Exhibit 8

From: Padmanabhan, Bharanidharan
Sent: Wednesday, July 14, 2010 1:48 PM
To: Hulka, Carol
Subject: just like Tuskegee

Dear Dr Hulka,
Greetings!
In the case of [redacted]'s MRI I asked you point blank if radiologists in your department actually viewed the images

before generating an official report and billing insurance.
You assured me that every scan is viewed before a report is generated.
It is impossible to believe that now.

Naturally this has implications not only for the lives of patients but also in terms of insurance fraud.

Here is the official report on Mr [redacted].
My dictated note is pasted below.

Department of Diagnostic Radiology
PATIENT: [redacted]
DOB: [redacted]
AGE: 64
SEX: M
ACCT#: [redacted]
LOCATION: WHMRI
UNIT#: [redacted]
STATUS: REG
REF ORD PHY: MARIE O GIRAULT NP
Exam Date: 06/23/10
Exam Status: Signed

Exam: MRI BRAIN NON CONTRAST
Reason for Exam: Psychotic\H\
Indication: Psychotic
Comparison: None available
Technique: MR images of the brain were obtained according to standard department protocol without contrast. No IV contrast was used.

Findings: There is **mild T2 hyperintensity** in the periventricular white matter **which is nonspecific**, but **probably related to chronic small vessel ischemic disease**. There is a small oval-shaped old lacunar infarct in the periventricular white matter adjacent to the right frontal horn and a tiny old lacunar infarct in the left centrum semiovale. No evidence of intracranial hemorrhage, mass-effect or midline shift. There is no diffusion restriction to suggest infarct. The ventricles are normal in size. There is mild mucosal thickening of the left maxillary sinus. The calvarium and mastoids are unremarkable. The soft tissues are normal.

Impression: No acute intracranial abnormality. Likely chronic small vessel ischemic disease with tiny old lacunar infarcts as described above.

Dictated By: ARTHUR C. CHANG MD
Reviewed and Electronically Signed By: ARTHUR C. CHANG, MD

If this patient had not been sent to see me, someone clearly obsessed with actually viewing MR images,
there is no denying that this official CHA Radiology report would have condemned a man
to a life of increasing disability, loss of independence and early death
for an eminently treatable disease !!!!!!!

Who says Tuskegee does not happen everyday?

And no, I am not trying to make myself seem a saviour.
This is what I come across **repeatedly** and have brought to your attention before.

With regards,
Bharani

07/13/2010

Marie Olene Girault, NP
Cambridge Health Alliance - Windsor Street Health Center
119 Windsor Street
Cambridge, MA 02139

RE: [redacted]

Dear Nurse Girault:

I had the pleasure of seeing your pleasant patient, Mr[redacted], at the MS Clinic at the Whidden this morning. Mr. [redacted] is brought here by his sister, [redacted], who reports 5 years of increasing memory loss.

The patient had been living in Fort Lauderdale for some years. He was married, had a wife and 3 children. He is educated and worked initially as a Customs officer in his native [redacted] and as a policeman here. He was last seen by his sister and brother here in Massachusetts back in 2003. Since then, all contact had been lost until 12/2009.

It turns out the family have just learned that Mr. [redacted] was found to have worsening problems with maintaining his job. He had

difficulty completing his tasks. He was let go. He could not pay his rent. His wife threw him out and he became homeless.

He lived on the streets of Fort Lauderdale for about a year and a half. He lost his insurance. He had dveloped bladder frequency and urgency and had to void every 90 minutes. He had been diagnosed with prostate hypertrophy at that time and had religiously been taking hs prostate medicine. Once he lost his insurance that also was stopped. He ended up in a homeless shelter and the social worker took the trouble to find his kith and kin. He is now living in Massachusetts with his mother and 2 siblings who are taking care of him.

Mr [redacted] is also able to report these today. He states that about 5 years ago is when he started feeling foggy. It took him a lot longer to do the things that he had been used to doing both at work and in terms of activities of daily living. He also started having bladder urgency and frequency which was chalked up to prostate hypertrophy. He still goes to the bathroom to empty his bladder every hour and a half. When he gets up in the morning, he has to immediately rush into the bathroom. He has been given a bottle to be used by the bedside if he cannot make it. He does end up using it most days. He tells me, however, that he has never had incontinence. He denies constipation or diarrhea.

His balance has been off for about 6 months. He feels his balance is off from within. He states that no one has ever told him that he walks like a drunk.

