October 13th 2015

I am an Associate Professor in the Department of Emergency Medicine, University of Ottawa. I have expertise in Emergency Medicine and hold a certificate of special competency with the College of Family Practice and current certification with the American Board of Emergency Medicine. I have been in independent medical practice for 34 years. From 1985-2006 I practiced emergency medicine full time at St. Paul's Hospital in Vancouver. From 2007 until the present I have been practicing 2/3 time at the Ottawa Civic and General Hospitals. My highest academic rank is as an Associate Professor with the University of British Columbia and the University of Ottawa. I have been actively involved in teaching medical students, residents, nurses, paramedics, and practicing physicians since 1985. Through my education, training, knowledge of medical literature relevant to my specialty and other professional activities I am familiar with the standard of care as it currently exists in Canadian teaching and community hospitals and as it existed in January of 2014. Details of my training, work experience, and academic achievements are documented in my attached CV.

I have been requested to review the medical file of Mr. AS to address the adequacy of the medical care provided from January 21st 2014 through April 11th 2014. It is my intention to render an opinion on the standard of care, particularly as it relates to Mr. AS's emergency care on January 21st 2014. My conclusion is that the standard of care was not met by Dr. RB. My opinions are based on review of relevant medical records, my education and training, my knowledge of the relevant literature, and my experience and expertise in emergency medicine.

I have been provided with medical records from Hospitals A, B, C, D and from Dr. AH, a family physician. I will first provide a summary of medical care received from January 21st 2014 through January 24th 2014.

At the time of his stroke, Mr. AS was 43 years old. His past history was significant for hypertension, diabetes, hypercholesterolemia, and tobacco dependence. His medications included sitagliptin, bisoprolol, atorvastatin, perindopril, and nifedipine as prescribed by Dr. AH. These medical conditions were longstanding. There was no previous history of a stroke or myocardial infarction.

Late in the morning of January 21st 2014 Mr. AS experienced the sudden onset of a headache, severe dizziness, vomiting, pallor, diaphoresis, and an inability to ambulate independently. His co workers had to carry him out of his workplace. He arrived at Hospital's A's emergency department triage desk at 1310 hours and was assessed by Nurse TT. She documented his chief complaint as "sudden dizziness and headache while working today persists on arrival, history of niddm and htn, no cardiac history, alert and orientated, breathing well, pale. Pulse 57, Systolic BP 210 (no diastolic recorded), resps 18, O2 sat 100%." He was placed in a bed at approximately 1430 hours and assessed by another nurse who recorded dizziness with headache and vomiting as his chief complaint. Repeat blood pressure was 185/95. Brief physical exam documents him as alert and orientated with no weakness in either arms or legs.

He was seen by the emergency physician, Dr. RB, at 1510 hours. Dr. RB's dictated note records the chief complaint as acute vertigo with weakness and vomiting. Pertinent information from his history includes: " ….suddenly became acutely vertiginous……vertigo continued for about an hour…..no headache and no neck pain……intermittent vomiting…….when he stays still symptoms are less……no paralysis, no loss of sensation, no confusion, no difficulty with speech." Relevant physical exam findings were recorded as: "able to move arms and legs symmetrically, reflexes equal, toes downgoing,…..no spontaneous nystagmus, on gaze to the right there was approximately 5-7 seconds of nystagmus with a rapid component to the right, on left gaze no nystagmus, on gaze straight ahead I was unable to detect any nystagmus. I attempted a Dix Hallpike maneuver, at the end of the maneuver he began vomiting uncontrollably and complained of vertigo. I re -examined his eyes at that time and there may have been a hint of nystagmus…. differential diagnosis includes benign positional vertigo, vestibulopathy and possibly the beginning of labyrinthitis." Additional comments: " this man was able to stand but was extraordinarily unsteady and tended to fall back……there is no hearing loss or fullness this makes Meniere's disease less likely."

Initial investigations consisted of routine blood work, an ECG, a chest x ray, and a non contrast head CT. The head CT was read as normal by Dr. RB and this was confirmed by a formal radiology report issued the following day. Blood work was non contributory. Treatment was initiated with IV fluids and an intravenous dose of an antiemetic. Dr. RB reassessed the patient at 2042 hours and noted…."the patient appears better, he was not pale, he was able to stand but slightly unsteady and he complained of slight vertigo when he stood. Examination of his eyes continued to reveal nystagmus with a fast gaze on looking right, on looking to the left there was no nystagmus. The patient most likely has developed labyrinthitis and not benign positional vertigo, this is based on the persistence of nystagmus." At that time he received more intravenous fluid, another dose of an antiemetic, and a dose of metformin to lower his blood sugar. At 0015 hours January 22nd Dr. RB reassessed Mr. AS for a final time noting "…..feeling somewhat better, complexion normal, he has a sense of ongoing vertigo with movement his gait was better but certainly there was a hint of unsteadiness. This patient has almost certainly developed labyrinthitis, I do not see any evidence of brainstem infarct or Horner's syndrome." Mr. AS was advised to follow up with his family physician, the Ear Nose and Throat Clinic and to take dimenhydrinate (Gravol) for nausea. He was then discharged from the emergency department and he returned home. His condition for the remainder of January 22nd is unknown to me.

