2018 September 19th

- **Dr. Eric Williams – UCLA Hospital – Los Angles, California**
  - **Negligence →**

UCLA (University of California Los Angeles) Health
Dr. Eric Williamson:

Dr. Eric Williamson in UCLA neurology was consulted on September 19th 2018 (within a week of plasmapheresis) for follow-up therapy. After plasmapheresis a patient with MS is required to continue treatment with palliative therapies (in my case Tecfidera 240 mg twice a day). Considering the need for plasmapheresis it was apparent that my case of MS was severe and progressing.

The reason to consult Dr. Eric Williamson is specifically because the medications for MS are unaffordable without a prescription from a doctor in the US through insurance. The US restricts medications by requiring that the medications be prescribed by specifically a US neurologist.
All the tests required for MS are done at this point, MRIs, VEPs, optic tests are shows to Dr. Williamson but he refuses to acknowledge any of the tests as being relevant (they are the typical tests for MS). He writes a report that doesn't entirely correlate with the medical condition, but it shows that the US as a generality despite an overabundance of evidence to show the disease pathology and progression will always ignore the evidence.

Progress Notes by Williamson, Eric M., MD at 09/19/18 0800

Author: Williamson, Eric M., MD | Service: (none) | Author Type: Physician
Filed: 09/19/18 1217 | Encounter Date: 9/19/2018 | Status: Signed
Editor: Williamson, Eric M., MD (Physician)

Chief complaint:
I'm here for near follow up post plasma exchange. Relays that he has taken different therapies in the past, done research, and feels that dimethyl fumarate [his current Rx] has been better than prior interferon [that was poorly tolerated], yet may not be keeping his disease at bay and would like us to prescribe fingolimod for a diagnosis of multiple sclerosis.

History of Present Illness:
This is a very unusual encounter and begins with this young man having records scattered about in a binder and on computers, but little of which was sent to us from any other institution - more on that to come. He starts by providing report from an OCT and showing some MRimaging - mentioning occipital lesions and findings corresponding with visual pathway conduction abnormalities. Says he feels that plasma exchange should be followed by longer term immunotherapy and must be redirected to discuss his symptoms versus medical diagnosis or treatment strategy/plan a number of times.
Only current symptoms are some blurring of vision in the left eye and report of double vision and tingling in inner palms, middle thighs and under the eyes bilaterally but on the right more. He speaks quickly and continually says that taking medications has improved symptoms in the past but has not remained on therapy due to costs or other like issues but is hoping we can prescribe medication that can go through his insurance here in this country - noting Rx's to date were suggested or provided elsewhere.
When asked to focus on the chronology of symptoms, he again proves a difficult historian/reporter, but eventually agrees he was healthy prior then had gradual onset of 'physical pain' circa 2007-08 as he recounts his fingers and toes went numb - he reportedly first sought attention in 2008. Unfortunately, doesn't have discs with him and insteads provides documents including screenshots of brain MRI study with some mild hyperintensity in both basal ganglia - apparently not much was made of this and this doesn't appear on later studies. There may also be some mild signs of atrophy on same study, but this one looks similar to later pictures. He also reports he had severe headaches at the time - persistent and bilateral without remission; comments that the only time this remitted was with IV steroids starting 9.2017 and that valproic acid or other things were failed therapies in the interim.
Next reportedly experienced acute problems with mobility in 2008,09,10 - details leg stiffness and weakness episodes and reports around the same time he was experiencing trouble with pale vision bilaterally and interestingly shows what he says is a "photoshop" picture to try to demonstrate how parts of his vision were blurred bilaterally [shows a picture of a beach with peripheral areas and part of the center 'whited out' or more pale than blue sky in other areas.
Has extensive records, which amount to a computer file and some paper copies, that he has pieced together himself rather than physical or complete reports from other medical facilities; does, however, have some of the more complete MRI sequences loaded on to his computer - but again, does not provide us with copies of same for loading into our system.
Says he next obtained a 10.2012 MRI brain because he couldn't see extremities but could see central vision [when looking at other people] - this MRI of the brain looks essentially normal but in the occipital area there may be subtle hyperintensity, while the basal ganglia that may have had subtle hyperintensity on 2008 study looks normal. Says he had an optic test that confirmed 'occluded vision' but states nothing was done then.
Says he was in college at this time, but had to lay in bed due to headaches; then states pain in his extremities or face would often be associated with headaches. Nonetheless, he finished school, started working as an IT engineer, but by 2016 was suffering headaches to the point where he "couldn't think." Says a number of over the counter medications didn't help, eventually sought FDG-PET scan out of country that states there was hypometabolism in posterior parts of the brain consistent with a "neurodegenerative process." interestingly he later showed a normal study of the same reportedly from the same institution in East Asia and makes the argument that steroid course was the cause of the improvement.

