HD*1	IgA – low	<40	70-400 mg/dL
	IgG	971	700-1600 mg/dL
	IgM	152	40-230 mg/dL
	TSH	.65	.46-4.68 uIU/mL
HD1	Streptococcal Ab* screen	Positive	Negative
	ASO semi-qnt titer – elevated	400	<200 IU
HD2	Lumbar puncture, CSF	Glucose: 54	40-70 mg/dL
		Protein: 47	12-60 mg/dL
		RBC 30, WBC 1	
		Clear & colorless	
HD2	CSF – viral culture -Adenovirus, Cytomegalovirus, Herpes simplex virus, Enterovirus, Respiratory syncytial virus, Varicella –zoster virus, Influenza virus, Parainfluenza virus	No virus isolated	
HD2	CSF – autoimmune Encephalopathy Evaluation -NMDA-R Ab; VKGKC-complex Ab, GABA-B R Ab, AMPA-R Ab, ANNA-1, ANNA-2, ANNA-3, AGNA-1, PCA-1, PCA-Tr, Amphiphysin Ab, CRMP-5-IgG, ANA	All negative	
	CSF – Histoplasma Ab, Coccidioides Ab, HSV 1 DNA PCR, HSV 2 DNA PCR,	None detected	
	CSF- VDRL	Non reactive	
	Urine – Trachomatis RNA, Neisseria Gonorrhoeae	Not detected	
	EEG	unremarkable	
HD1	CT chest, abdomen, pelvis with contrast	1 cm RUL* nodule with minimal central calcification; otherwise unremarkable CT scan	Note: follow-up in 3 months to monitor size
HD1	MRI lumbar spine with and without contrast	Unremarkable	
HD1	MRI thoracic spine with and without contrast	Unremarkable	
HD1	MRI cervical spine with and without contrast	Unremarkable	

Date	Diagnostic tests	Results	Normal range
HD1	MRI brain with and without contrast	Unremarkable	
HD2	Echo 2D	Normal echo; LVEF* normal (55-60%), RV* normal size and function	
HD1	Hb A1c (normal)	5.1%	
HD1	PT, INR	PT: 10.2 INR: 1.0	PT: 9.7-11.8 INR: .9-1.2

HD – hospital day; RUL – right upper lobe; LVEF – left ventricular ejection fraction; RV – right ventricle
Given lorazepam for suspected catatonia without improvement
Suspected PANDAS (pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections)
Dc with antibiotics: amoxicillin-clavulanate 875mg-125mg oral tablet; 1 tablet q12 hours & tinidazole 500 mg oral tablet: 1.5 tablets BID
Follow-up appointment with neuropsychiatrist, immunologist

Table 2. Diagnostic testing at the second private hospital

Total hospital days: 21

Date	Diagnostic tests	Results	Normal range
HD*14	IgA	<7	82-453 mg/dL
HD14	IgG	1070	751-1560 mg/dL
HD14	IgM	188	46-304 mg/dL
HD14	IgE	5	<114 kU/L
HD2	Total bilirubin mildly elevated	1.3	.1-1.2 mg/dL
HD2	Direct bilirubin mildly elevated	.4	<.3mg/dL
HD20	Total bilirubin	.7	.1-1.2 mg/dL
HD2	Plasma Ammonia	68	<42mcmol/L
HD20	Plasma ammonia	44	<42mcmol/L
HD2	TSH, Free T4 – normal	1.28, 1.24	TSH: 0.39-4.60 Free T4: 0.93-1,70
HD2	Vitamin B12	1647*	211-911
HD2	Folate	9.5	2.5-22 ng/mL
	Vitamin D 25 hydroxy	35.7	20.0-80.0 ng/mL
HD2	LP opening & closing pressure	140mmH20, 100mmH20	
	CSF glucose, protein, WBC	68,23,0 WBC, 0 RBC	
HD2	CSF: Anti-neuronal cell Ab	2.8	0-1.0
	CSF anti-RI Ab (paraneoplastic lab testing), NMDA-R Ab	negative	

