Novel therapies in Multiple Sclerosis treatment: Reverse engineering a prolonged remission case

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Highlights

- MS remains without known cause or cure, 157 years after being named and defined by Marie Charcot
- Patient x, using a self-directed n-of-1 strategy, underwent a series of seemingly unrelated medical treatments between the years of 2012 and 2017
- After experiencing an 8-year period of remission without disease modifying therapies, patient x developed a theory
- This theory may act as a framework to be replicated in the development of patient-specific MS treatments

Abstract

Background

Multiple Sclerosis is an autoimmune disorder affecting the central nervous system (CNS). Clinical manifestations are highly heterogeneous among individuals with MS. The variability in symptoms, as well as the unpredictable nature of relapse and remission periods, complicates efforts to determine the underlying cause and develop effective treatments.

Methods

An n-of-1 patient-directed research (PDR) study was conducted from March 2005 to July 2025 to determine the factors associated with both the onset and sustained remission of the disease in patient X. The study utilized approximately fifty years of research data provided by patient X, which were cross-referenced through an extensive online literature review. A reverse Six Sigma DMAIC methodology was employed to identify the controls responsible for inducing remission of multiple sclerosis (MS), thereby establishing the root cause of the condition.

Results

Current medical literature indicates that multiple sclerosis (MS) results from a complex interplay of genetic (**GEN**), environmental (**ENV**), infectious (**INF**), and immune system dysfunction (**ISD**) factors. This study documents 15 distinct diagnoses in patient X that align with this multifactorial hypothesis. Specifically, among the identified conditions are: **GEN** | familial MS, MTHFR C677T mutation, increased susceptibility to fungal infections, cardiomegaly; **ENV** | exposure to mold (Gliotoxin); **INF** | Malassezia furfur, periodontal

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disease, trigeminal and vagus nerve damage, Helicobacter pylori infection, envenomation by venomous spider, Chlamydia pneumoniae; **ISD** | Tinea Versicolor, blood-brain barrier (BBB) dysfunction, and autonomic imbalance.

Limitations

The limited sample size inherent in n-of-1 clinical trials restricts the ability to make statistical generalizations to larger populations. Nevertheless, given the rarity of patients who have sustained remission without disease-modifying therapies, an emphasis on personalized medicine may offer increased effectiveness in such cases.

Conclusions

The study found that the interaction between the MTHFR C677T gene mutation and the subsequent methylation imbalance served as a shared factor in 13 out of 15 (87%) conditions experienced by patient X. Medical and surgical interventions addressing the environmental and infectious contributors facilitated restoration of autonomic equilibrium, resolving the chronic "fight or flight" response in the immune system. The observed improvement in specific MS symptoms following each intervention suggests the need for further research into the systemic effects of nerve damage, associated stressors, and the often-overlooked neurological impact of mycotoxins such as gliotoxin. These findings may contribute to the development of profiles that support disease prevention strategies and inform new therapeutic approaches for individuals diagnosed with multiple sclerosis.

Keywords

MS; Multiple Sclerosis; MTHFR C677T; Methylenetetrahydrofolate reductase C677T polymorphism; single nucleotide polymorphism (SNP) rs1801133; dysregulated methylation

1. INTRODUCTION

Multiple Sclerosis (MS) impacts an estimated 2.8 million people worldwide as of 2020. (King et al., 2010) While the disease was discovered 157 years ago, by Jean Martin Charcot, little progress has been made in understanding the pathogenesis. (Zalc, 2018) Emerging research indicates that multiple sclerosis (MS) arises from a combination of risk factors, such as genetic predisposition, environmental influences, infectious agents, and immune system dysfunction, which may be manifested following exposure to specific triggers. (Ghasemi et al., 2016) Data shows that individuals with MS are twice as likely to present with periodontal disease (Tsimpiris et al., 2023); furthermore, 65% of periodontitis patients tested positive for H. pylori in dental plaque (Asqah et al., 2009) and at least one study has demonstrated that 86.4% of MS patients exhibited H. pylori infection (Gavalas et al., 2015). A frequently overlooked aspect is the presence of gliotoxin—identified in 91% of MS cases—which is associated with Aspergillus (mold) metabolism(Purzycki and Shain 2010). Another important consideration is the prevailing misconception that MS manifests in only one form. This study seeks to propose a hypothesis regarding an individual with MS, periodontal disease, gliotoxin, and H. pylori infection, who experienced a transition from recurrent relapses every 2–3 months to no relapses over an eight-year period following surgical interventions.

2. METHODS

This study employs a reversed Six-Sigma DMAIC framework to clarify problem definition. (Ilin et al., 2023)

2.1. Study design

This is a novel use of the tool for clinical research, as extended remission (**Control**) from MS was the unexpected result of seemingly unrelated medical procedures (**Improve**) applied to a yet-to-be identified root cause (**Analyze**). Patient-focused and modeled as a self-audit, this n-of-1 study aims to establish with 95% confidence the absence of clinically significant misstatements. The main goal is to identify factors behind patient X's eight-year remission after treatment without the assistance disease modifying therapies (**Measure**). Data are analyzed to pinpoint key variables and root causes, which once defined (**Define**), may inform discussions on replicating such outcomes for similar patients.

2.2. Study population and parameters

This study uses an n-of-1 approach, wherein a single participant is observed throughout the experimental process. Anecdotal data pertaining to unidentified family members may be referenced solely for the purpose of informing future testing methodologies. Consent was obtained from family members for the inclusion of their anonymized data in relation to patient X. The research objective is to delineate novel genetic, environmental, infectious, and immunological factors associated with patient X's multiple sclerosis. During the "Control" phase of DMAIC, the investigation assesses how dental implant surgery, daclizumab administration, H. pylori eradication, and sleeve gastrectomy collectively contributed to symptom management and sustained an eight-year remission.