Westwood Neurology
300 Medical Plaza Suite B200
Los Angeles CA 90095-0001

Jana, Narendra
MRN: 5526268, DOB: 10/27/1984, Sex: M

Progress Notes by Williamson, Eric M., MD at 09/19/18 0800 (continued)

He is difficult to correct or redirect and is allowed to expound on how this changed over time. The only notes I received from outside institution mention he was cared for in India around this time but he doesn't mention this nor confirm/deny at any point.
He also had a 2016 EEG [in yet another country in 'Asia'] showing sharp waves in posterior parts of the brain prompting additional EEG testing that showed predominantly right frontal discharges - he was reportedly started on keppra [possibly in addition to valproate]. Complains that at this point he was experiencing "pseudobulbar affect" [arguing that this is a clear sign of multiple sclerosis when point that its unclear why this diagnosis had been considered] - saying he was experiencing spells of crying or laughing for no reason. Says antiepileptic medications didn't help or even worsened symptoms for the 2-3 months without benefit in physical symptoms either - complaining that his optic "neuropathy" seemed to get worse as did lack of sensation in limbs.
By 1.2017 he sought attention in Europe that led to C and T spine MRI - which reportedly showed central lesions presumed to explain some of his symptoms. Encounter becomes quite frustrating at this point as he withholds control of the computer/study so I can review but when he is forced to allow me to look at the pictures I can confirm that there is lack of lesions nor atrophy that he is arguing and instead we see prominence of the tracts symmetrically and bilaterally in the c spine on axial images. He says the reports are in another language from eastern europe when I asked to review them then says he doesn't have them with him when I requested to see them anyways. After much debate, he then allows me to see the t spine pictures which similarly shows subtle central intensity - more apparent than any in the c spine yet only visible on sagittal pictures and not seen on limited axial slices that do not show any abnormality nor extend low enough to examine the suspected finding on t spine study. Despite all the reported findings, he says he had no follow up out of country at the time and instead came back to the united states at this point. The one record we have from another healthcare system is scanned in as an 8.2017 visit to an ER with neurology consult [nonetheless says he was in a southern California ER prior in 4.2017 for visual issues described as loss of vision and sent home] - he is confronted with the fact that he has not relayed any information consistent with diagnosis of MS nor the reported from outside record treatment by this time as stated in said record and then says he went to tijuana to see a neurologist [versus ER] that reportedly confirmed or had suspicion of MS. Next contradicts and says he went to mexico city or that in 2016 it had been suggested he may have an immune condition then but when asked pointedly says it wasn't until 9.2017 that he started on any immune therapy then says he took interferon around this time due to findings on spine MRI which "helped some of his physical pain and numbness but didn't do very much." Oddly the patient uses terms like give-way weakness to detail what other physicians may have found. Next says because symptoms got worse he went [back?] to mexico city where they gave him 5 days of methylprednisone which helped him recover sensation for a couple months at most. Provides follow up imaging from 'shortly after that time' 9.2017 of his brain that again shows subtle occipital hyperintensities and the questionable mild atrophy. Would comment that this is less impressive than mild atrophy. Again asking for report, he claims he has same from mexico city hospital but doesn't provide. Next says circa 10.2017 for recurrence of visual and sensory symptoms he reportedly sought medical attention in Brazil, says he got steroids there, which again helped. Next he followed up in mexico and shows limited brain images dated 10.2017 that were unchanged from 9.2017 study. Confuses dates but sometime between 10 and 12.2017 he started fingolimod - reports he did well on this but discontinued by late 2017 due to costs.
Says 1.2018 he went back to tijuana for recurrence of symptoms and they again dosed steroids for a "third time." says his ability to see the color of skin tones improved again thereafter, albeit briefly. By 3 or 4.2018 he went to an ER in Germany while 'on vacation again' where he was told the reason he comes back to the ER is because he needs consistent outpatient management for his MS and was treated with steroids - he subsequently saw a physician in Berlin named Dr. Klatkfe who reportedly prescribed interferon again. Says he's been paying out of pocket for the medication, but its more expensive than steroids or ER visits. Says he had another visit to east asia where FDG-PET was repeated 4.2018 - reportedly they dosed methylprednisone and believed afterwards there was an improvement in brain metabolism thereafter. Had another brain MRI 5.2018 that he shows brief sagital picture from with possible T2 changes posteriorly again and when I ask about any other spine MRI's says he's had 7 total but doesn't provide any additional images than those 1.2017 studies above aside from briefly showing a jpeg capture from limited normal appearing sagital studies reported

