

**2018 September 21<sup>st</sup>**

- **Dr. Philip Kinkel – UCSD- San Diego, California**
  - **Criminal Negligence →**

**UC San Diego Health**

**Dr. Revere Kinkel:**

Dr. Revere Kinkel would be the very last neurologist that I saw in the United States. And the intent to perpetuate medical negligence was present in the very last appointment as well. The appointment is the typical format of past US neurology appointments, even when there is clear indication with all the medical evidence to substantiate giving the medications for MS the doctor denies all the diagnostics and many of the medical statements are not substantiable. This was immediately after two neurologist appointments abroad that confirmed that I do have a secondary progressive form of MS that requires persistent management and treatment.

Dr. Kinkel's report is given below, followed by a statement by station negation of his report.

UC San Diego Health 200 W. Arbor Dr.  
UC San Diego Health  
San Diego CA 92103

JANA, NARENDRA NIRMAL  
MRN: 30340724  
DOB: 10/27/1984, Sex: M  
Enc Date: 09/21/18

Visit Information

Arrival: 09/21/2018 1629	Department: Mon Neurology	First Attending:
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Progress Notes

Progress Notes by Kinkel, Revere Philip, MD at 09/21/18 1400

Version 1 of 1

Author: Kinkel, Revere Philip, MD	Service: (none)	Author Type: Attending Physician
Filed: 09/30/18 1042	Encounter Date: 9/21/2018	Status: Signed
Editor: Kinkel, Revere Philip, MD	(Attending Physician)	

University of California San Diego  
Multiple Sclerosis Center

First visit: 9/21/2018 Last visit: NA Current visit: 9/21/2018

Consultation Source: self referred

Reason for Visit: "to get an authorization to receive Tecfidera in the united states".

Principle Neurological Diagnosis: Deferred pending further diagnostic evaluation and review of entire records

Narrative History for Current Visit: Narendra Nirmal Jana is a 33 year old male residing in Massachusetts who returns to San Diego every two months. He travels frequently to Europe and Mexico and is also seen by providers in those locations. He was seen alone today.

The encounter with this young man was extremely odd and confusing and would have required a visit in excess of 1-2 hrs to make any headway, as for the note in EPIC from a recent visit to a UCLA Neurologist (Dr Williamson) who he visited on 9/19/18, two days before his visit with me. As described Dr Williamson's consultation note (much of which I copied below), I too entered the exam room and found Narendra seated with his laptop open to show me MR images. He rapidly informed me of his prior diagnosis of MS by various neurologists in Europe, Mexico and elsewhere (none in the states) and that the diagnosis was unquestionably correct and well documented. He would not initially provide any history (more on this later) and would only agree to provide some of his extensive records; he was evasive when asked why he refused to provide all of his records and imaging studies; in fact he specifically uploaded only certain MR images into our PACs system and refused to upload the rest, concerned that the extra images would bias my viewpoint.

Eventually and with much effort he provided some historical evidence of symptoms, albeit not necessary consistent with MS. Interesting the account I received was similar to that provided to Dr Williamson at UCLA (his history is provided below), so I have appended his history as well.

Narendra informed me that his symptoms began in 2009 when he noticed "pale vision" and gradual constriction of temporal fields which progressed over several years. He then showed me Humphrey Perimeter tests with severely constricted fields (tunnel vision). He became argumentative when I informed him that the Humphrey perimetry results were not consistent with an optic neuropathy or MS (more consistent with glaucoma). From here the history became disjointed and consisted mostly of multi-focal numbness and paresthesias with gait problems developing at some point in time. I left the room at this point to review epic records and found the following note by Dr Williamson. I was able to confirm much of Dr Williamson's history when I subsequently returned to talk with Narendra further.

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Progress Notes (continued)

Progress Notes by Kinkel, Revere Philip, MD at 09/21/18 1400 (continued)

Version 1 of 1

History provided to Dr Williamson at UCLA (9/19/18) (Copied from his note verbatim)

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 instead 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 'blotted 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.

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.