2.3. Systematic review of genetic factors in the onset of MS

Risk Factor 1. Although this category highlights genetic factors linked to patient X's MS, there is overlap with immune system dysfunction, referenced later in the study. A 2018 study found up to a 20% chance that MS patients have a family member with MS. Patient X has a family member who also has MS. (Patsopoulos, 2018)

Risk Factor 2. Patient X, and 4 family members polled, showed increased susceptibility to fungal infections, possibly linked to the inherited MTHFR C677T mutation, found in about 20–40% of people. (Biomark Med, 2019) Testing of familial histories for fungal infections could produce a valuable data point in the fight against MS.

Risk Factors 3-5. Two family members of patient X were diagnosed with scoliosis and Crohn's disease independently. (Morningstar et al., 2017)(Karban et al., 2016) Research indicates that MS, scoliosis, and Crohn's disease are loosely linked with the MTHFR C677T mutation, which can reduce enzyme production by 30-65% and contribute to autoimmune risk. (Ribeiro et al., 2024)(Moll et al., 2015) These conditions share a similar mean age of onset (around 22), with MS historically averaging 23.79 years between 1970 and 1979—suggesting that MTHFR mutations may be underreported. (Gales et al., 2018)(Romero-Pinel et al., 2022) While scoliosis and Crohn's disease often appear in the mid-to-late teens, this aligns with onset patterns for MS and MTHFR-related conditions. (Mengert et al., 2023)(Cantoro et al., 2019) Additionally, patient X was born with a heart

murmur and later diagnosed with cardiomegaly and inverted t-waves; studies associate the MTHFR C677T polymorphism with increased risk for congenital heart defects (CHD), including these specific conditions. (Wang et al., 2013)

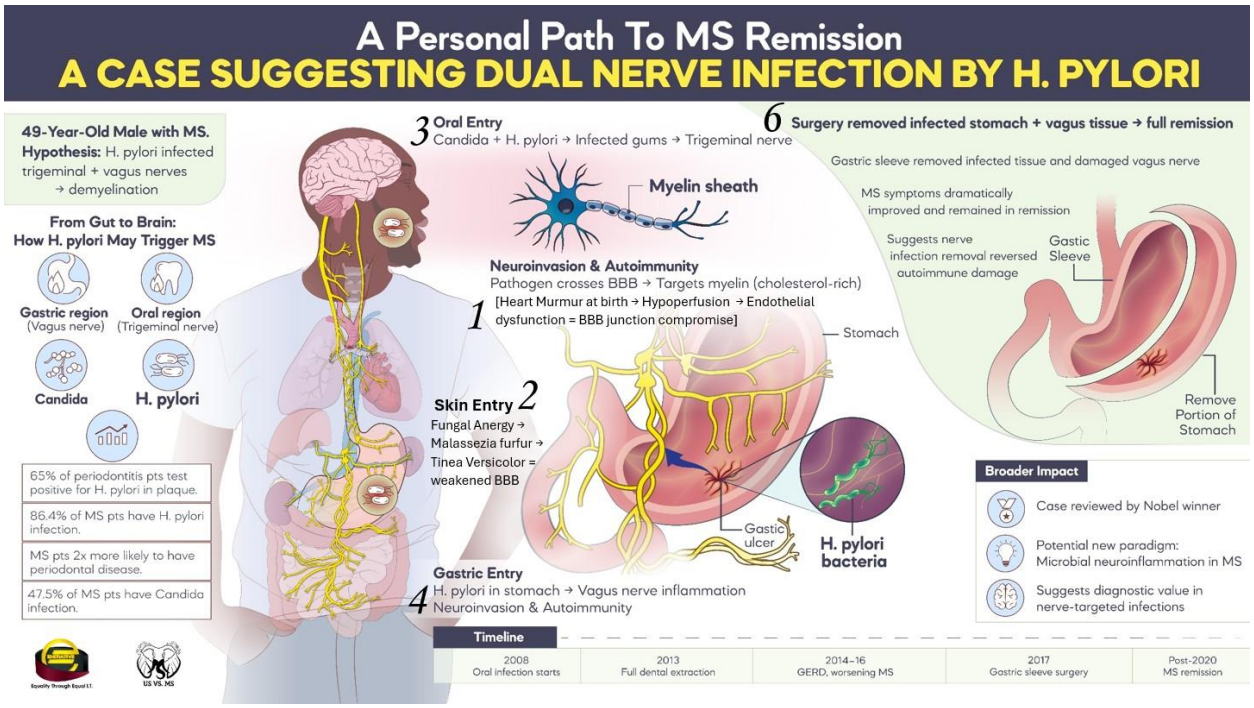


Figure 1 The figure shows the proposed pathogenesis and possible reason for extended remission in Multiple Sclerosis for patient X. Although MS has no established cause or cure, the analysis relies on peer-reviewed literature applied to patient x and other participants.

2.4. Systematic review of environmental factors in the onset of MS

Risk Factor 6. Environmental exposure is a potential risk factor for multiple sclerosis (MS), though its mechanisms remain unclear. Patient X, a military member stationed briefly in Egypt in 2001, noted mold in his barracks' bathroom. Recent research suggests this exposure may have played a role in his MS development. (Pizzorno et al., 2016)

Patient X began exhibiting symptoms of multiple sclerosis (MS) in 2005, notably experiencing leg weakness during the second mile of the Army Physical Fitness Test, four years after a documented mold exposure. Mold is known to produce mycotoxins over time, some of which are carcinogenic and have been associated with approximately half of reported illnesses (Hope, 2013). Notably, the mycotoxin gliotoxin has immunosuppressive properties and can induce DNA damage. It is important to note that manifestations of mold toxicity may arise several years following initial exposure (Brewer et al., 2013). Patient X did not receive mold or mycotoxin testing after travel to Egypt; however, additional exposures occurred in 2020 due to basement flooding and during travel to Bermuda for his father-in-law's funeral.

