

Neurocysticercosis: Isolated Lesion in the Left Middle Frontal Gyrus

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ABSTRACT

Objective: Neurocysticercosis (NCC) is one of the most common parasitic infections of the central nervous system. We present a case study of a 21-year-old African man with an isolated NCC lesion to the left middle frontal gyrus, which is also known as the dorsolateral prefrontal cortex (dlPFC).

Method: A neuropsychological evaluation was requested by the patient's inpatient psychiatry team regarding worsening attention and depressive symptoms approximately 6 months after NCC diagnosis and treatment.

Results: Neuropsychological findings revealed deficits in the aspects of executive functioning, attention, working memory, and significant depressive symptoms.

Conclusion: To our knowledge, this is the first case study of its kind demonstrating deficits in cognitive functioning consistent with the dlPFC lesion location. Sociocultural and linguistic considerations, clinical findings, and limitations are discussed.

Keywords: Neurocysticercosis; Left medial frontal gyrus; dlPFC; Attention

INTRODUCTION

Neurocysticercosis (NCC), an infection caused by the larval stage of the pork tapeworm *taenia solium*, is one of the most common parasitic infections of the central nervous system (Wallin & Kurtzke, 2004). Following ingestion, the tapeworm embryo passes through the stomach lining, moves through the bloodstream, lodges into blood vessels of various organs, and develops into cysts approximately 2–3 months later (Nash & Garcia, 2011). Clinical manifestations are dependent upon the number and location of lesions and may include seizures (the most common manifestation worldwide), focal neurological deficits, intracranial hypertension, or cognitive decline (Villarán et al., 2009). Lesion location has served as a subtle predictor of clinical outcomes. For example, lesions located in the parenchyma are associated with seizures and typically have a good prognosis (Nash & Garcia, 2011). By contrast, extraparenchymal NCC can lead to mass effects or hydrocephalus with a generally worse outcome (Nash & Garcia, 2011). Although NCC is considered as the main cause of acquired epilepsy in developing countries, it is becoming more frequent in industrialized nations due to immigration from the endemic areas (Villarán et al., 2009). In the United States, patients typically present with seizures, hydrocephalus, and headaches, with associated lesions

in the parenchyma, ventricles, subarachnoid space, and spinal cord (Wallin & Kurtzke, 2004).

Individuals with NCC are typically diagnosed based on epidemiology (e.g., travel to endemic regions), symptoms (severity of seizures, headache), and brain imaging (presence of cysts/calcalcification; Del Brutto et al., 2001). These clinical markers may be useful in determining the duration and degree of treatments (Nash et al., 2006). Treatment for NCC typically includes anthelmintic drugs to kill the viable parasite, antiepileptic or analgesic drugs to treat neurological symptoms (seizure or headache), and steroids to reduce inflammation (Nash & Garcia, 2011). In some cases, surgical intervention such as neuroendoscopy may be used to remove cysts, or shunt placement could be used to treat hydrocephalus (Jimenez-Vazquez & Nagore, 2008; Torrez-Corzo et al., 2010).

Given the vast heterogeneity of symptom presentation and cognitive changes following NCC, a neuropsychological evaluation can serve as a valuable resource to evaluate the changes in cognition. Neuropsychological research has shown lower attention, processing speed, memory, executive functions, and quality of life among NCC patients compared to healthy controls, with correlations related to number of lesions (potentially due to volume loss) as compared to the lesion site alone (Nau et al., 2018;

Varghese et al., 2016; Wallin et al., 2012). With appropriate treatment of NCC, however, longitudinal studies have found improvement in cognitive and social function (Forlenza et al., 1998). Ramirez-Bermudez et al. (2005) found correlations between dementia and NCC, showing that an increase in the number of parasitic lesions in the frontal, temporal, and parietal lobes were associated with cognitive and functional decline. After 6 months post-treatment, ~80% of patients in their study no longer met the criteria for dementia and significantly improved, although some continued to show a mild cognitive decline (Ramirez-Bermudez et al., 2005). In terms of psychiatric comorbidities, patients with resulting intracranial hypertension were noted to have an increased risk of psychiatric symptoms, particularly depressive disorders (Forlenza et al., 1998).

