

“But man is not made for defeat”: insights into Ernest Hemingway’s dementia

“Mas o homem não foi feito para a derrota”: visões sobre a demência de Ernest Hemingway

Léo COUTINHO^{1,2}, Hélio Afonso Ghizoni TEIVE^{1,2}

ABSTRACT

Ernest Hemingway is widely regarded as one of the greatest fiction writers of all time. During his life, he demonstrated several signs of psychological suffering with gradual worsening and presentation of cognitive issues over his late years. Some of his symptoms and the course of his disease suggest that he might have suffered from an organic neurodegenerative condition that contributed to his decline, which culminated in his suicide in 1961. In this historical note, we discuss diagnostic hypotheses compatible with Hemingway’s illness, in light of biographical reports.

Keywords: History of Medicine; Neurology; Art; Dementia; Neurodegenerative Diseases.

RESUMO

Ernest Hemingway é considerado um dos escritores mais lidos de todos os tempos. Durante sua vida, ele demonstrou diversos sinais de sofrimento psicológico com piora gradual durante seus últimos anos, associado à apresentação de distúrbios cognitivos. Alguns de seus sintomas, assim como o curso da doença, sugerem que ele talvez tenha padecido de uma condição neurodegenerativa orgânica que contribuiu para o seu declínio, culminando em seu suicídio em 1961. Nesta nota histórica, discutimos hipóteses diagnósticas compatíveis com a doença de Hemingway, à luz de relatos biográficos.

Palavras-chave: História da Medicina; Neurologia; Arte; Demência; Doenças Neurodegenerativas.

INTRODUCTION

Ernest Hemingway (1899–1961) (Figure 1) is one of the world’s most praised literary writers. His objective prose created masterpieces such as *For whom the bell tolls* and *The old man and the sea*¹⁻⁴.

Born in 1899 in Oak Park, Illinois, Hemingway began writing in journalism and war correspondence. He married four times and had three children.

Hemingway was given the ultimate accolades in literature: the Pulitzer in 1953 and the Nobel Prize in 1954^{5,6}. Despite an achieved life, Hemingway presented signs of psychiatric suffering⁷⁻¹², which culminated in his suicide in 1961 in Ketchum, Idaho. Recent evidence suggests that in his late years he presented neurological signs attributable to dementia³.



At the 60th anniversary of Hemingway’s death, we discuss his neurological condition, emphasizing organic hypotheses based on his biographic reports.

COGNITIVE AND BEHAVIORAL DECLINE

Hemingway presented early signs of a psychiatric condition, possibly bipolar disorder, with documented maniac and depressive episodes, in addition to a significant family history of suicide (Figure 2), although only his father had committed suicide before him^{1-3,7}. Many patients with this condition share outstanding creativity^{7,8}. Patients with bipolar disorder have an increased dementia risk with an incidence of 25.2% in a recent cohort⁹. Previous papers detailed his psychological ailment¹⁰⁻¹³.

¹Universidade Federal do Paraná, Serviço de Neurologia, Unidade de Distúrbios de Movimento, Curitiba PR, Brazil.

²Universidade Federal do Paraná, Programa de Pós-Graduação em Medicina Interna e Ciências da Saúde, Grupo de Doenças Neurológicas, Curitiba PR, Brazil.

LC  <https://orcid.org/0000-0003-4921-2939>; HAGT  <https://orcid.org/0000-0003-2305-1073>

Correspondence: Léo Coutinho; Email: leocoutinho23@hotmail.com.

Conflict of interest: There is no conflict of interest to declare.

Authors’ contributions: LC: organization, execution, writing of the first draft, writing of the final manuscript; HAGT: conception, review and critique, writing of the final manuscript.

Received on July 26, 2021; Received in its final form on September 27, 2021; Accepted on October 04, 2021.



Source: Ernest Hemingway Collection. John F. Kennedy Presidential Library and Museum, Boston. Licensed under a Public Domain Mark.

Figure 1. Ernest Hemingway in the cabin of his boat, *Pilar* (circa 1950s).

was marked by a primacy of behavioral symptoms with late cognitive issues, raising several hypotheses (Table 1)¹⁻³.