Although patient X did not experience MS symptoms after subsequent exposures, he reported a change in his condition one year later, in 2021. It required an additional four years to identify the underlying cause of these changes. Despite completing a comprehensive series of personal medical evaluations, he had not undergone testing for potential mold exposure due to the relatively high associated cost, which had previously discouraged him from pursuing this assessment.

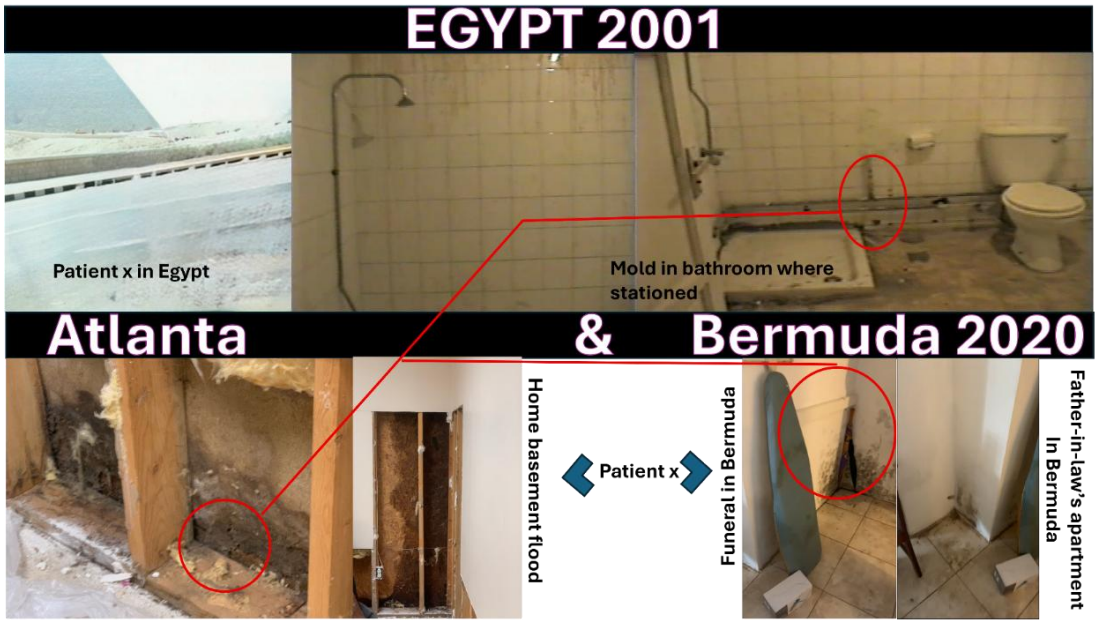


Figure 2 The graphic shows patient X's first mold exposure in Egypt (2001). Symptoms of mold toxicity appeared in 2021, about a year after two separate exposures in 2020, as shown in the lower part of the graphic.

2.5. Systematic review of infectious factors in the onset of MS

Risk Factors 7-12. This paper examines infections through the lens of molecular mimicry, which is posited to be functionally analogous to autoimmunity (Suliman, 2024). Molecular mimicry warrants further exploration; it resembles the concept of "friendly fire," whereby the immune system fails to discriminate between foreign antigens structurally like T or B cells and endogenous self-antigens. Such immunologic misrecognition may partly result from pathogen-induced dysregulation of methylation, as with Helicobacter pylori, leading to errors in S-adenosylmethionine (SAMe) synthesis, the universal methyl donor. In 1992, Patient X was diagnosed with Tinea Versicolor, attributed to Malassezia furfur, a leading fungal constituent of the human gut microbiome known for its capacity to promote molecular mimicry (Nash et al., 2017). While molecular mimicry has been linked to the pathogenesis of Multiple Sclerosis, the definitive identification of the causative infectious agent often remains elusive (Westall, 2006). Additionally, Patient X presented with both Helicobacter pylori colonization in the gastrointestinal tract and Chlamydia pneumoniae infection in the cerebrospinal fluid, identified via lumbar puncture; both pathogens are documented to exhibit molecular mimicry. A recorded envenomation by a venomous spider in 2005—the year multiple sclerosis symptoms emerged—may have also acted as a precipitating factor in the demyelination process, activating the sympathetic nervous state, colloquially known as "fight or flight." (Madill et al., 2022).

Extended sympathetic activation may have damaged the vagus and trigeminal nerves.

2.6. Systematic review of immune system dysfunction factors in the onset of MS

Risk Factors 13-15. Immune system dysfunction is characterized by either a weakened immune response or an inability to distinguish between foreign and native antigens. Patient x was diagnosed with Tinea Versicolor in 1991; although this condition results from the overgrowth of a common skin yeast, it may indicate compromised immune function. (InformedHealth.org, 2006) The preceding section provided a systematic review of infectious agents potentially implicated in the etiology of multiple sclerosis (MS), as well as a spider bite experienced by Patient X. The MTHFR C677T gene mutation—also referred to as Single Nucleotide Polymorphism (SNP) rs1801133—has been shown to cause elevated homocysteine levels, an amino acid strongly linked to chronic inflammation. (Liew et al., 2014)