Overall, NCC is a debilitating condition and tracking cognition during the onset and course of treatment may be useful in disentangling the heterogeneous nature of the disease. We here present a case study of a 21-year-old man with an NCC lesion to the left middle frontal gyrus. To our knowledge, this is the first case study that has evaluated an isolated NCC lesion in this region, also known as the dorsolateral prefrontal cortex (dlPFC). The evaluation was requested by the patient's inpatient psychiatry team regarding worsening attention and depressive symptoms approximately 6 months after NCC diagnosis and treatment. As such, a full neuropsychological battery of measures, particularly in the areas of attention, processing speed, and executive functioning were administered.

MATERIALS AND METHODS

Clinical Background

The patient was a 21-year-old right-handed man with 12 years of formal education from Africa. Though English was not his primary language, he was fluent, his schooling was primarily in English, and he preferred testing in English. The patient relocated to the United States to pursue higher education within the last year. Within 1 month of his arrival to the United States, the patient had one tonic-clonic seizure, which prompted his admittance to the hospital. Neuroimaging revealed a cortically based lesion or scolex (i.e., head/anchoring organ of the tape-worm; pathognomonic sign of NCC) with adjacent vasogenic edema in the left middle frontal gyrus consistent with a diagnosis of NCC (Fig. 1). He was treated with the following daily medications: albendazole (14-day course, 400 mg twice per day (BID); antiparasitic), levetiracetam (ongoing, 750 mg BID, anti-seizure), and prednisone (7-day course, 60 mg daily; steroid). These medications are useful in reducing the parasite load within the central nervous system and improving the clinical prognosis (Del Brutto, 2014). Levetiracetam and albendazole are both associated with a reduction in the number of seizures that may occur as a result of NCC (Romo et al., 2015). After 3–4 days of hospitalization, the patient was discharged with a referral to Psychiatry for medication management and therapy, as worsening depressive symptoms and cognitive difficulties were noted.

Approximately 6 months following his hospitalization, the patient was admitted to an inpatient psychiatric unit after a suicide attempt, in which he consumed 17,100 mg tablets of sertraline. After stabilization, neuropsychological services were

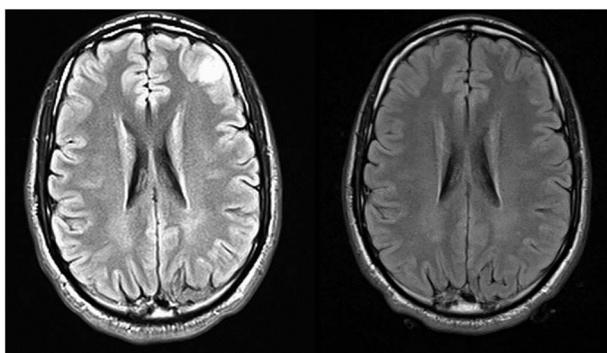


Fig. 1. A T2 FLAIR brain image in axial views from baseline (left) to 1-year follow-up (right). An 8 × 11 millimeter scolex, peripherally enhancing, and cortically based lesion with adjacent vasogenic edema in the left middle frontal gyrus most consistent with a vesicular form of neurocysticercosis. MRI of the brain in radiological view (i.e., left is right and right is left).

consulted to evaluate the patient's mood and attention concerns. Despite resolution of lesion on neuroimaging, the patient reported significant suicidal ideation over the months following his treatment due to residual deficits in attention. After NCC treatment, particularly following steroid use, he noted worsening concentration (e.g., inability to focus for longer than 10–15 min each day) and memory problems (e.g., unaware of his mother's birthday, forgetting to take daily medications, and distrust of his recollection of events). He also described hopelessness about his condition and his treatment team's inability to fix his inattention/concentration. Additional depressive symptoms included loss of pleasure in daily activities, sadness, anergia, amotivation, and social isolation. He reported skipping meals (sometimes, going up to 2 days without eating) and using food as a reward mechanism for productive behavior. He was previously engaged in outpatient mental health services, including medication management and psychotherapy, but noted minimal benefit as his cognition had not improved. He reported feeling angry with himself for his concentration difficulties. He informed the interviewer that if he was suspended from his university and continued to have concentration difficulties, then he would end his life. He denied psychotic symptoms, visual/auditory hallucinations, or delusional thinking. He reported that he slept approximately 6–8 hr each night. He mentioned that it would take him 20–30 min to fall asleep, but he denied problems with sleep maintenance. He described feeling well rested upon waking. He reportedly drank little to no alcohol on social occasions. He denied tobacco or illicit substance use. In addition to NCC, the patient's medical history was significant for daily headaches for approximately 1 year prior to his seizure. Since steroid treatment, he reported only two headaches to date. Family history was reportedly unremarkable for psychiatric or neurological conditions.