Hemingway experienced progressive disinhibition. He would often say inappropriate things during social gatherings and engage in more sexually liberated experiences. This disinhibition can be perceived in the sexual themes present in his last works, *A moveable feast* and *Garden of Eden*¹⁻³. The former cruelly depicts his first two wives and his friendship with Scott Fitzgerald, possibly motivated by disinhibition.

His paranoia and delusions increased, with a belief that he was under FBI surveillance. Other sources of paranoia were his hypochondria, fear of impoverishment, and the possibility of arrest for illegal hunting and for “taking liberties with a minor”¹⁻³.

Hemingway would present frequent and unpredictable bursts of aggressiveness, particularly towards his last wife, Mary, who endured significant abuse¹⁻³.

After his first admittance to the Mayo Clinic (1960), Hemingway presented amnestic symptomatology. This would be a burden to his writing, and he would consider finishing *A moveable feast* impossible. He needed help with the manuscript revision from his wife and editor and his last works would be published only posthumously¹⁻³.

The following evidence supports the presence of dementia:

1. Hemingway’s decline was inexorable, despite electroconvulsive therapy carried in the Mayo Clinic.
2. In his last years, his cognition sharply declined, impairing his writing.
3. He presented risk factors for dementia, namely alcoholism, sexual risk behavior, and repeated head trauma³.

The precise onset of Hemingway’s decline is unclear, but it possibly initiated during his fifth decade of life¹⁻³. The disease

DISCUSSING THE POSSIBILITIES

Frontotemporal dementia (FTD)

Hemingway’s clinical features are compatible with the behavioral variant of frontotemporal dementia (bvFTD). FTD presents an earlier onset than other neurodegenerative etiologies, as seen in the writer’s case. Genetics plays a significant role in FTD, and up to 40% of patients present a family history of dementia. However, other bvFTD features, such as lack of empathy, obsessive behavior, and a dysexecutive syndrome were not present¹⁴.

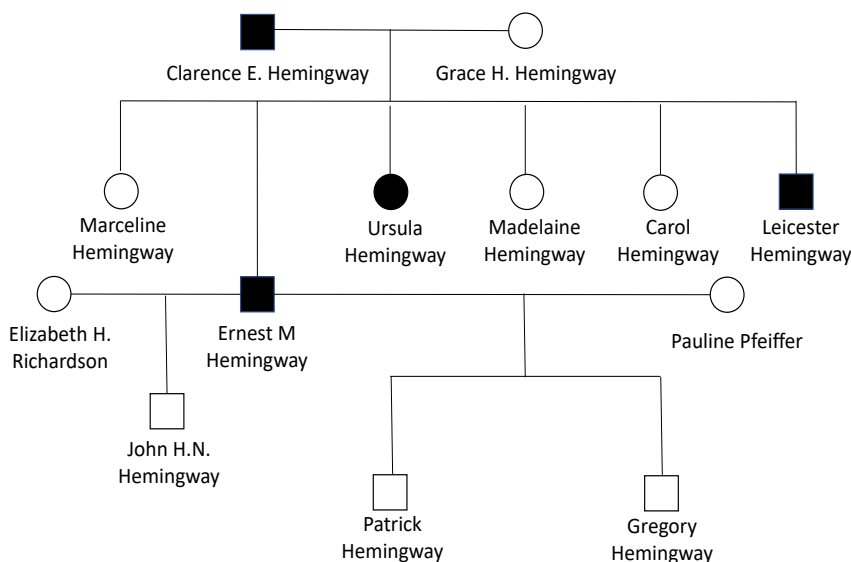


Figure 2. Pedigree chart for the Hemingway family, showing first-degree relatives to Ernest^{3,7}. Icons in black represent family members who committed suicide. Marriages with Martha Gellhorn and Mary Welsh were omitted, as they did not have children.

Table 1. Diagnostic possibilities compatible with Ernest Hemingway's condition and epidemiology.