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 it's 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 withdraws 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

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**Progress Notes (continued)****Progress Notes by Kinkel, Revere Philip, MD at 09/21/18 1400 (continued)**

Version 1 of 1  
pictures and not seen on limited axial slices that do not show any abnormality nor extend low enough to examining 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 methylprednisolone 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. Klatte 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 methylprednisolone and believed afterwards there was an improvement in brain metabolism thereafter. Had another brain MRI 5.2018 that he shows brief sagittal 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 sagittal 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. 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.

**Review of Systems: Complete 10 point review of symptoms from the patient self report questionnaire**

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**Progress Notes (continued)****Progress Notes by Kinkel, Revere Philip, MD at 09/21/18 1400 (continued)**

Version 1 of 1

was reviewed with the patient today. Any pertinent positives are listed above in the narrative history or problem list.

No Known Allergies

**Current Outpatient Prescriptions**

Medication:  Siq  

- adapalene (DIFERIN) 0.1 % cream Apply 1 Application topically nightly. Use a small amount as directed
- Dimethyl Fumarate (TECFIDERA) 240 MG CPDR Take 240 mg by mouth 2 times daily.
- HYDROcodone-acetaminophen (NORCO) 5-325 MG tablet Take 1 tablet by mouth every 6 hours as needed for Moderate Pain (Pain Score 4-6).

No current facility-administered medications for this visit.

**Past Medical History:**

Diagnosis:  Date  

- Multiple sclerosis (CMS-HCC)

**Past Surgical History:**

Procedure:  Laterality Date  

- NO PAST SURGERIES

**Family Medical History:**

Family History: Problem:  Relation:  Age of Onset:  

- Diabetes Maternal Grandmother
- Diabetes Maternal Grandfather

**Examination:**

BP 123/76 (BP Location: Left arm, BP Patient Position: Sitting, BP cuff size: Regular) | Pulse 95 | Temp 98.2 °F (36.8 °C) (Oral) | Ht 5' 6" (1.676 m) | Wt 52.2 kg (115 lb) | SpO2 98% | BMI 18.56 kg/m<sup>2</sup>  
General: Examination of the skin, joints and extremities revealed no abnormalities ; There were no cervical, ocular or cranial bruits.

Cognitive/Behavioral: Examination of cognition, language and prosody revealed no abnormalities . Behavioral and affect was appropriate.

Cranial Nerves: Near visual acuity was J1+ OD and J5 OS with glasses for correction.10 /10 color plates were identified OD and 3/10 OS. Visual fields were full to confrontation testing. Lids were normal . Pupils were 7mm and reactive with no RAPD . Fundoscopic exam revealed definite optic nerve pallor OS> OD and large optic cups. Eyes were orthotropic . Pursuit and saccadic eye movements revealed no abnormalities. Facial movements revealed no abnormalities. Testing of facial sensation revealed no abnormalities. Hearing was normal AD and normal AS. Bulbar examination revealed no abnormalities .

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**Motor:** Marked giveaway weakness on the left side with normal tone and no atrophy. Resistive strength in all limbs

**Cerebellar:** Very slow FTN with dysmetria on the left (see gait for description of midline/axial cerebellum dysfunction)

**Sensory:** Sensation to pp and temperature was NT. Vibration sensation duration (secs) was (R/L): Upper extremity middle finger ND/ND; Lower extremity big toe NT/NT. Joint position sensation was normal. Stereognosis was normal

**Reflexes:** (R/L): Biceps 0/0, BR 2-/2-, Triceps 2-/2-, Patella 2/2, Ankle 0/0. There was no clonus. The right toe was downgoing and the left toe downgoing on plantar stimulation

**Gait Description:** Slow hesitant narrow based gait with slight LLE leg and knee hyperextension

**Performance Measures:**

9-Hole PEG Test	9/21/2018
RUE	30.21
RUE (Best)	26.03
LUE	46.23
LUE (Best)	43.6

25 Ft. Ambulation Time	9/21/2018
25 ft Ambulation Time - 1st Trial (Seconds)	10.8
Independently or With Assistance?	Independently
25 ft Ambulation Time - 2nd Trial (Seconds)	9.84
Independently or With Assistance?	Independently

No flowsheet data found.