In terms of social history, the patient noted mild depressive symptoms since his father's death when he was a child, but he denied functional impairment due to these symptoms in childhood or adolescence. He described limited social interactions with others aside from his family and a few close friends. He

noted a preference for a smaller social circle, and as a result, had few friends at his current university. In terms of academic functioning, the patient was reportedly “bored,” daydreamed, and “did not find the work challenging” throughout school. Per the medical record, the patient was extremely bright, at the “top of his class,” and was selected by his homeland’s program to attend advanced education at the collegiate level.

Behavioral Observations

Note that since this evaluation was conducted during the coronavirus disease-2019 pandemic, the examiner wore an N95 mask and eye goggles. The patient did not wear any personal protective equipment. He was alert and aware of the purpose of the evaluation and agreed to participate. Ambulation and motor movements were unremarkable. He wore glasses to aid with vision. Hearing was adequate for the purpose of the evaluation. Eye contact was normal, though he often looked down while responding during the interview. He spoke softly and slowly, with frequent pauses before responding as if dissociating. Occasionally, the latent responses were preceded by a blank stare. Fluency, repetition, and comprehension were normal. Thought processes were linear and goal-oriented and content was normal. Mood was depressed with congruent affect. He was cooperative and participated fully in all aspects of the evaluation. Notably, he was engaged for 5 hr of testing and did not show signs of inattention or distractibility. He was polite and patient and stated that testing was “fun” and that he would keep working all day if needed.

RESULTS

Summary

Results of the neuropsychological evaluation can be found in Table 1. In the context of an at least average range estimated premorbid cognitive functioning, the patient’s performance was variable (ranging from moderately impaired to high average) on measures of attention and executive functions. He displayed deficits on measures of mental flexibility and verbal inhibition. Visuospatial abilities were within expectations. Language was generally intact, though confounded by cultural and linguistic variables. Verbal learning was in the borderline range. Verbal immediate recall ranged from mildly impaired to borderline, and delayed recall was in the moderately impaired range. By contrast, visual learning was in the average range with high average range delayed recall. Memory storage was intact (both verbal and visual). Overall, the patient appeared to show deficits in verbal encoding and auditory working memory. By contrast, performance in visual encoding and visual working memory tests were within expectations. Although the patient reported comfortability with English and attended an English medium school, it is possible that the deficits observed in verbal encoding and auditory working memory may be due to pre-existing difficulties unrelated to the NCC lesion. However, this is difficult to disentangle without prior baseline assessment. He endorsed moderate depressive symptoms and mild anxiety symptoms on self-report questionnaires. Symptoms of inattention and hyperactivity in childhood and adulthood were clinically significant on a self-report measure.

Clinical Impressions

The patient’s amotivation, anergia, concentration difficulties, suicidal ideation, and reduced processing speed on formal testing appeared most consistent with a diagnosis of major depressive disorder (moderate). Although certain subtests showed deficits in set-shifting and mental flexibility, and report of worsening attention difficulties raised concern for attention deficit-hyperactivity disorder-inattentive type (ADHD), the patient’s depressive symptoms appeared to better account for these residual attention difficulties. Though the examiners were unable to definitively rule out a neurodevelopmental disorder, like ADHD, behavioral observations were notable for his strong concentration abilities in the context of a structured testing environment, which argued against this possibility. His inattention reported in childhood appeared to stem from not feeling challenged in school, causing him to daydream and seek external stimulation. Further, the patient’s dysexecutive tendencies (e.g., difficulties with switching and sequencing, category switching, and inhibition) likely had downstream effects on his memory and concentration abilities. The most likely etiological cause of these symptoms is the NCC diagnosis, given that his depressed mood and attention difficulties began after the diagnosis and, despite resolution on neuroimaging, he continued to show residual deficits. Further, the location of the dlPFC lesion and empirical support from the literature are consistent with his subjective cognitive and psychiatric complaints.