Date	Diagnostic tests	Results	Normal range
	CSF: neuron specific enolase	2.9	<8.9
	CSF fungus culture, virus, bacteria; West Nile IgM & IgG, VDRL, CMV IgG, Toxoplasma IgM & IgG, HSV PCR, Enterovirus RT PCR	Negative for fungus, virus, bacteria Negative results/nonreactive	
	CSF: Lyme Disease Abs, IBL	No bands detected	
	CSF: Cysticercus Ab	<0.75	<0.75
	CSF ammonia	40	<42mcmol/L
	CSF India Ink	negative	
	CSF: Coccidiomycosis IgM & IgG, Cryptococcus antigen	negative	
HD14	Serum Mycoplasma pneumonia antibody IgG	1.92	<=,90
HD14	Serum Mycoplasma pneumonia antibody IgM	587	<770U/mL
HD14	Serum Angiotensin Converting Enzyme	7	9-67 U/L
HD14	Serum B henselae IgG & IgM screen	negative	
HD14	Serum enterovirus PCR	not detected	
HD14	Serum Tropheryma whippelli DNA	not detected	
HD17	Creatine Kinase	48	0-171 U/L
HD2	RPR	negative	
HD14	FTA-ABS	nonreactive	
HD2	HIV Ag/Ab combo	negative	
HD3	HBV core Ab IgM, HCV Ab	negative	
HD3	HBV surface Ab	110	>12 MIU/MLpost vaccination protection
HD14	Anti neutrophic cytoplasmic Ab (ANCA)	<10	<10
HD5	Immune electrophoresis (IEP)	Normal pattern, no monoclonal proteins detected	
HD14	Serum protein electrophoresis (PEP) – mildly elevated Alpha 1, serum & alpha 2 serum; normal Total protein, albumin, beta, total gamma	Alpha 1: 0.37 Alpha 2: 1.00	Alpha 1: 0.18-0.34G/dL Alpha 2: 0.43-0.87
HD5	Serum ASO	297	<116 IUM/mL
HD14	CMV DNA PCR	0	<5 copies
HD14	EBV DNA PCR	0	<5 copies
HD14	Quantiferon TB Gold	negative	
HD14	C-Reactive Protein	.9	<5mg/L
HD1	PT	14.2	11.9-14.4sec

Date	Diagnostic tests	Results	Normal range
HD1	INR	1.0	<3.6
HD1	PTT	46	22-37
HD2	Urine toxicology screen	Negative	
HD2	Urine analysis	Negative	
HD1	EKG	Prolonged QTc interval	
HD7	CSF: NMDAR Ab	1:1, negative	1:1
HD7	Serum: NMDAR	<1:10, negative	<1:10
HD2	MRI Brain with and without contrast	No mass, hemorrhage, or acute infarction. No evidence of rhomboencephalitis. Mild cerebellar atrophy	
HD2	Chest X-ray	Normal exam of the chest	
HD2	Abdominal Ultrasound	4.0x4.5x3.8 cm liver mass on left lobe with internal blood flow	
HD3	MRI Abdomen and Pelvis with and without contrast	Liver with exophytic mass measuring 4.9x4.1x4.3cm within the lateral segment left lobe likely focal nodular hyperplasia	
HD3	EEG	Mildly abnormal EEG due to presence of very mild slowing	
HD8-9	Continuous Video-EEG monitoring	Normal	

HD- Hospital day, Tx: 5 doses if IVIG, no clinical effect, Lorazepam: 1 mg q4 hrs injection, Olanzapine 5 mg, Mirtazapine 45 mg, Discharged with, -4 mg of methylprednisolone -Vita D3, 1000units/ tab, 2 tabs po daily, 60 days, -Megestrol acetate 400 mg BID, 30 days, -Mirtazapine: 45 mg daily