No flowsheet data found.

No flowsheet data found.

No flowsheet data found.

**Review of Imaging Studies:**

1. MRI of brain 12/18/2008 was reviewed: Normal
2. MRI of brain 10/27/12 was reviewed: normal except vague T2 hyperintensity posterior PVWM of no significance
3. MRI of the cervical spine 1/10/17 was reviewed: no intramedullary abnormalities
4. MRI of thoracic spine off of his computer only (not allowing me to handle the controls) may show a mid

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Version 1 of 1

**Non Imaging Studies**

1. VEP 8/10/18 P100 118 OS and 105 OD
2. Humphrey Perimetry results with severe constricted fields

**Review of Labs:**

1. CSF reportedly normal

**Assessment:**

Very difficult to assess this young man. He has done some extensive doctor shopping all over the world (Europe, India, Asia, USA, Mexico, South America) and is convinced of his diagnosis and needs. I am very concerned that the visual symptoms and visual evaluations are more suggestive of glaucoma or other process. Certainly his Humphrey Perimetry results are not consistent with an optic neuropathy. At the end of the visit I told him I would be willing to see him again and work with him only if he follows through on my recommendations listed below.

After discussing the options available and the potential benefits (pros) and risks (cons), we mutually decided on the following management plan

**Plan/contract:**

If you are willing to upload all your films, get blood drawn today, see ophthalmology, and get standard CSF analysis (which you are going to do outside this country) we will consider prescribing medications if the evaluation is consistent with MS or another inflammatory disorder. Otherwise we will not see you again

Total time spent by Dr Kinkel with the patient today was 70 minutes with over 50% of the time spent on education and counseling regarding their problem list and the assessment and plan

**All Results**

No results found

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A statement by statement negation of Dr. Kinkel's report is given below:

<p><b>1</b></p> <p><b>9.21.2018-Dr. Philip Revere Kinkle Summary Original Text</b></p> <p>Consultation Source: self referred Author Type: Attending Physician Status: Signed Reason for Visit: "to get an authorization to receive Tecfidera in the united states" Principle Neurological Diagnosis: Deferred pending further diagnostic evaluation and review of entire records Narrative History for Current Visit: Narendra Nirmal Jana is a 33 year old male residing in Massachusetts who returns to San Diego every two months. He travels frequently to Europe and Mexico and is also seen by providers in those locations. He was seen alone today. The encounter with this young man was extremely odd and confusing and would have required a visit in excess of 1 1/2 hrs to make any headway, if not for the note in EPIC from a recent visit to a UCLA neurologist (Dr Williamson) who he visited on 9/19/18, two days before his visit with me. As described Dr Williamson's consultation note (much of which I copied below), I too entered the exam room and found Narendra seated with his laptop open to show me MR images.</p>	<p><b>Statement by Statement Negation</b></p> <p>It was referred by Dr. Nicholas Dembitsky from Sharp hospital</p>	<p><b>2</b></p> <p><b>9.21.2018-Dr. Philip Revere Kinkle Summary Original Text</b></p> <p>He rapidly informed me of his prior diagnosis of MS by various neurologists in Europe, Mexico and elsewhere (none in the states) and that the diagnosis was unquestionably correct and well documented. He would not initially provide any history (more on this later) and would only agree to provide some of his extensive records; he was evasive when asked why he refused to provide all of his records and imaging studies; in fact he specifically uploaded only certain MR images into our PACS system and refused to upload the rest, concerned that the extra images would bias my viewpoint. Eventually and with much effort he provided some historical evidence of symptoms, albeit not necessary consistent with MS. Interesting the account I received was similar to that provided to Dr Williamson at UCLA (his history is provided below), so I have appended his history as well. Narendra informed me that his symptoms began in 2009 when he noticed "pale vision" and gradual constriction of temporal fields which progressed over several years. He then showed me Humphrey Perimeter tests with severely constricted fields (tunnel vision).</p>
<p><b>3</b></p> <p><b>9.21.2018-Dr. Philip Revere Kinkle Summary Original Text</b></p> <p>He became argumentative when I informed him that the Humphrey perimetry results were not consistent with an optic neuropathy or MS (more consistent with glaucoma). From here the history became disjointed and consisted mostly of multi-focal numbness and paresthesias with gait problems developing at some point in time. I left the room at this point to review epic records and found the following note by Dr Williamson. I was able to confirm much of Dr Williamson's history when I subsequently returned to talk with Narendra further. History provided to Dr Williamson at UCLA (9/19/18) (Copied from his note verbatim) 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.</p>	<p><b>Statement by Statement Negation</b></p> <p>The Humphrey tests shows constricted visual fields but the VEP and analysis of the optic disk are more for the diagnosis and treatment of MS. The doctor comments on both during the appointment.</p> <p>I gave the full MRI of the brain from 2008, the brain from 2012, the cervical spine and the lumbar spine.</p>	<p><b>4</b></p> <p><b>9.21.2018-Dr. Philip Revere Kinkle Summary Original Text</b></p> <p>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 'blown 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.</p> <p>I stated that the peripheral vision was restricted, identical to the Humphrey test. Not the central vision.</p> <p>There are copies of it in the system, brain cervical and lumbar.</p>