Progress Notes by Williamson, Eric M., MD at 09/19/18 0800 (continued)

to be from 8.2017. Does provide papers with heading stating they are from Bumrungrad International hospital in "east asia" which state there is slight delay in average P100 latency of the left eye circa 8.2018.
Finally, Says he was treated with plasma exchange in mexico city last week for complaint of worsened symptoms above, and this helped again.
I interjected again at end of history to confront report from outside hospital stating he had been treated with cyclophosphamide and methotrexate in the past, he neither denies nor makes it clear that these things did or didn't happen and becomes agitated when I discuss that we would not make nor confirm a diagnosis of multiple sclerosis with what we have and certainly could not prescribe a medication such as fingolimod given the relayed history and findings below, including feigned weakness on exam and questionable report of sensory disturbance on the left hemibody splitting the midline and including the face and scalp.

No past medical history on file.

No outpatient prescriptions prior to visit.

No facility-administered medications prior to visit.

No family history on file.
Denies any history of multiple sclerosis or known neurologic illness in family

Social History
Substance Use Topics

- Smoking status: Never Smoker
- Smokeless tobacco: Never Used
- Alcohol use: Not on file

EXAM
BP 125/80 | Pulse 102 | Temp 36.7 °C (98 °F) | Ht 1.676 m (5' 6") | Wt 53.9 kg (118 lb 12.8 oz) | BMI 19.17 kg/m²
Cranial nerve exam is unremarkable, pupils are equal round and reactive to light and extraocular movements are intact but when asked to look left he blinks repeatedly.
Motor power: Right/Left [x out of 5] - complicated by inconsistent effort and give-way weakness on the left at times, best recordable/observed as follows:
Deltoids: 5/5-
Triceps: 5/5-
Wrist extension: 5/5-
Finger abduction: 4/4
Grip: 5/5-
Hip Flexion: 5-/4+
Knee flexion: 5/5-
Ankle dorsiflexion: 5/5
Sensory -reports decrease sensation to light touch and pinprick throughout the left hemibody splitting the midline in bilateral extremities, trunk, buttocks, back and neck/face/scalp
Gait - completely normal when observed walking in or out of the exam room from waiting room

Labs and Studies:
Reviewed the provided above but would caution that none of them came from outside institution and are provided by patient

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Jana, Narendra
MRN: 5526268, DOB: 10/27/1984, Sex: M

Progress Notes by Williamson, Eric M., MD at 09/19/18 0800 (continued)

Assessment and Plan:
The clinical picture, which should be the most important factor in considering a diagnosis of multiple sclerosis, lacks a history of acute focal deficits in different areas with lasting symptoms that may be result of central nervous system inflammatory disease such as MS. Imaging can be particularly helpful in considering such a diagnosis and his brain MRIs in particular are largely devoid of findings as we might see in multiple sclerosis. The spinal MRI's were similarly without clear T2 changes that may raise suspicion of CNS inflammatory condition. He indeed may have subtle atrophy, but this was consistent across studies and similarly not pathologic. Discussed all this with patient and agreed that for us to move forward with any further consideration of this diagnosis we would like to start with imaging performed here or at other facility that can communicate result directly to us and offered return visit within 1-2 months thereafter.
We would not make/confirm or suggest a diagnosis of multiple sclerosis at this time otherwise, and certainly would caution against initiating disease modifying therapy with lack of clear clinical relapses [different focal problems] or MRI changes that we think it may be beneficial for at this time, especially when weighing some of the not insignificant side effects that may be encountered with same.