The patient reported life-long social isolation and nuances in social comportment (e.g., inconsistent eye contact and unintentionally offending others), which became more noticeable upon his immigration to the United States within the last year. It was unclear whether part of this may be due to cultural differences. For example, Singelis & Brown (1995) describe how many African cultures value high-context communication (i.e., emphasis on nonverbal interactions), whereas individualistic cultures like the United States value low-context communication (i.e., emphasis on verbal interactions). These cultural differences may make socialization in an individualistic culture more difficult for the patient and further exacerbate depressive symptoms. As such, careful consideration and sensitivity suggesting optional social skills building was recommended to the patient to help with adjustment to a new culture, while simultaneously emphasizing that this social difference was not pathological in nature. We believed that as the patient continued to recover and manage his depressive symptoms, his cognition and concentration abilities would likely improve. Recommendations, including compensatory strategies to optimize his cognitive and emotional well-being, were also outlined.

DISCUSSION

This is the first case study, to our knowledge, to show an isolated NCC lesion in the left middle frontal gyrus or dlPFC. Consistent with what would be expected with a lateralized lesion in the left dlPFC, our patient demonstrated lower verbal working memory performance (Wechsler Adult Intelligence Scale-Fourth Edition Digit Span) compared to visual working memory (WMS-IV Spatial Span). He also showed difficulties in verbal memory

Table 1. Neuropsychological assessment results

Test by domain	Raw	Z	Percentile	Process analysis and errors
Validity				
Reliable Digit Span	7			Valid
CVLT-3 Forced Choice	16			Valid
Premorbid				
TOPF	47	0.6	73	Average
Attention, speed, and executive				
Digit Span Total	22	-1.0	16	Borderline
Digit Span Forward	9	-0.7	25	Low average; longest span: seven
Digit Span Backward	7	-0.7	25	Low average; longest span: four
Digit Span Sequencing	6	-1.3	9	Borderline; longest span: four
WMS-IV Spatial Span Total	16	-0.3	37	Average
SDMT-Written	44	-1.5	7	Mildly impaired; three errors
DKEFS Trails-Visual Scanning (Time)	20	0.0	50	Average; zero error
DKEFS Trails-Visual Scanning (Error)	0		100	
DKEFS Trails-Number Seq. (Time)	34	-0.3	37	Average; zero error
DKEFS Trails-Number Seq. (Error)	0		100	
DKEFS Trails-Letter Seq. (Time)	32	-0.3	37	Average; zero error
DKEFS Trails-Letter Seq. (Error)	0		100	
DKEFS Trails-Switching (Time)	101	-1.0	16	Borderline; two sequencing errors
DKEFS Trails-Switching (Error)	2		14	
DKEFS Trails-Motor Speed (Time)	25	0.3	63	Average; zero error
DKEFS Trails-Motor Speed (Error)	0		100	
DKEFS Color Naming (Time)	41	-2.0	2	Moderately impaired; two self-corrections
DKEFS Word Reading (Time)	29	-1.3	9	Borderline; zero error
DKEFS Inhibition (Time)	64	-1.0	16	Borderline; two self-corrections
DKEFS Inhibition (Cor. Errors)	2		15	
DKEFS Inhibition/Switching (Time)	90	-2.7	<1	Moderately impaired; two uncorrected errors, six self-corrections
DKEFS Inhibition/Switching (Uncor. Errors)	2		25	
DKEFS Inhibition/Switching (Cor. Errors)	6		1	
DKEFS Letter Fluency	40	0.3	63	Average; one repetition
DKEFS Category Switching Total	7	-2.7	<1	Moderately impaired; zero error (slow pace)
DKEFS Category Switching Acc.	6	-2.0	2	Moderately impaired; zero error
DKEFS Verbal Fluency Rep. Errors	1	0.7	76	High average
DKEFS Tower Total Achievement	21	1.0	84	High average
Score				
DKEFS Tower Mean First-Move Time	2.5	0.7	76	High average
DKEFS Time-Per-Move Ration	2.3	0.3	62	Average
DKEFS Move Accuracy Ratio	1.5	0.3	62	Average
DKEFS Total Rule Violations	0		100	
Rey-O Figure Copy	35		>16	
Wisconsin Card Sort				
Categories Completed	6		>16	
Trials to Complete 1st Category	11		>16	
Failure to Maintain Set	1		>16	
Learning to Learn	0.15		>16	
Language				
DKEFS Category Fluency	31	-1.0	16	Borderline; zero error
MiNT	17	-6.5	<1	Severely impaired; six correct with phonemic cues; several unfamiliar items
Visuospatial				
Beery VMI	30	0.5	68	Average
WAIS-IV Matrix Reasoning	20	0.3	63	Average
Memory				
CVLT-3				T1-T5 raw: 5, 8, 6, 10, 9 T1-T5 scaled: 8, 9, 4, 8, 6
T1-T5 Raw Sum (Sum of Scaled)	38 (35)	-1.2	12	Borderline; two semantically-related intrusions, three repetitions