Condition	Arguments in favor	Arguments against
Behavioral variant Frontotemporal Dementia (bvFTD)	Disinhibition. Early onset. Late cognitive disability.	Absence of other features (lack of empathy, obsessive behavior, problems in executive function).
Lewy Body Dementia (LBD)	Psychosis. Delusions. Fluctuations.	Absence of Parkinsonism. Lack of well systematized hallucinations.
Vascular dementia	Multiple risk factors. Family history of vascular complications.	No history of strokes.
Chronic traumatic encephalopathy (CTE)	Multiple concussions. He was a notorious brawler. Predominantly behavioral presentation.	None.
Alcohol toxicity/Vitamin deficiency	Heavy alcohol consumption. Other documented complications of alcohol intake (hepatopathy, withdrawal syndrome).	Lack of early amnesic symptomatology.
Neurosyphilis	Risk sexual behavior.	Lack of other neurosyphilis hallmarks (motor symptoms, cranial nerve palsy). Lack of a well-documented primary treponemal infection.
Huntington's disease (HD)	Phenotypes with pure behavioral/cognitive symptomatology.	Rare presentation. No members of the Hemingway family presented motor phenomenology compatible with HD.

Lewy body dementia (LBD)

Hallucinations and psychosis with marked fluctuations are the hallmarks of LBD¹⁵. This diagnosis was recently proposed as etiology for Hemingway's decline³. However, parkinsonism was never described in his case. Moreover, Hemingway's psychosis presented more delusions than well-substantiated hallucinations, making LBD an unlikely diagnosis¹⁻⁵.

Vascular dementia

In the Mayo Clinic, Hemingway was diagnosed with severe hypertension, prediabetes, and dyslipidemia. He was under suspicion of hemochromatosis, but a liver biopsy was contraindicated considering his precarious health¹⁻⁵. Hemingway had a family history of vasculopathy, particularly related to diabetes¹⁻⁵.

Although Hemingway's biography reports no strokes, these comorbidities are risk factors for small vessel disease and subcortical ischemic vascular dementia. As clinical presentation is variable and overlapping with other dementia etiologies is common, this is a consistent hypothesis¹⁶.

Chronic traumatic encephalopathy (CTE)

Hemingway endured nine major head traumas during his war service, including a mortar blast. In 1954, he survived two plane crashes. He practiced football and boxing from an early age, acted as an amateur bullfighter, and was a reckless driver¹⁻⁵. CTE is a plausible hypothesis, as this condition often presents with behavioral symptomatology, particularly aggressiveness and mood changes, while cognition is of late affection¹⁷.

Alcohol toxicity/Vitamin deficiency

To say Hemingway was a heavy drinker would be an understatement. He spent a significant part of his time in Havana at the bar *La Floridita*, being served with *Papa Dobles* (the Hemingway daiquiri) by the bartender^{2,3}. The role of alcohol consumption in dementia is documented, being a risk factor for vascular dementia and Alzheimer's disease. The proposed mechanisms include direct neuronal toxicity and secondary vitamin deficiencies¹⁸.

Neurosyphilis

Hemingway had multiple sexual partners, including extramarital relationships. Although he was at risk for syphilis, a more diverse clinical picture would be expected. The absence of motor symptomatology, cranial nerve palsy, and other hallmarks of neurosyphilis, besides the absence of a well-documented primary treponemal infection, make this diagnosis improbable¹⁹.

Huntington's disease (HD)

Certain phenotypes of HD have a predominance or exclusivity of non-motor symptomatology, presenting behavioral and cognitive symptoms, such as the exhibited in Hemingway's case and family history; motor phenomena may have a late onset or never occur. However, this is a rare presentation and an unlikely hypothesis. Phenotypic variability occurs within the same family, and other members of the Hemingway family would present motor symptomatology²⁰.

In conclusion, Hemingway's case would remain a challenge in modern days. His personality traits would pose an obstacle for the detection of behavioral symptoms of neurodegeneration.

Although a psychiatric condition is acknowledged, Hemingway's symptomatology is compatible with organic dementia. In the author's opinions and in accordance with

recent literature³, bvFTD and CTE, possibly associated with a vascular component, might have contributed to his decline.

Remarkably, Hemingway tried to write to his very end despite his cognitive impairment; a display of tenacity worthy of Santiago, the main protagonist of *The old man and the sea*.