There are a number of confounding factors, but if one took additional reported studies at reported face value - which, we would caution against, the only other clear abnormalities or things worthy of further evaluation/consideration might be slowed conduction of the left optic nerve or abnormal EEG findings. Would suggest repeat testing to either of these points should be considered before making any such plan and would not offer here in our multiple sclerosis clinic at this time.

Aside from strong suspicion of somatoform disorder as contributory if not explanatory of much of this clinical picture, one could also suspect primary optic conditions and it may prove useful to seek additional consultation and/or study to this affect.

Would suggest communicating with Hospital Angeles del Pedregal, Centro de espcialidades quiranges is warranted but says he doesn't want any notes sent elsewhere; we will, however, at a minimum, communicate this consultation report with his primary care doctor and refer him to medical records if he would like a copy himself.

I spent a total of 110 minutes face to face with the patient of which greater than 50% of that time was spent in counseling/coordination.

Electronically signed by Williamson, Eric M., MD at 09/19/18 1217

*** End of Report ***

END OF REPORT

A statement to statement negation is included with the report:

1

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Chief complaint: I'm here for near follow up post plasma exchange. Relays that he has taken different therapies in the past, done research, and feels that dimethyl fumarate [his current Rx] has been better than prior interferon [that was poorly tolerated] , yet may not be keeping his disease at bay and would like us to prescribe fingolimod for a diagnosis of multiple sclerosis. **History of Present Illness:** This is a very unusual encounter and begins with this young man having records scattered about in a binder and on computers, but little of which was sent to us from any other institution - more on that to come. He starts by providing report from an OCT and showing some MRimaging - mentioning occipital lesions and findings corresponding with visual pathway conduction abnormalities. Says he feels that plasma exchange should be followed by longer term immunotherapy and must be redirected to discuss his symptoms versus medical diagnosis or treatment strategy/plan a number of times.	Its logical, after plasmapheresis most patients with MS are required to stay on their medications. Without a prescription from a doctor in the US under insurance the medications are too expensive (2-5 thousands per month).

2

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Only current symptoms are some blurring of vision in the left eye and report of double vision and tingling in inner palms, middle thighs and under the eyes bilaterally but on the right more. He speaks quickly and continually says that taking medications has improved symptoms in the past but has not remained on therapy due to costs or other like issues but is hoping we can prescribe medication that can go through his insurance here in this country - noting Rx's to date were suggested or provided elsewhere. When asked to focus on the chronology of symptoms , he again proves a difficult historian/reporter, but eventually agrees he was healthy prior then had gradual onset of 'physical pain' circa 2007-08 as he recounts his fingers and toes went numb - he reportedly first sought attention in 2008. Unfortunately, doesn't have discs with him and insteads provides documents including screenshots of brain MRI study with some mild hyperintensity in both basal ganglia - apparently not much was made of this and this doesn't appear on later studies. There may also be some mild signs of atrophy on same study, but this one looks similar to later pictures.	The 2008 and 2017 MRI are uploaded into the system. Both are gross pathologies. Th atrophy is significant by year 2012, posterior brain shows significant atrophy relative to 2008.

3

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
He also reports he had severe headaches at the time - persistent and bilateral without remission; comments that the only time this remitted was with IV steroids starting 9.2017 and that valproic acid or other things were failed therapies in the interim. Next reportedly experienced acute problems with mobility in 2008,09, 1 O - details leg stiffness and weakness episodes and reports around the same time he was experiencing trouble with pale vision bilaterally and interestingly shows what he says is a "photoshop" picture to try to demonstrate how parts of his vision were blurred bilaterally [shows a picture of a beach with peripheral areas and part of the center 'whited out' or more pale than blue sky in other areas. Has extensive records , which amount to a computer file and some paper copies, that he has pieced together himself rather than physical or complete reports from other medical facilities; does, however, have some of the more complete MRI sequences loaded on to his computer - but again , does not provide us with copies of same for loading into our system.	The records were uploaded to the system immediately after the appointment. My objective is to determine if the doctor is honest about the condition (he wasn't) during the appointment, which is more than trivial at that point and clearly apparent when I met the doctor. The prescription is relatively benign for any patient and there would be no risks in giving it. There is greater risk in not giving the medication than giving the medication since its right plasmapheresis. I did upload MRIs of the brain, cervical spine, optic neuropathy tests and the initial large lesions from 2008. This evidence is more than enough to substantiate the need for medications for MS.