Antagonism of N-methyl-D-aspartate (NMDA) receptors may occur in conjunction with high homocysteine concentrations and exposure to certain spider venoms, leading to neurotoxicity and an increased risk of cognitive decline and autonomic imbalance.(Parks et al., 1991)(Lipton et al., 1997) Another risk factor identified in patient X is blood-brain barrier (BBB) dysfunction, also called hypoperfusion, which arises due to congenital heart and vascular defects associated with the MTHFR C677T mutation. Elevated homocysteine further contributes to vascular oxidative stress, a process that can promote the development of Cerebral Small Vessel Disease (CSVD). (Papatheodorou et al., 2007) In a study involving 330 CSVD patients, 56.1% exhibited cognitive impairment, and 71.5% presented with White Matter Hyperintensities (WMH) analogous to MS-related plaques. (Wang et al., 2024) Notably, distinguishing between CSVD and MS using magnetic resonance imaging (MRI) alone is challenging, and current literature suggests that differentiation is only possible through pathology: CSVD is associated with vascular lesions, whereas MS results from demyelination. (Wang et al., 2022)

In the context of the MTHFR C677T mutation, the etiologies of CSVD and MS may be more closely related than previously understood. Vascular abnormalities are well established in cases of dysfunctional methylation (MTHFR), yet the influence of methylation on myelin stability and Oligodendrocyte Progenitor Cell (OPC) renewal—which is essential for myelin sheath formation—merits further investigation. (Webb and Guerau-de-Arellano, 2018) Furthermore, a study of 411 participants (180 with MS, 231 healthy controls) found that individuals carrying the MTHFR C677T variant had a 2.9-fold increased risk of developing MS. (Naghibalhossaini et al., 2015)

3. CONCLUSION

3.1. Reverse Engineering the MS Disability Resolution Process for Patient X

Patient X has remained free of MS symptoms following five medical interventions. The Six Sigma DMAIC process improvement framework will be applied in reverse order to develop a plausible theory regarding the cause of this prolonged remission. (Monday, 2022)

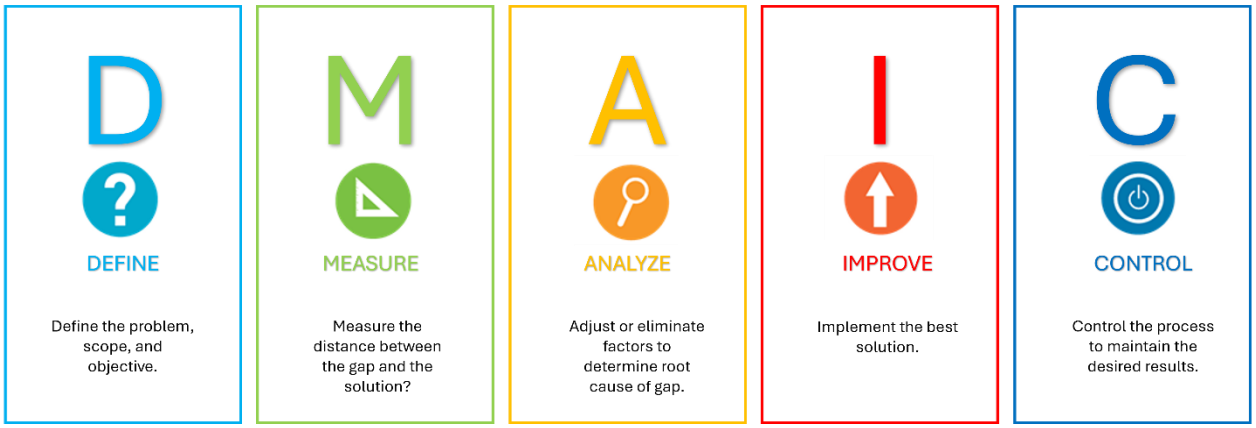


Figure 3 This illustration presents the Six Sigma DMAIC frameworks employed in process improvement initiatives. Reversing the sequence of process steps can help clarify underlying causes that were successfully managed. While the etiology and cure for MS remain unidentified, further reviews of this research journal may provide insights addressing both aspects, particularly in relation to patient x.

3.2. Control – *Develop and sustain an effective solution that reduces or prevents relapses be- yond the typical 12-to-18-month interval observed in Relapsing Remitting Multiple Sclerosis (RRMS).*

Patient X has remained asymptomatic for multiple sclerosis (MS) for eight years, exhibiting no adverse reactions to heat or other external factors that previously precipitated relapses. This sustained remission was attributed to dental implant surgery, which alleviated symptoms including cognitive dysfunction ("brain fog"), trigeminal paresthesia, back pain, and foot drag. Prior to undergoing sleeve gastrectomy, Patient X received a regimen of Omeprazole 20 mg orally twice daily, Amoxicillin 1 g twice daily, and Clarithromycin 500 mg twice daily for ten days to treat an H. pylori infection identified during endoscopic evaluation. This intervention significantly improved the patient's heat tolerance. Following the sleeve gastrectomy, the patient reported complete resolution of MS-associated symptoms.

Before these medical and surgical interventions, Patient X had been managed with Copaxone, Rebif, and subsequently Avonex until approximately 2011. Due to suboptimal disease control, the patient enrolled in a clinical trial for Daclizumab in late 2011, later approved as Zinbryta in 2016 but discontinued in February 2017 due to safety concerns arising internationally. Daclizumab provided the greatest reduction in relapse frequency and MS symptoms among prior treatments, although some symptoms persisted. Upon discontinuation of a disease-modifying therapy (DMT), a six-month washout period was observed before initiation of a new agent; Ocrevus was subsequently approved during this interval. After experiencing a six-month symptom-free period and improved heat tolerance postoperatively, patient X elected to discontinue all disease-modifying therapies.