He does complain of fatigue. Apparently he has had this fatigue for about 2 years. Many days he cannot get out of his own way because of the fatigue. He wishes that something could be done to make the fatigue go away. He is quite sure the fatigue has no relation to any physical effort the day before. He denies trouble

with his vision. He wears glasses and tells me he can see well through them. His family had just gotten him a new prescription and he is happy with it. He denies any significant numbness or tingling or burning in his arms or his feet or anywhere else.

He used to get migraine with aura with flashing lights, bitemporal throbbing, nausea and polka dots. It has been about 4 years since the last aura episode with pounding headache he says. He is very glad that they seem to have gone away for the moment. He still gets a headache a month for which he takes excedrin.

He does note that he has been unable to maintain his current account, pay his taxes, or even think about simple things like reconciling his passbook for some years. He has been slowing down he feels intellectually. Most days he states between the fatigue and the brain fog, he feels unable to do anything beyond the simplest of things. He has also been absolutely petrified that he is coming down with a dementia. He says many people told him along the way that he may have Alzheimers disease or similar dementia and that makes him extremely perturbed, but he had eventually come around to accepting that he did indeed have Alzheimer's dementia and that he was simply going to worsen quietly over time.

In the meantime, he has been diagnosed with atrial fibrillation, and he is now on Coumadin since moving to Massachusetts. He has also been receiving excellent care at Cambridge Health Alliance and so his family was able to arrange for him to come to the neurology clinic today. His sister has brought him in today and filled in some of the biographical details regarding Florida, but once the patient got going he was able to give a very cogent and chronologically accurate history about his life. When he walked in, he literally shuffled in, downcast, looking hopelessly withdrawn,

but as the visit proceeded, he became brighter, smiled more, and participated fully in the discussion and the neurological exam.

FAMILY HISTORY: Apparently is positive for migraine with aura in numerous members as well as atrial fibrillation in both of his brothers. There is no family history of dementia at all. His mother is 80 years old and is still cognitively intact according to his family.

SOCIAL HISTORY: Mr. [redacted] used to work as a customs officer in [redacted] before moving to the U.S. In Florida, he worked for many years as a law enforcement officer. He steadfastly denies ever breaking the law in any aspect including illicit substances. He never smoked. He does not drink. He was never an alcoholic. He did not even sniff glue. He tells me he was faithful to his wife throughout their period of marriage. He is a lifelong Baptist.

REVIEW OF SYSTEMS: As above.

On examination today, Mr [redacted]'s blood pressure is 116/52, heart rate is 85, respiratory rate is 18, weight is 74.8 kilograms, and height is 1.68 meters. His O2 sat is 98%.

He is well groomed and social graces are preserved. On mental status testing, he is awake, alert, oriented fully and able to participate well with mental status testing. He has preserved language comprehension, abstract reasoning etc. He could recall 2 out of 3 at 5 minutes.

His cranial nerve exam shows no loss of smell. Cranial 2 is normal; 3, 4, and 6 show a right INO with saccadic breakdown to gaze in all directions. There is no Graefe sign. Cranial nerve 5 is normal. Cranial nerve 7 is normal; 8 is normal; 9, 10 and 11 are also normal. He does have a gag still; 12 is normal too. His

pupils are about 2 mm. There is no afferent pupillary defect on exam today.

His motor exam shows normal tone and bulk. Power was 5 throughout except for finger extension in has right hand, which was at 5-, and hip flexion in both legs at 5-.

Coordination was somewhat off in both hands. Playing patty-cake for example showed after some time decreased rate of opening of the fingers on his right hand compared to the left.

Looking for pronator drift, he has marked right palm cupping compared to the left; however, he also appears to have a bit more of flexion in his right ring finger and little finger, suggesting he may have chronic ulnar neuropathy. At 60 seconds, however, a mild drift was observed in his left arm.

Finger tapping is off on the right compared to the left. Hand clapping was off on the left compared to the right in terms of rhythm, denoting some mild cerebellar dysfunction. Finger-to-nose, however, essentially okay re targeting but I thought his left arm was slightly off by about 0.5 cm. Heel-to-shin again shows that his left is slightly off.

He was able to stand up from a chair without using his arms. When standing with feet together and eyes closed, there is a significant increase in body sway. He retropulses and also sways more towards his left side. There was, however, no complete Romberg.