References

38. Wagner-Martin LW. A historical guide to Ernest Hemingway. 1st ed. New York (NY): Oxford University Press; 2000. 256 p.
39. Hotchner AE. Papa Hemingway: a personal memoir. 1st ed. New York (NY): Random House; 1966. 299 p.
40. Farah A. Hemingway's brain. 1st ed. Columbia (SC): University of South Carolina Press; 2017. 216 p.
41. Hemingway E. The old man and the sea: the Hemingway library edition. New York (NY): Scribner; 2020. 155 p.
42. The Nobel Prize [Internet]. Ernest Hemingway – Biographical. NobelPrize.org. [cited 2021 Jul 7] Available from: <https://www.nobelprize.org/prizes/literature/1954/hemingway/facts/>
43. The Nobel Prize [Internet]. Ernest Hemingway – Facts. NobelPrize.org. [cited 2021 Jul 7]. Available from: <https://www.nobelprize.org/prizes/literature/1954/hemingway/facts/>
44. Jamison KR. Touched with fire: manic-depressive illness and the artistic temperament. 1st ed. New York (NY): The Free Press; 1996. 384 p.
45. Goodwin FK, Jamison KR. Manic-Depressive illness: bipolar disorders and recurrent depression. 2nd ed. New York (NY): Oxford University Press; 2007. 1288 p.
46. Callahan BL, McLaren-Gradinaru M, Burles F, Laria G. How does dementia begin to manifest in bipolar disorder? A description of prodromal clinical and cognitive changes. *J Alzheimers Dis*. 2021;82(2):737-48. <https://doi.org/10.3233/JAD-201240>
47. Dieguez S. 'A man can be destroyed but not defeated': Ernest Hemingway's near-death experience and declining health. In: Bogousslavsky J, Hennerici MG, Bänzner H, Bassetti C, editors. *Neurological disorders in famous artists – Part 3*; Karger. 2010. p. 174-206.
48. Martin CD. Ernest Hemingway: a psychological autopsy of a suicide. *Psychiatry*. 2006 Dec;69(4):351-61. <https://doi.org/10.1521/psyc.2006.69.4.351>
49. Trent B. Hemingway: could his suicide have been prevented? *CMAJ*. 1986 Oct 15;135(8):933-4.
50. Yalom ID, Yalom M. Ernest Hemingway. A psychiatric view. *Arch Gen Psychiatry*. 1971 Jun;24(6):485-94. <https://doi.org/10.1001/archpsyc.1971.01750120001001>
51. Olney NT, Spina S, Miller BL. Frontotemporal dementia. *Neurol Clin*. 2017 May;35(2):339-74. <https://doi.org/10.1016/j.ncl.2017.01.008>
52. Gomperts SN. Lewy body dementias: dementia with Lewy Bodies and Parkinson disease dementia. *Continuum (Minneapolis)*. 2016 Apr;22(2):435-63. <https://doi.org/10.1212/CON.0000000000000309>
53. Iadecola C, Duering M, Hachinski V, Joutel A, Pendlebury ST, Schneider JA, et al. Vascular cognitive impairment and dementia: JACC scientific expert panel. *J Am Coll Cardiol*. 2019 Jul 2;73(25):3326-44. <https://doi.org/10.1016/j.jacc.2019.04.034>
54. Albayram O, Albayram S, Mannix R. Chronic traumatic encephalopathy – a blueprint for the bridge between neurological and psychiatric disorders. *Transl Psychiatry*. 2020 Dec 8;10(1):424. <https://doi.org/10.1038/s41398-020-01111-x>
55. Rehm J, Hasan OSM, Black SE, Shield KD, Schwarzinger M. Alcohol use and dementia: a systematic scoping review. *Alzheimers Res Ther*. 2019 Jan 5;11(1):1. <https://doi.org/10.1186/s13195-018-0453-0>
56. Ropper AH. Neurosyphilis. *N Eng J Med*. 2019 Oct 3;381(14):1358-63. <https://doi.org/10.1056/NEJMra1906228>
57. Oosterloo M, Greef BTA, Bijlsma EK, Durr A, Tabrizi SJ, Estevez-Fraga C, et al. Disease onset in Huntington's disease: when is the conversion? *Mov Disord Clin Pract*. 2021 Jan 31;8(3):352-60. <https://doi.org/10.1002/mdc3.13148>