3.3. Improve – *Identify the causes of nervous system dysfunction and devise strategies to regulate the immune response to stop self-harm.*

3.3.1. Autonomic Nervous System Imbalance

The following brain and spinal cord regions have been identified as experiencing immune stress

resulting from aggressive h. pylori infection, immunosuppression due to mycotoxins, and impaired SAMe synthesis. Under such conditions, individuals with dysregulated methylation may face challenges converting norepinephrine to epinephrine (adrenaline), which can impede the proper regulation of adrenaline within the sympathetic nervous system, potentially leading to a persistent "fight or flight" response (Funes et al., 2021). In contrast, the parasympathetic nervous system promotes a "rest and digest" state. Therapeutic interventions should aim to restore balance within the autonomic nervous system to address autonomic dysregulation. Termination of the trigeminal and vagus nerves may have functioned as a physiological tourniquet for patient X, contributing with the eradication of h. pylori to halt attacks and restore autonomic balance. Binders such as activated charcoal, bentonite clay, diatomaceous earth, and probiotics were effective in reducing or eliminating mycotoxins that suppressed immune function. Notably, patient X did not experience multiple sclerosis symptoms during the combined six weeks of pre- and postoperative liquid diet required for sleeve gastrectomy, suggesting that a liquid diet may serve as an alternative therapeutic approach to binder usage.

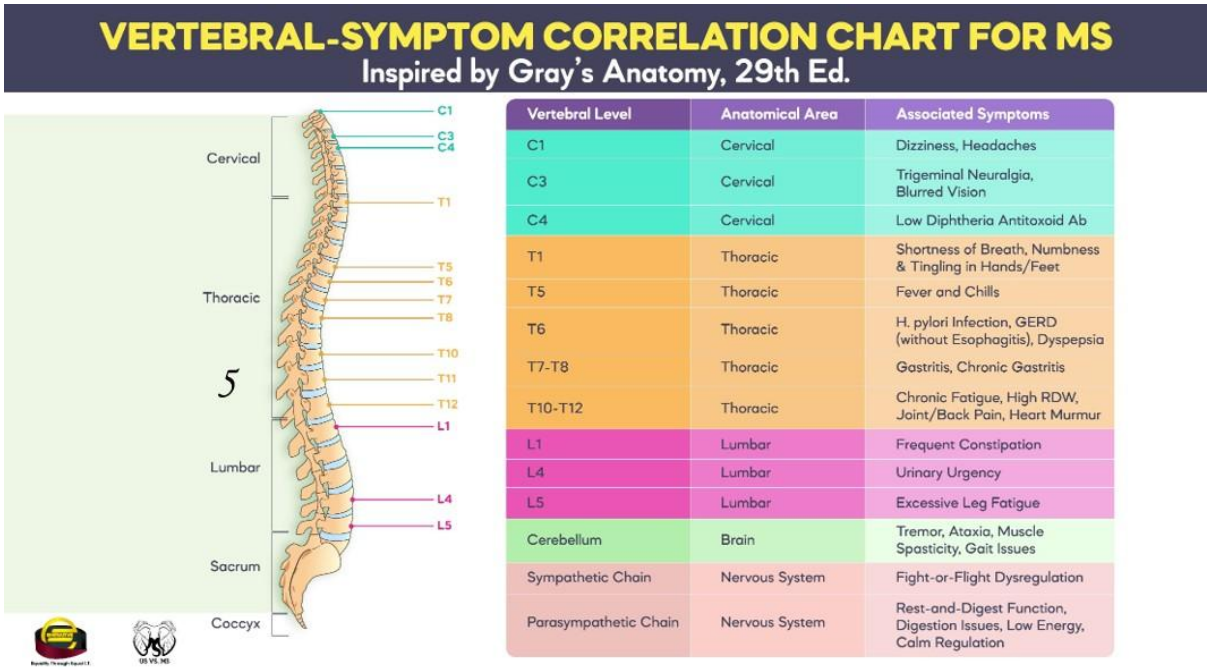


Figure 4 This illustration provides a detailed account of the multiple sclerosis symptoms observed in patient x and indicates the corresponding areas within the brain and spinal cord from which these symptoms originate, as referenced in Gray's Anatomy, 29th Edition.

3.4. Analyze – *Assess the factors present at the onset of disability in conjunction with relevant medical literature and personal or family history to establish a credible underlying cause.*

3.4.1. "The Suspect List"

In 2001, Patient x encountered mold exposure while stationed in Egypt. Mold produces mycotoxins, many of which are carcinogenic. Gliotoxin, a specific mycotoxin, has been associated with the pathogenesis of multiple sclerosis due to its cytotoxic effects on microglial cells, which play critical roles in the development, maintenance, and repair of myelin. (Matsusaka et al., 2014)

Before Binders & Probiotics

US BioTek LABORATORIES — Mycotoxin Panel

Company: [redacted] HD
Patient:
Sex: M
Date of Birth: [redacted]
Accession #:
Collected: 04/16/2025
Received: 04/21/2025
Completed: 04/25/2025

Procedure Type: Semi-Quantitative ELISA (Enzyme-Linked Immunosorbent Assay)

Code	Test - Urine	Result	Value (ppb)	Reference Range
OCHRA	Ochratoxin A	Present	9.765	<1.8 Not Present 1.8 to <2 Equivocal >=2 Present
AFLA	Aflatoxin Group: (B1, B2, G1, G2)	Present	2.084	<0.8 Not Present 0.8 to <1 Equivocal >=1 Present
TRICHO	Trichothecene Group (Macrocyclic): Roridin A, Roridin E, Roridin H, Roridin L-2, Verrucarin A, Verrucarin J, Satratoxin G, Satratoxin H, Isosatratoxin F	Present	0.172	<0.07 Not Present 0.07 to <0.09 Equivocal >=0.09 Present
GLIO	Gliotoxin Derivative	Present	4.723	<0.5 Not Present 0.5 to <1 Equivocal >=1 Present
ZEA	Zearalenone	Equivocal	0.632	<0.5 Not Present 0.5 to <0.7 Equivocal >=0.7 Present