Table 3. Diagnostic testing at UCI

Total hospital days: 96

Date	Diagnostic tests	Results	Normal range
HD*7	CK	75	30-223
HD7	C-Reactive Protein	.2	0-1.0mg/dL
HD9	ANA Screen	negative	
HD9	Ribosomal Protein P Antibody	1	0-40 AU/mL
HD15	Serum NMDA Ab, IgG	<1:10	<1:10
HD18	Anti ENA Ab	14	0-19 units
HD18	Serum tryptase	3.4	<11.5ng/mL
HD18	Anti Double-Stranded DNA and ANCA screen	negative	
HD65	Lupus Sens aPTT screen	45.7	<48.1
HD65	Lupus anticoagulant DRVVT and HEX test	negative	
HD31	IgA – low	<7	68-378mg/dL
HD31	Anti-TPO antibodies	17	<9 IU/mL
HD31	ANNA-1 (Hu), ANNA-2 (Ri), PCA-1 (Yo)	Not detected	Note: confirmatory testing will not be performed
HD31	ASO elevated	400	<200
HD31	Lyme Disease Antibody total - negative	.90	>1.09
HD32	TSH	151	0.50-5.00 uIU/mL
HD32	CSF tube 4	Glucose 68, protein 40, clear, 0 RBC, 3 nucleated cells, 90% lymphocytes, 10% monocytes	
HD32	CSF bacterial & viral culture	No growth, no viruses isolated	
HD32	CSF: Lyme titers (Borrelia Burgdorferi Ab, ELISA), VDRL	Negative	
HD32	CSF: anti-neuronal cell Ab	.8	0-1.0 units
HD32	CSF: anti-Neuronal cell antibody	20	0-54 units
HD32	CSF: Paraneoplastic panel: ANNA-1, ANNA-2, ANNA-3, AGNA-1, PCA-1, PCA-2, PCA-Tr, Amphiphysin Ab, CRMP-5, P/Q-type Calcium Channel Ab, N-type Calcium Channel Ab, AChR Ganglionic Neuronal Ab, Neuronal (V-G) K+ Channel Ab	negative	
	CSF: Striational (striated muscle) Ab – High	1:480	<1:120
HD32	CSF pathology	No malignant cells	
HD32	Spinal LDH	<25	<25 U/L

Date	Diagnostic tests	Results	Normal range
HD32	Thyroglobulin Ab	Negative (note: suboptimal specimen)	0-4,0 IU/MI
HD34	BNP	6	0-100pg/mL
HD34	Troponin	<.03	<.03 ng/dL
HD39	Iron Panel: transferrin, Iron, TIBC, Percent Saturation, Ferritin	normal	
HD62	Vitamin B12	750	180-1241 pg/mL
HD62	Vitamin D 25-hydroxy total	23.5	>30 ng/mL
Admission	Urine analysis	negative	
Admission	Comprehensive urine drug screen	No drugs detected	
Admission	Comprehensive blood drug test: Alcohol Ethyl	Not detected	
Admission	Blood cultures	No growth, no anaerobic growth, no acid fast bacilli isolated, no fungus isolated	
HD10	MRI Brain with and without contrast	Calipers of the ventricles, sulci, basal cisterns, are prominent for age 25, including some cerebral atrophy. No other abnormalities detected	
HD15	CT Abdomen/Pelvis with contrast	5.5 cm enhancing mass with central scar in left liver lobe suggestive of focal nodular hyperplasia	
HD15	CT Chest with contrast	Calcified granuloma located on RUL*. No intrathoracic neoplastic or infectious process	
HD20	MRI Abdomen with & without contrast	Liver lesion 5.5x4.3x4.5cm mass consistent with focal nodular hyperplasia; no acute abdominal findings	
HD60	MRI Abdomen with & without contrast	Stable 5 cm mass in left hepatic lobe with central scar, consistent with focal nodular hyperplasia	
HD20	Ultrasound of Scrotum/Testicle	No focal testicular lesions noted	
HD32	Thyroid Sonogram	Normal. No focal thyroid nodule or mass	