Table 1. Continued.

Test by domain	Raw	Z	Percentile	Process analysis and errors
List B	8	1.0	84	High average; two intrusions (one from first list), two repetitions
SDFR	8	-1.0	16	Borderline; one semantically-related intrusion
SDCR	6	-1.7	5	Mildly impaired; three semantically-related intrusions
LDFR	3	-2.3	<1	Moderately impaired; two semantically-related intrusions
LDCR	5	-2.0	2	Moderately impaired; five semantically related intrusions
Recognition Hits	15	-0.3	37	Average
Recognition FP	8	-2.0	2	Moderately impaired
BVMT-R				
Total Recall (T1, T2, T3)	30(7,11,12)	0.3	62	Average
Delayed Recall	12	0.9	82	High average
Retention (%)	100		>16	WNL
Recognition (TP, FP)	6 (6, 0)		>16	WNL
Emotional/behavioral				
BDI-II	21			Moderate depression
AMAS-A Total Anxiety	18	1.4	92	Mild
Worry/Oversensitivity	11	1.4	92	Mild
Physiological Anxiety	4	1.1	86	Mild
Social Concerns/Stress	3	0.6	73	Mild
L Scale	2	-0.2	42	Valid
Barkley ADHD Current				
Inattention	6			Meets criteria
Hyperactivity	5			Meets criteria
Barkley ADHD Childhood				
Inattention	7			Meets criteria
Hyperactivity	4			Below cutoff

Notes: TOPF = Test of Premorbid Functioning; WAIS-IV = Wechsler Adult Intelligence Scale-Fourth Edition; SDMT-W = Symbol Digit Modality Test-Written Version; DKEFS = Delis-Kaplan Executive Function System: Trails, Color-Word, Verbal Fluency, Tower; Rey-O Figure Copy = Rey-Osterrieth Complex Figure Copy; WCST = Wisconsin Card Sort; MiNT = Multilingual Naming Test; Beery VMI = Beery Visual-Motor Integration; WMS-III = Wechsler Memory Scale-Third Edition; CVLT-3 = California Verbal Learning Test; BVMT-R = Brief Visuospatial Memory Test-Revised; BDI-II = Beck Depression Inventory-Second Edition; AMAS-A = Adult Manifest Anxiety Scale-Adult Version; ADHD, attention deficit-hyperactivity disorder-inattentive type; Barkley's ADHD Scales (Childhood/Current).