After Binders & Probiotics

US BioTek LABORATORIES — Mycotoxin Panel

Company: [redacted] HD
Patient:
Sex: M
Date of Birth: [redacted]
Accession #:
Collected: 07/14/2025
Received: 07/15/2025
Completed: 07/17/2025

Procedure Type: Semi-Quantitative ELISA (Enzyme-Linked Immunosorbent Assay)

Code	Test - Urine	Result	Value (ppb)	Reference Range
OCHRA	Ochratoxin A	Not Present	1.527	<1.8 Not Present 1.8 to <2 Equivocal >=2 Present
AFLA	Aflatoxin Group: (B1, B2, G1, G2)	Present	1.038	<0.8 Not Present 0.8 to <1 Equivocal >=1 Present
TRICHO	Trichothecene Group (Macrocyclic): Roridin A, Roridin E, Roridin H, Roridin L-2, Verrucarin A, Verrucarin J, Satratoxin G, Satratoxin H, Isosatratoxin F	Present	0.134	<0.07 Not Present 0.07 to <0.09 Equivocal >=0.09 Present
GLIO	Gliotoxin Derivative	Equivocal	0.810	<0.5 Not Present 0.5 to <1 Equivocal >=1 Present
ZEA	Zearalenone	Present	0.715	<0.5 Not Present 0.5 to <0.7 Equivocal >=0.7 Present

Figure 5 The graphic above demonstrates that patient x exhibited elevated levels of gliotoxin for an estimated five years following mold exposure in 2020. Utilizing binders, as indicated in the subsequent graphic, patient x was able to reduce mycotoxin concentrations to normal levels within approximately three months. Individuals with typical MTHFR detoxification functionality would likely eliminate these toxins more rapidly.

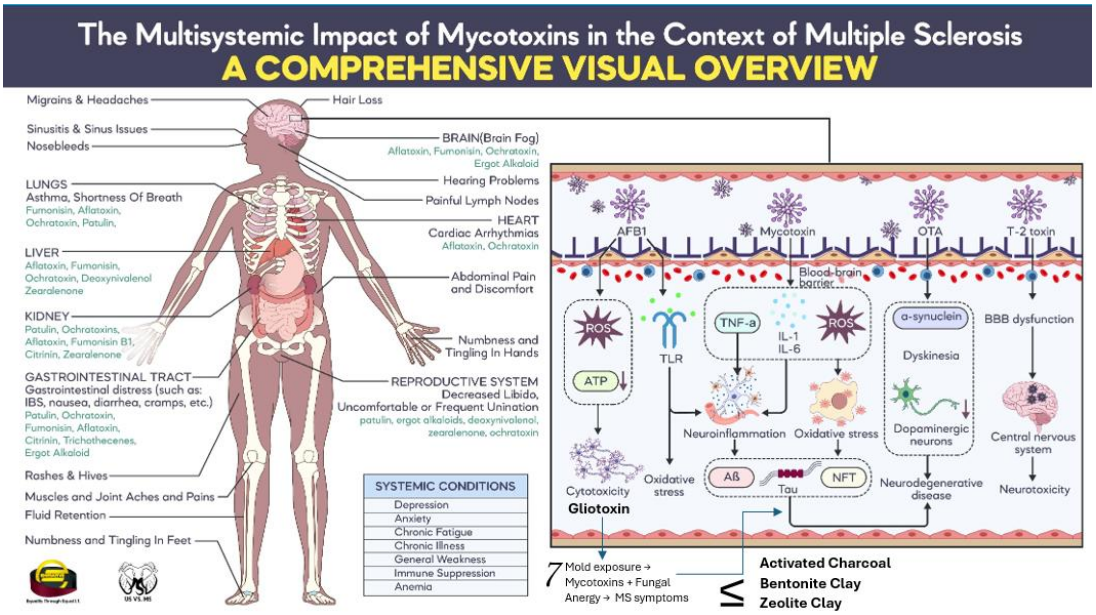
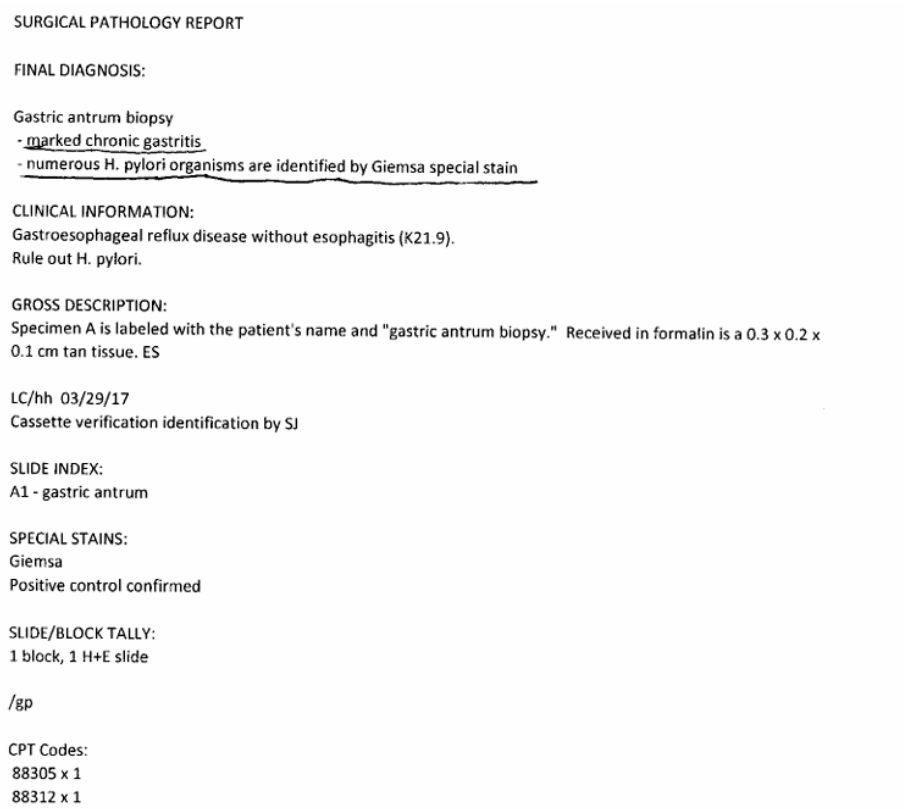