4

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Says he next obtained a 10.2012 MRI brain because he couldn't see extremities but could see central vision [when looking at other people] - this MRI of the brain looks essentially normal but in the occipital area there may be subtle hyperintensity, while the basal ganglia that may have had subtle hyperintensity on 2008 study looks normal. Says he had an optic test that confirmed 'occluded vision' but states nothing was done then. Says he was in college at this time, but had to lay in bed due to headaches; then states pain in his extremities or face would often be associated with headaches. Nonetheless , he finished school, started working as an IJ engineer , but by 2016 was suffering headaches to the point where he "couldn't think." Says a number of over the counter medications didn't help, eventually sought FDG-PET scan out of country that states there was hypometabolism in posterior parts of the brain consistent with a "neurodegenerative process." interestingly he later showed a normal study of the same reportedly from the same institution in East Asia and makes the argument that steroid course was the cause of the improvement.	That's an inaccurate statement, the FDG pet shows improvements from a medication for MS, methylprednisolone, but it doesn't show a normal FDG pet. Its simply an indication that its specifically an neuroinflammatory process that causes the condition responding to methylpredsiolone. The doctor should immediately suspect MS with such a response.

5

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
He is difficult to correct or redirect and is allowed to expound on how this changed over time. The only notes I received from outside institution mention he was cared for in India around this time but he doesn't mention this nor confirm/deny at any point.	I had an EEG done in India in 2016 but medical treatment there hadn't taken place till 4 months later in January 14th of 2019. India was never mentioned in the appointment except in reference to the EEG.
He also had a 2016 EEG [in yet another country in 'Asia'] showing sharp waves in posterior parts of the brain prompting additional EEG testing that showed predominantly right frontal discharges - he was reportedly started on keppra [possibly in addition to valproate] .	
Complains that at this point he was experiencing "pseudobulbar affect" [arguing that this is a clear sign of multiple sclerosis when point that its unclear why this diagnosis had been considered] - saying he was experiencing spells of crying or laughing for no reason. Says antiepileptic medications didn't help or even worsened symptoms for the 2-3 months without benefit in physical symptoms either - complaining that his optic "neuropathy" seemed to get worse as did lack of sensation in Limbs.	That's fairly typical effect in MS, its because its not wise to give antiepileptics in neuroinflammatory conditions. The withdrawal effects from antiepileptics would cause pseudobulbar effects. Even coffee causes pseudobulbar effect.

6

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
By 1.2017 he sought attention in Europe that led to C and T spine MRI - which reportedly showed central lesions presumed to explain some of his symptoms. Encounter becomes quite frustrating at this point as he withholds control of the computer/study so I can review but when he is forced to allow me to look at the pictures I can confirm that there is lack of lesions nor atrophy that he is arguing and instead we see prominence of the tracts symmetrically and bilaterally in the c spine on axial images. He says the reports are in another language from eastern europe when I asked to review them then says he doesn't have them with him when I requested to see them anyways. After much debate, he then allows me to see the t spine pictures which similarly shows subtle central intensity - more apparent than any in the c spine yet only visible on sagittal pictures and not seen on limited axial slices that do not show any abnormality nor extend low enough to examine the suspected finding on t spine study.	I didn't withhold the control of the computer but I did have to read the summary of the appointment in the computer as well. The atrophy and neurodgeration is readily apparent in the MRI images. Neurologists are specifically interested in the FLAIR sequences to checks for signs of atrophy or neurodegeration and my MRI shows neurodegeration from C2 to C5, unavoidable. Its easy for any lay person to pick out the neurodegeration in about 5 seconds due to its prominence. The t spine does central intensity that turns into atrophy in the next series, thus showing the nature of how atrophy occurs. All neurologists know this.