encoding and retrieval compared to visual memory. Research has shown that individuals with lesions in the dlPFC have difficulties with working memory, rule-learning, planning, attention, and motivation (Szczepanski & Knight, 2014), which is consistent with the patient's history and results of formal neuropsychological testing. Importantly, the patient reported worsening depression symptoms, to the point of suicidal ideation, following steroid treatment for NCC. Although we cannot completely rule out the effect of steroid use on the patient's mood and cognitive symptoms, the patient's use of the medication was short term (i.e., 1 week), and studies have found that the most common side effects include abdominal discomfort, skin rash, swelling, and hot flash (Min et al., 2012). Moreover, studies have shown that psychiatric or cognitive symptoms resulting from steroid use generally resolve with dosage reduction or discontinuation (Brown et al., 1999; Kenna et al., 2011). It is also important to note that while the patient's depressive and inattentive symptoms may be complicated by his transition to the United States approximately 1 year prior to the evaluation, the level and duration of functional impairment appear greater than adjustment-related stressors alone. In terms of ongoing antiseizure medication use via levetiracetam, research has shown minimal side effects, with some studies suggesting improvement in

cognitive performance (Frackowiak et al., 2019). Taken together, the manifestation of both cognitive and psychiatric symptoms appear to be better explained by residual effects of the NCC diagnosis.

Consistent with previous research, a lesion in the dlPFC confers vulnerability for depression and may have increased the severity of depressive symptoms reported by our patient (Forlenza et al., 1998; Koenigs et al., 2008). The literature on transcranial magnetic stimulation has shown that the left dlPFC appears to be a target area for anti-depression treatment, as stimulation induces striatal dopamine release (Avissar et al., 2017). Although the patient reported increased depressive symptoms because of his attention difficulties, it is possible that the worsening depression was a direct result from the NCC lesion, leading to downstream effects on cognition and vice versa. Indeed, studies have shown significant deficits in executive function, memory, and attention in patients with depression (Lee et al., 2012; Rock et al., 2014). Thus, residual effects of the NCC lesion in the left dlPFC, particularly in the context of worsening depression and attention difficulties, may be affecting cognition and cannot be ruled out.

To this end, the literature suggests that clinical recovery from NCC, accompanied by improvement shown on neuroimaging,

takes approximately 2–6 months (Li et al., 2016; Ramirez-Bermudez et al., 2005). As such, it would be expected that our patient would have experienced a full clinical recovery, with no residual cognitive/psychiatric symptoms, particularly given normal neuroimaging findings 8 months post-treatment (i.e., no discernable lesion or inflammation). However, subjective cognitive and psychiatric symptoms remained. There are a few reasons this may be the case. First, clinical recovery has a myriad of definitions in the literature. For example, clinical recovery may encompass partial or complete structural resolution (i.e., reduced inflammation and undetectable lesions), which may or may not include resolved cognitive deficits. Second, premorbid psychiatric or cognitive concerns may worsen, depending on the location and number of lesions (Forlenza et al., 1998; Koenigs et al., 2008). Third, NCC is a complex diagnosis with a spectrum of neurological and radiographical features (Ramirez-Zamora & Alarcon, 2010). As such, the variable course and treatment of the condition is specific to the individual.

This case study is not without limitations. First, the patient's cultural and linguistic background differs from the sociocultural context in which the standardized measures administered in this evaluation were developed. As mentioned earlier, verbal versus nonverbal measures were carefully selected, while also considering the differences in socio-cultural background. With this being said, the examiners interpreted these data with caution using the most appropriate normative data available in tandem with the patient's concerns and cultural context. Second, it is important to note that baseline neuropsychological assessment was not available. Given his high-achieving academic background (e.g., being at the top of his class and scholarships to attend top-tier collegiate education), the test results do appear to be a significant decline from premorbid functioning. However, it is difficult to definitely disentangle long-standing cognitive or psychiatric difficulties from the effects of the lesion. As such, repeat neuropsychological testing was recommended. Third, the generalizability of the findings are complicated by the vast heterogeneity of NCC. Future neuropsychological studies may compare cognitive profiles based on the presence of inflammation, number, location, growth, and stage of degeneration of cysts (Garcia et al., 2005; Verma et al., 2010).

CONCLUSION

The neuropsychological findings of this case study were consistent with what would be expected of a lesion to the left dlPFC. Specifically, the patient showed difficulties with basic attention, working memory, and significant depressive symptoms. Despite normal neuroimaging and reduced inflammation approximately 8 months post-treatment, residual cognitive and psychiatric difficulties persisted. The heterogeneity of NCC should continue to be studied to further improve the diagnostic clarity and to better understand the risks associated with cognitive and psychiatric protracted recovery.

CONFLICT OF INTEREST

None declared.

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