Figure 6 This diagram illustrates the systemic effects of mycotoxins on patient x and demonstrates the correlation between associated symptoms and multiple sclerosis (MS).

During preoperative evaluation for sleeve gastrectomy, Helicobacter Pylori (h. pylori) was identified upon gastric antrum biopsy. This bacterium is known to induce DNA methylation, which may contribute to the development of gastric cancer. (Kalafatakis, 2021) Patient X was diagnosed with periodontal disease in 2004. Studies indicate that individuals with multiple sclerosis are twice as likely to develop periodontal disease, and approximately 65% of patients with periodontal disease exhibit H. pylori in their dental plaques. The increased homocysteine levels from the MTHFR polymorphism are key to the inflammation experienced. H. pylori has been linked to various neurodegenerative conditions and may have contributed to damage to both the trigeminal and vagus nerves in patient X. (VanElzakker, 2013)



SURGICAL PATHOLOGY REPORT

FINAL DIAGNOSIS:

Gastric antrum biopsy
- marked chronic gastritis
- numerous H. pylori organisms are identified by Giemsa special stain

CLINICAL INFORMATION:
Gastroesophageal reflux disease without esophagitis (K21.9).
Rule out H. pylori.

GROSS DESCRIPTION:
Specimen A is labeled with the patient's name and "gastric antrum biopsy." Received in formalin is a 0.3 x 0.2 x 0.1 cm tan tissue. ES

LC/hh 03/29/17
Cassette verification identification by SJ

SLIDE INDEX:
A1 - gastric antrum

SPECIAL STAINS:
Giemsa
Positive control confirmed

SLIDE/BLOCK TALLY:
1 block, 1 H+E slide

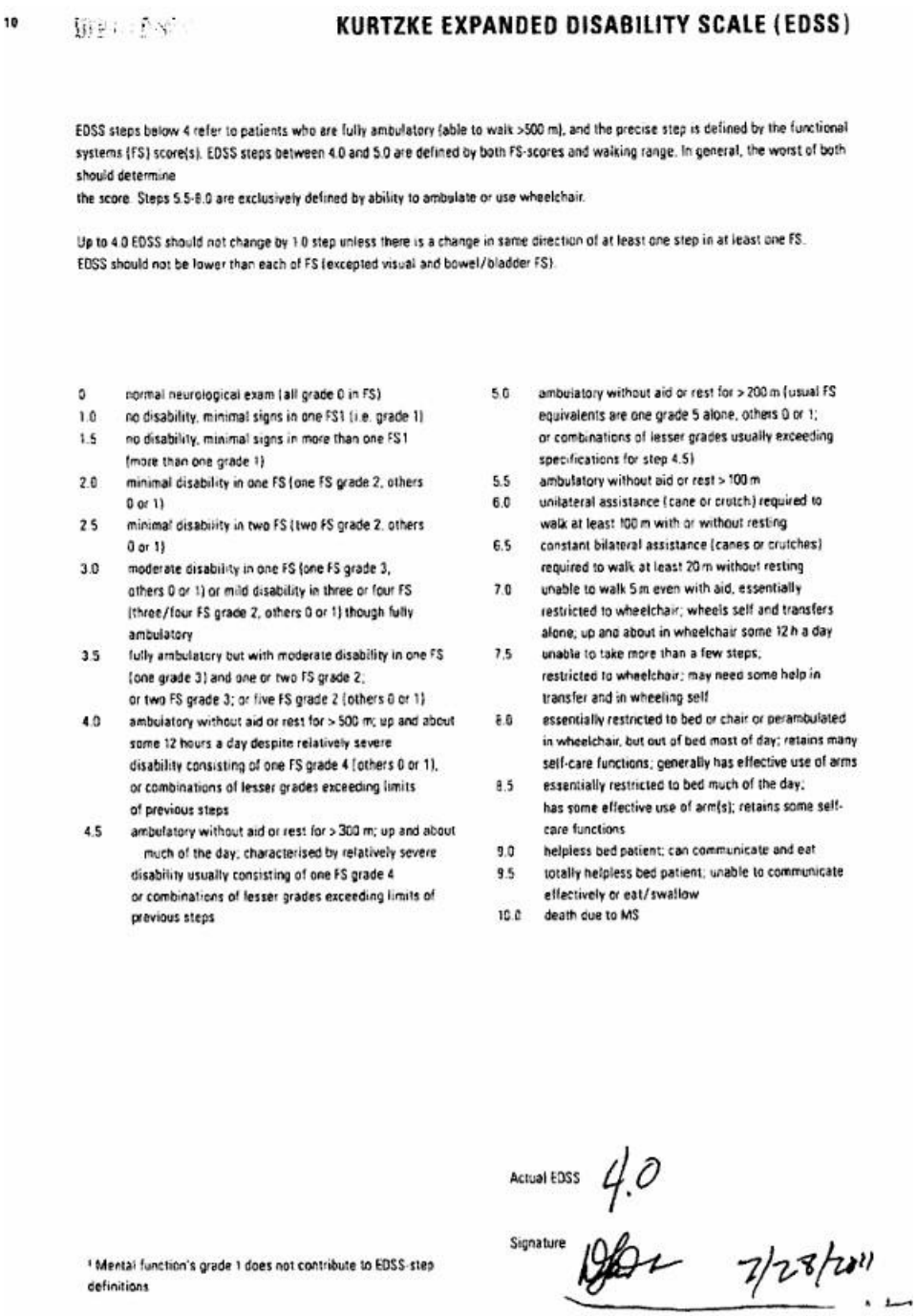
/gp

CPT Codes:
88305 x 1
88312 x 1

Figure 7 This graphic shows that chronic gastritis, GERD, and h. pylori were found during the preoperative endoscopy for sleeve gastrectomy.