HD- Hospital day; RUL- right upper lobe

Table 4. Medication table at UCI

Medication	Date and dosage
Lorazepam	Up-titrated: in mg/day: 3 →10 →16 → 24 → 28 → 32→ 48→ 72mg for 3 days. Since patient showed little improvement, he was tapered off from 72 → 36→ 24. Patient had poor PO intake so dose was increased back up to 36 for 3 days and titrated down → 24 → 12 → 8 → 6 → 4→ 2 → 4 up-titrated due to withdrawal symptoms of poor PO intake → 2; patient was discharged with 2 mg TID
Lithium	HD* 31: started 600 mg daily PO→ 900 mg daily; stopped HD 45
Olanzapine	HD 29: started 5 mg daily PO→ 10 mg daily→ 30 mg daily; stopped HD 45
Selegiline (transdermal)	HD 53: started 6 mg daily patch→ 12 mg daily patch until HD 96; discharged with 12 mg daily patch
Minocycline	HD 81: started 200 mg/day until HD 96; discharged with 200 mg/day
Memantine	HD 85: started 5 mg/day→ 10 mg/day until HD 96; discharged with 10mg/day
ECT	Received 9 times starting HD 68 After session #6, patient began speaking in phrases After session #7, patient was able to recall long-term memories <u>Busch-Francis Catatonia Assessments:</u> Before ECT: 32 After ECT #4: 14, 13 After ECT #5→ 0 and remained 0
Vancomycin/ Piperacillin-Tazobactam	Patient developed pneumonia likely secondary to aspiration and completed a 10-day course of antibiotics.
Amphetamine/ Dextroamphetamine	HD 89: start 10 mg → 20 mg → 30 mg

HD- hospital day

Patient was supplemented with thiamine 50mg daily, cholecalciferol 400 IU daily, and fish oil. He was initially given a trial of valproic acid but refused any PO intake at that time so the drug was discontinued. He was given methylprednisolone 1x for possible autoimmune encephalitis with possible etiologies including post-streptococcal infection related to autoimmune PANDAS versus post-Lyme infection vs paraneoplastic syndrome; however, it was discontinued

Functional imaging can provide further evidence of this. In a study (5), behavioral interventions showed cerebral activation in executive control and default mode networks, using functional magnetic resonance imaging. In a recent meta-analysis of studies using diffusion tensor imaging (DTI), there was significant correlations between long term changes in large tracts and cognition [19].

In case of Mr. A, the patient was discharged with partial hospitalization program (PHP) follow up and psychiatrist appointments to manage his medications. The ECT treatment was stopped without immediate deterioration. Two situations deserve more consideration. First, when the compliance to medications decreased after hospitalization, the symptoms of catatonia started to resurface (mutism, waxy flexibility and stereotypical behavior) which successfully remitted with better medication compliance. Second, when the frequency of the PHP decreased from five to three days a week due to insurance considerations, the same scenario re occurred.

The memory, immediately after the resolution of most of Mr. A's catatonia symptoms, was poor, including his immediate memory as well as his long-term memory. After initiation of memantine immediate memory improved within the first week (he started to remember details of the visits he just received from friends). Long term memory continued to improve over the weeks following, first devoid of emotions, but as time passed by, at eight weeks he started to recall the fear and horror of first days of psychiatric hospitalization when an occasional code was called and knew he could not initiate any body movements.

4. CONCLUSION

TBI is a common condition and a significant cause of morbidity and mortality. Catatonia as result of TBI can present as a very insidious condition, happening at various intervals from the date of initial trauma. The complexity of TBI anatomical variability and the consequences, both short and long term from the original trauma can explain sparsity of research in catatonia secondary to TBI. Therefore, we believe that is very important to increase physician's awareness of this condition in patients with TBI.

CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this paper and accompanying images.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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