7

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Despite all the reported findings , he says he had no follow up out of country at the time and instead came back to the united states at this point. The one record we have from another healthcare system is scanned in as an 8.2017 visit to an ER with neurology consult [nonetheless says he was in a southern California ER prior in 4.2017 for visual issues described as loss of vision and sent home] - he is confronted with the fact that he has not relayed any information consistent with diagnosis of MS nor the reported from outside record treatment by this time as stated in said record and then says he went to tijuana to see a neurologist [versus ER] that reportedly confirmed or had suspicion of MS. Next contradicts and says he went to mexico city or that in 2016 it had been suggested he may have an immune condition then but when asked pointedly says it wasn't until 9.2017 that he started on any immune therapy then says he took interferon around this time due to findings on spine MRI which "helped some of his physical pain and numbness but didn't do very much ."	It should be readily apparent by this point in the appointmet that I do have MS in the appointment. With complaints of psychical immobility (and ER to substantiate it), optic neuropathy, neruospinal degeration, and dementia as a secondary effect of MS that's the 100 % correlation of symptoms that would indicate that the patient definitely has MS. It didn't do very much because it was determined soon afterwards that I do have a more serious form of MS called secondary progressive MS, which wouldn't respond to interferons.

8

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Oddly the patient uses terms like give-way weakness to detail what other physicians may have found. Next says because symptoms got worse he went [back?] to mexico city where they gave him 5 days of methylprednisone which helped him recover sensation for a couple months at most.	Dr. William's explains in the appointment that "give away" means "pretending". Given that I have lesions in the brain and spinal column that only respond to medications for secondary progressive MS that statement would not be substantiated. About 1.5 to 2 months at most due to the secondary progressive nature of my MS (which was unknown at that point).
Provides follow up imaging from 'shortly after that time' 9.2017 of his brain that again shows subtle occipital hyperintensities and the questionable mild atrophy. Would comment that this is less impressive than mild atrophy . Again asking for report, he claims he has same from mexico city hospital but doesn't provide. Next says circa 10.2017 for recurrence of visual and sensory symptoms he reportedly sought medical attention in Brazil , says he got steroids there , which again helped. Next he followed up in mexico and shows limited brain images dated 10.2017 that were unchanged from 9.2017 study. Confuses dates but sometime between 10 and 12.2017 he started fingolimod - reports he did well on this but discontinued by late 2017 due to costs .	Atrophy is as large as placing several fingers in the empty space. Not present in earlier series. The cost is the intended purpose of the appointement.
Says 1.2018 he went back to tijuana for recurrence of symptoms and they again dosed steroids for a "third time." says his ability to see the color of skin tones improved again thereafter , albeit briefly .	Change in ability to to see indicates optic neuropathy.

9

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
By 3 or 4.2018 he went to an ER in Germany while 'on vacation again' where he was told the reason he comes back to the ER is because he needs consistent outpatient management for his MS and was treated with steroids – he subsequently saw a physician in Berlin named Dr. Klatkfe who reportedly prescribed interferon again . Says he's been paying out of pocket for the medication, but its more expensive than steroids or ER visits. Says he had another visit to east asia where FOG-PET was repeated 4.2018 - reportedly they dosed methylprednisone and believed afterwards there was an improvement in brain metabolism thereafter . Had another brain MRI 5.2018 that he shows brief sagital picture from with possible T2 changes posteriorly again and when I ask about any other spine MRI's says he's had 7 total but doesn't provide any additional images than those 1.2017 studies above aside from briefly showing a jpeg capture from limited normal appearing sagital studies reported to be from 8.2017 . Does provide papers with heading stating they are from Bumrungrad International hospital in "east asia" which state there is slight delay in average P100 latency of the left eye circa 8.2018.	There were full optic tests that are part of the same document that that were also shows to Dr. Willam's. The optic tests show optic nerve inflammation directly in the image.

10

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Finally, Says he was treated with plasma exchange in mexico city last week for complaint of worsened symptoms above, and this helped again . I interjected again at end of history to confront report from outside hospital stating he had been treated with cyclophosphamide and methotrexate in the past , he neither denies nor makes it clear that these things did or didn't happen and becomes agitated when I discuss that we would not make nor confirm a diagnosis of multiple sclerosis with what we have and certainly could not prescribe a medication such as fingolimod given the relayed history and findings below, including feigned weakness on exam and questionable report of sensory disturbance on the left hemibody splitting the midline and including the face and scalp.	The "feigned weakness" shown to be further neurodegeration along the cervical spine that leads to an emergency situation in a foreign nation that requires IV rituximab to manage the rate of deterioration. The doctors pretense is readily apparent in the appointment. There are repeated videos of the presentation for substantiation as well.