His gait is slow. He employs multi-step turns. He was barely able to complete tandem gait, but with practice was able to do so. He can walk on his toes and on his heels.

Reflexes are 2 for the right biceps, 2+ for the left, 2 for the right brachioradialis, 2+ for the left, 3 for the right patellar, 3+ for the left, and 2 for both ankles. Both toes were mute.

I have gone over his MRI with him and his sister frame by frame.

Contrary to the official radiology report, his MRI is highly abnormal.

Specifically going through the all-important Sagittal FLAIR sequence, the first thing that jumps out is the high degree of atrophy that he has already at this age.

Mr [redacted] also has numerous widespread bright hyperintense T2 lesions both periventricularly and juxtacortically. The periventricular hyperintensities start at slice #6 on series 9. Slice #7 shows lesions in the white matter; 1 in the deep white matter, 1 in the right occipital junction, and 1 fairly large lesion smack in the middle of his frontal lobe. He has a very large Dawson's finger/ conical lesion with evident demyelination in the middle at slice #9 coming off the atrium of his lateral ventricle along with juxtacortical lesion. Slice #10 shows an almost 2 cm long Dawson's finger stretching up into the cortex in the classic radial pattern, along with numerous other lesions and other slices; all of which are distributed in a very highly specific pattern. He clearly has T2 bright lesions directly involving his U-fibres.

This is seen in both hemispheres. For example, slice #20 on series 9 shows a very large, very bright hyperintense T2 lesion in the deep white matter, which actually continues on through 5 separate slices.

On the sagittal T1 scan the demyelination around the atrium of the ventricle is apparent, along with severe thinning of the corpus

callosum, especially along the middle third, which naturally fits with the severe atrophy that is also evident in the whole brain.

The lesion distribution pattern and appearance is very highly specific, unmistakable and consistent only with a diagnosis of multiple sclerosis.

The literature is very clear that this degree of involvement of the corpus callosum, long Dawson's fingers, heavy involvement of U-fibres and clear T1 signal loss in the middle of an inflammatory T2 lesion is pathognomonic for multiple sclerosis and not for nonspecific minimal small vessel disease.

It also has been shown to not bode well for the patient in the future in terms of disability.

Even on the Axial T2 FLAIRs, series 5 slice #15 for example, the involvement of the anterior corpus callosum is clearly evident.

IMPRESSION: Mr. [redacted] is a pleasant 64-year-old right-handed gentleman who first started having difficulty more than five years ago with keeping his life together with multitasking and with activities of daily living due to fatigue and brain fog. He also had bladder frequency and urgency. He was not diagnosed with any neurological disease at that time.

His family threw him out without a diagnosis.

He ended up homeless and lived on the streets of Fort Lauderdale. He denies ever being worked up for a neurological disorder throughout that period. He received an MRI on 06/23/10 thanks to his new care providers here at Cambridge Health Alliance who also carefully ordered a Sagittal FLAIR sequence to go with the rest of his MRI scan.

Based on his history, his exam and his very highly specific MRI, I am forced to conclude that this unfortunate gentleman most likely has multiple sclerosis rather much rarer entities such as Chronic Lyme disease, cerebral angiitis, lupus cerebritis, Hashimoto encephalopathy, Sjogren syndrome, or even the oft-cited mild nonspecific small vessel disease.

I am also convinced he has had multiple sclerosis from the time of his first symptoms at least 5 years ago.

Mr. [redacted] burst into tears upon being given this diagnosis. While having a cry however, he sat upright, perked up, a large smile broke out on his face and he said, "It's treatable, isn't it Doc?" I assured him this was a treatable condition and that he was not going to slowly rot.

He agreed to an infusion of 1 gram of Solu-Medrol right away today, which he has received. Prior to infusion, I have explained the potential risk of aseptic or avascular necrosis of the hip or the knee to both him and his sister. He agreed to the Solu-Medrol nonetheless. I have sent him and his sister home with the kits to go through and books to read regarding multiple sclerosis. He and his family will go through them together and he shall return here in 2 weeks time, an informed patient better able to make decisions regarding the safeguarding of his future.

I thank you once again for the kindness of this referral and I am immensely grateful that the Sagittal FLAIR sequence was acquired at the time of his initial MRI brain scan. Naturally I shall keep you fully updated regarding any decisions we arrive at in the future and any changes to his neurological state.

Yours most sincerely,
Bharani Padmanabhan MD PhD