5

9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

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 'occurred 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.

Statement by Statement Negation

Methylprednisolone for steroids show an improvement in the FDG pet but they don't make the FDG pets normal. Its simply a positive response from a medication that indicate the underlying disease process.

6

9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

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.

Statement by Statement Negation

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These are reiterations from Dr. Williams.

Its consistent with the focal points of seizures in epilepsy.

People with MS usually have pseudobulbar effects when antiepileptics are given due to withdrawal effects. Which is why they aren't typically prescribed antiepileptics.

7

9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

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 withdraws 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.

Statement by Statement Negation

He is reiterating Dr. William's report.

8

9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

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.

Statement by Statement Negation

Optic neuropathy, neurodegeneration of the cervical spinal column, and occasional immobility that responds to methylprednisolone are consistent with MS.

Dr. Williams stated that "give away" means pretending. But its apparent and clear from MRIs that I wouldn't have the capability to move considering the lesions over the MRIs.

9

9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

Next says because symptoms got worse he went [back?] to mexico city where they gave him 5 days of methylprednisolone 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.

Statement by Statement Negation

1.5 to 2 months.

The brain MRI shows T2 intensities along the posterior brain, corpus callosum, and mild features of Dawson fingers. Which is typical for patients with MS.

The cost of the medications is the objective reason for the appointment, at a cost of 2.5 k USD per month it could only be covered under insurance.

That improvement indicated optic neuropathy.

10

9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

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. ER visits. Says he had another visit to east asia where FOG-PET was repeated 4.2018 - reportedly they dosed methylprednisolone 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.

Statement by Statement Negation

Most of the MRIs are repetitions that show similar features and progressive atrophy of the spinal column.

I show the full MRI series.

The visual evoked potential test is typical of optic neuropathy.

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9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

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.

Examination:  
BP 123/76 (BP Location: Left arm, BP Patient Position: Sitting, BP cuff size: Regular) | Pulse 95 | Temp 98.2 °F (36.8 °C) (Oral) | Ht 5' 6" (1.676 m) | Wt 52.2 kg (115 lb) | SpO2 98% | BMI 18.56 kg/m2

Statement by Statement Negation

Plasmapheresis is typically effective immediately due to exchanging out the entire white blood system.

They were medication trials but the mainstay drug was interferon (Rebif). Cyclophosphamide isn't used in medicine to treat MS.

The "feigned weakness" is shown to be progressive MS, causing neurodegeneration of both the cervical spine and worsening of optic neuropathy.

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9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

General: Examination of the skin, joints and extremities revealed no abnormalities ; There were no cervical, ocular or cranial bruits.

Cognitive/Behavioral: Examination of cognition, language and prosody revealed no abnormalities . Behavioral and affect was appropriate.