11

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
No past medical history on file . No outpatient prescriptions prior to visit. No facility-administered medications prior to visit. No fam ily history on file . Denies any history of multiple scleros is or known neurologic illness in family Social History Substance Use Topics • Smoking status : • Smokeless tobacco : • Alcohol use EXAM Never Smoker Never Used Not on file BP 125/80 I Pulse 102 I Temp 36. 7 °C (98 °F) I Ht 1.676 m (5' 6") I Wt 53. 9 kg (118 lb 12.8 oz) I BMI 19.17 kg/m2 Cranial nerve exam is unremarkable , pupils are equal round and reactive to light and extraocular movements are intact but when asked to look left he blinks repeatedly. Motor power: Right/Left [x out of 5] - complicated by inconsistent effort and give-way weakness on the left at times , best recordable/observed as follows :	Drug tests are included. The statement "give-way weakness" is a way to circumvent giving the medication. Its clearly apparent that I woudlnthave the capability to move my limbs.

12

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Deltoids: 5/5- Triceps : 5/5- Wr ist extension : 5/5- Finger abduction : 4/4 Grip: 5/5- Hip Flexion: 5-/4+ Knee flexion: 5/5- Ankle dorsiflexion : 5/5 Sensory -reports decrease sensation to light touch and pinprick throughout the left hemibody splitting the midline in bilateral extremities , trunk , buttocks , back and neck/face/scalp Gait - completely normal when observed walking in or out of the exam room from waiting room Labs and Studies: Reviewed the provided above but would caution that none of them came from outside institution and are provided by patient	

13

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Assessment and Plan: The clinical picture, which should be the most important factor in considering a diagnosis of multiple sclerosis, lacks a history of acute focal deficits in different areas with lasting symptoms that may be result of central nervous system inflammatory disease such as MS. Imaging can be particularly helpful in considering such a diagnosis and his brain MRIs in particular are largely devoid of findings as we might see in multiple sclerosis. The spinal MRI's were similarly without clear T2 changes that may raise suspicion of CNS inflammatory condition . He indeed may have subtle atrophy , but this was consistent across studies and similarly not pathologic . Discussed all this with patient and agreed that for us to move forward with any further consideration of this diagnosis we would like to start with imaging performed here or at other facility that can communicate result directly to us and offered return visit within 1-2 months thereafter.	

14

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
We would not make/confirm or suggest a diagnosis of multiple sclerosis at this time otherwise, and certainly would caution against initiating disease modifying therapy with lack of clear clinical relapses [different focal problems] or MRI changes that we think it may be beneficial for at this time , especially when weighing some of the not insignificant side effects that may be encountered with same. There are a number of confounding factors , but if one took additional reported studies at reported face value - which , we would caution against , the only other clear abnormalities or things worthy of further evaluation/consideration might be slowed conduction of the left optic nerve or abnormal EEG findings. Would suggest repeat testing to either of these points should be considered before making any such plan and would not offer here in our multiple sclerosis clinic at this time. Aside from strong suspicion of somatoform disorder as contributory if not explanatory of much of this clinical picture , one could also suspect primary optic conditions and it may prove useful to seek additional consultation and/or study to this affect.	The medical findings would indicate a gross pathology of MS and the "optic condition" is a typical presentation of optic neuropathy in the setting of MS. The doctor appears to be trying to circumvent giving the medications.

15

9.19.2019-UCLA-Dr. Eric William's Summary Original Text	Statement by Statement Negation
Would suggest communicating with Hospital Angeles del Pedregal, Centro de espcialidades quiranges is warranted but says he doesn't want any notes sent elsewhere; we will, however , at a minimum , communicate this consultation report with his primary care doctor and refer him to medical records if he would like a copy himself . I spent a total of 110 minutes face to face with the patient of which greater than 50% of that time was spent in counseling/coordination .	

Considering the course of events (immediately proceeding a plasmapheresis procedure) and that the doctors acknowledged that most of the typical features of MS are present in this appointment, not giving the medications would be dangerous to the patient. Its clear negligence. It may even be typified as a form of assault since the clinician is required to prescribe the follow-up medication after plasmapheresis.