3.5. Measure - *Record the established disability process to provide an initial benchmark of the progression prior to effective disability management*

Patient X was diagnosed with multiple sclerosis (MS) in 2008 following three years of intermittent symptoms that became increasingly persistent after a spider bite on his left thigh, which resulted in a nickel-sized area of necrotic tissue. After the formal diagnosis, his condition progressed significantly, leading to his application for disability retirement in February 2011, for which he received a fully favorable decision. At the time of retirement, his Expanded Disability Status Scale (EDSS) score was documented at 4.0.



10

KURTZKE EXPANDED DISABILITY SCALE (EDSS)

EDSS steps below 4 refer to patients who are fully ambulatory (able to walk >500 m), and the precise step is defined by the functional systems (FS) score(s). EDSS steps between 4.0 and 5.0 are defined by both FS-scores and walking range. In general, the worst of both should determine
the score. Steps 5.5-8.0 are exclusively defined by ability to ambulate or use wheelchair.

Up to 4.0 EDSS should not change by 1.0 step unless there is a change in same direction of at least one step in at least one FS. EDSS should not be lower than each of FS (excepted visual and bowel/bladder FS).

0	normal neurological exam (all grade 0 in FS)
1.0	no disability, minimal signs in one FS1 (i.e. grade 1)
1.5	no disability, minimal signs in more than one FS1 (more than one grade 1)
2.0	minimal disability in one FS (one FS grade 2, others 0 or 1)
2.5	minimal disability in two FS (two FS grade 2, others 0 or 1)
3.0	moderate disability in one FS (one FS grade 3, others 0 or 1) or mild disability in three or four FS (three/four FS grade 2, others 0 or 1) though fully ambulatory
3.5	fully ambulatory but with moderate disability in one FS (one grade 3) and one or two FS grade 2; or two FS grade 3; or five FS grade 2 (others 0 or 1)
4.0	ambulatory without aid or rest for > 500 m; up and about some 12 hours a day despite relatively severe disability consisting of one FS grade 4 (others 0 or 1), or combinations of lesser grades exceeding limits of previous steps
4.5	ambulatory without aid or rest for > 300 m; up and about much of the day; characterised by relatively severe disability usually consisting of one FS grade 4 or combinations of lesser grades exceeding limits of previous steps
5.0	ambulatory without aid or rest for > 200 m (usual FS equivalents are one grade 5 alone, others 0 or 1; or combinations of lesser grades usually exceeding specifications for step 4.5)
5.5	ambulatory without aid or rest > 100 m
6.0	unilateral assistance (cane or crutch) required to walk at least 100 m with or without resting
6.5	constant bilateral assistance (canes or crutches) required to walk at least 20 m without resting
7.0	unable to walk 5 m even with aid, essentially restricted to wheelchair; wheels self and transfers alone; up and about in wheelchair some 12 h a day
7.5	unable to take more than a few steps; restricted to wheelchair; may need some help in transfer and in wheeling self
8.0	essentially restricted to bed or chair or perambulated in wheelchair, but out of bed most of day; retains many self-care functions; generally has effective use of arms
8.5	essentially restricted to bed much of the day; has some effective use of arm(s); retains some self-care functions
9.0	helpless bed patient; can communicate and eat
9.5	totally helpless bed patient; unable to communicate effectively or eat/swallow
10.0	death due to MS

Actual EDSS 4.0

Signature 2/28/2011

[1] Mental function's grade 1 does not contribute to EDSS-step definitions

Figure 8 These findings indicate that patient X exhibited a relatively severe degree of disability in 2011 and was progressing toward complete loss of ambulation.

Define – *Did a shared problem or solution act as a nexus for each of the four medical interventions?*

Patient X maintained a healthy lifestyle, participating in semi-professional football from 2002 to 2003, holding various corporate positions, and serving with the Army Reserve until 2007. The onset of multiple sclerosis (MS) symptoms in 2005 was followed by rapid development of morbid obesity, which may have contributed to Helicobacter pylori infection, trigeminal and vagus nerve

dysfunction, and gastroesophageal reflux disease (GERD). Additionally, the MTHFR C677T mutation present in patient X could have impaired detoxification processes, while decreased physical activity may have hindered toxin elimination. Evidence indicates that morbidly obese patients with MS experienced improved walking ability following bariatric surgery compared to those who did not undergo this procedure (Bencsath et al., 2017). This journal, chronicling the progression of MS in patient X through a prolonged period of remission, may provide empirical support for future development of tools designed to help individuals modify risk factors associated with multiple sclerosis.

Disclosures

No disclosures by author

Declaration of generative AI in scientific writing

During the preparation of this work the author used Microsoft Copilot in order to improve the readability of the manuscript. After using Copilot, the author reviewed and edited the content as needed and takes full responsibility for the content of the published article.

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Ethics Statement

All guidelines of The Declaration of Helsinki for patient protection were met

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