Cranial Nerves: Near visual acuity was J 1 + OD and J5 OS with glasses for correction. 10 /10 color plates were identified OD and 3/10 OS. Visual fields were full to confrontation testing. Lids were normal . Pupils were 7mm and reactive with no RAPD . Fundoscopic exam revealed definite optic nerve pallor OS> OD and large optic cups. Eyes were orthotropic . Pursuit and saccadic eye movements revealed no abnormalities . Facial movements revealed no abnormalities. Testing of facial sensation revealed no abnormalities. Hearing was normal AD and normal AS. Bulbar examination revealed no abnormalities .

Statement by Statement Negation

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Motor: Marked giveaway weakness on the left side with normal tone and no atrophy. Resistive strength in all Limbs

Cerebellar: Very slow FTN with dysmetria on the left (see gait for description of midline/axial cerebellum dysfunction)

Sensory: Sensation to pp and temperature was NT. Vibration sensation duration (secs) was (R/L): Upper extremity middle finger ND/ND; Lower extremity big toe NT/NT.

Joint position sensation was normal . Stereognosis was normal Reflexes : (R/L): Biceps 0/0, BR 2-/2-, Triceps 2-/2-, Patella 2/2, Ankle 0/0. There was no clonus . The right toe was downgoing and the left toe downgoing on plantar stimulation

Gait Description: Slow hesitant narrow based gait with slight LLE lag and knee hyperextension

Statement by Statement Negation

The "giveaway weakness" is determined to be caused by spinal atrophy.

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Review of Imaging Studies:

1. MRI of brain 12/18/2008 was reviewed: Normal
2. MRI of brain 10/27/12 was reviewed: normal except vague T2 hyperintensity posterior PVWM of no significance
3. MRI of the cervical spine 1/10/17 was reviewed : no intramedullary abnormalities
4. MRI of thoracic spine off of his computer only (not allowing me to handle the controls) may show a mid thoracic T2 hyperintensity on sagittal images but not able to determine if central or how longitudinally extensive

Non Imaging Studies:

1. VEP 8/10/18 P100 118 OS and 105 OD
2. Humphrey Perimetry results with severe constricted fields

Review of Labs:

1. CSF reportedly normal
- Assessment:  
Very difficult to assess this young man; He has done some extensive doctor shopping all over the world (Europe, India, Asia, USA, Mexico, South America) and is convinced of his diagnosis and needs.

Statement by Statement Negation

The 2008 MRI shows a very large lesion approximately 4 square centimeters big.

The cervical spine shows a gross pathology.

A optic test is included with the Humphrey test. He mentions it in the appointment.

CSF is never mentioned.

The condition worsens and is determined to be accurate and secondary progressive MS is determined to be caused by a lack of appropriate medications.

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9.21.2018-Dr. Philip Revere Kinkle Summary Original Text

I am very concerned that the visual symptoms and visual evaluations are more suggestive of glaucoma or other process. Certainly his Humphrey Perimetry results are not consistent with an optic neuropathy.

At the end of the visit I told him I would be willing to see him again and work with him only if he follows through on my recommendations listed below.

After discussing the options available and the potential benefits (pros) and risks (cons), we mutually decided on the following management plan

Plan/contract:

If you are willing to upload all your films , get blood drawn today , see ophthalmology, and get standard CSF analysis (which you are going to do outside this country) we will consider prescribing medications if the evaluation is consistent with MS or another inflammatory disorder. Otherwise we will not see you again

Total time spent by Dr Kinkle I with the patient today was 70 minutes with over 50% of the time spent on education and counseling regarding their problem list and the assessment and plan .

Statement by Statement Negation

Glaucoma doesn't present at age 33 and optic neuropathy typically occurs with reduced visual fields.

The essential MRIs were already added.

The statement by statement negation goes into detail as to how Dr. Kinkel's appointment is an exemplification of trying to circumvent medical diagnostics to try and avoid giving the medications. The difficult part is that even with a severe course of clinical events the medical statements in the US have largely remained unchanged from 2015, in Dr. Sloan's appointment (its almost complete medical ignorance since). Since then there was gross and easy to demonstrate clinical fraud.

This type of medical negligence when the patient has clear MS with all the evidence to show the condition is non trivial and the clinical fraud makes it criminal.