Sometime on January 23rd Mr AS was seen by his family physician, Dr. AH. He notes "patient at hospital 2 days ago nausea vomiting, CT scan normal, no heart attack, today looks sick, not in distress, good air entry, heart sounds normal…." There is absolutely no documentation of a neurological examination. He was noted to have a blood pressure of 210/120 and was given a nitroglycerin patch to lower his blood pressure. He then waited in the waiting room and I surmise he was discharged when his diastolic pressure had dropped below 120 mm Hg. Shortly after returning home he developed trouble swallowing, difficulty breathing, blurred vision, and marked unsteadiness. He was transported to Hospital B where he was rapidly assessed and transferred to Hospital C as a possible stroke code. His initial CT scan at Hospital C showed an infarct of the medulla, upper pons and a portion of the upper spinal cord. A CT angiogram identified a left vertebral artery (V4) occlusion secondary to a dissection. In addition, there was a focal right superior cerebellar artery narrowing possibly related to thrombus. Thrombolytics were not administered as his symptoms had started 60 hours earlier, well outside of the

4.5 hour window of treatment. Secondly, the presence of a dissection is a contraindication to thrombolysis. Endovascular intervention was deemed inappropriate due to the risk of pushing clot proximally into the basilar artery. He was therefore managed medically with intravenous heparin and medical treatment for his diabetes, hypertension, and hypercholesterolemia. He was repatriated to Hospital B January 27th and subsequently to Hospital D for rehabilitation. While in Hospital B he may have suffered another 2 small strokes related to temporary discontinuation of his anticoagulation prior to surgical procedures. It appears he was discharged home on April 11th 2014. At the time of discharge his deficits included residual dysarthria, dysphagia, left face and right arm/leg numbness and left upper limb dysmetria as well as an ataxic gait. I gather these deficits have remained stable since discharge from Hospital D.

In reviewing this case there are several important issues that need to be considered: the adequacy of the initial history and physical examination, the appropriateness and interpretation of the investigations, the need for further investigations and/or consultations, the possible outcome if the diagnosis had been made earlier and treatment initiated, and lastly whether performing a HINTS exam was a standard of care in a teaching hospital in January 2014.

Dizziness is a common presenting complaint for emergency patients comprising between 3-5% of emergency visits. It is rare to work a shift and not see a patient whose chief complaint is dizziness. Given the frequency of this symptom most emergency physicians have a fairly structured approach in their evaluation of these patients. When dizziness develops suddenly and is associated with vomiting, gait unsteadiness, intolerance to head motion, and sustained nystagmus, either at rest or gaze provoked, then this is considered to be an acute vestibular syndrome. Approximately 20% of patients presenting with dizziness will have this syndrome. Certainly Mr. AS fulfilled all of these criteria. From a diagnostic perspective the causes of this syndrome are numerous and can be considered in two broad categories: peripheral or central. Common causes of peripheral vertigo are: labyrinthitis, Meniere's disease, exposure to drugs such as aminoglycosides, anticonvulsants, or alcohol and traumatic inner ear injury (labyrinth concussion). Common central causes include: brain stem infarction, cerebellar infarction or hemorrhage, multiple sclerosis, and vertebral basilar migraine. From a clinical perspective it is of utmost importance to differentiate between central and peripheral causes. The majority of peripheral causes are clinically benign and self limited. However suspicion of a central cause, particularly cerebellar infarction/hemorrhage and brain stem infarction, usually requires consultation, advanced imaging (diffusion weighted MRI), admission, and goal directed treatment. Patients with brainstem infarctions are at risk for progression of stroke, acute hydrocephalus, and herniation secondary to cerebellar edema. In some instances neurosurgical interventions are needed to deal with complications. It is estimated that of patients presenting to an emergency department with an acute vestibular syndrome the incidence of stroke is approximately 20-25%. In some case series of patients referred to a neurology service with a chief complaint of vertigo the incidence of stroke approaches 60%. The obvious clinical challenge is to differentiate between peripheral causes and the much more sinister central causes.

Adequacy of Initial History and Physical Examination

Given that many of the cranial nerves have their origins in the brainstem then it is important to ask questions relative to their functions. These questions usually include double vision, facial

numbness, facial weakness, alterations in voice, difficulty swallowing, and sudden changes in hearing, either tinnitus or deafness. In addition it is useful to ask questions directed to cerebellar function such as unsteadiness, falling in a consistent direction and coordination of limbs. I can see no evidence from the medical record that these questions were asked. Physical examination of these patients should include a detailed neurological examination addressing cranial nerve, motor, sensory, and cerebellar function. Dr. RB does comment on the presence of nystagmus but not on any other cranial nerve examination such as facial asymmetry, facial sensation, hearing, palatal function, assessing or commenting on swallowing and phonation and lastly, tongue deviation. The motor examination consists of "moving arms and legs symmetrically". While this is acceptable as a gross measure of motor function it will certainly not elicit a subtle hemiparesis, as a test for pronator drift would have done. The majority of emergency physicians do not perform a routine sensory examination unless the patient is complaining of numbness in a particular area. With some brainstem strokes ipsilateral reduction of facial pain perception and contralateral pain and temperature loss can be prominent features as, ultimately, was the case with Mr. AS. In terms of cerebellar function Dr. RB did observe Mr. AS as he attempted to walk and he was noted to be very unsteady and tended to fall backwards. There is no evidence of any formal cerebellar examination such as past pointing, dysdiadochokinesia , truncal ataxia, or limb dysmetria. Focal neurological signs, albeit subtle, are present in about 80% of patients with brainstem strokes. I cannot state definitively whether these signs were present or not based on the information included in the medical record.

It is readily apparent from the medical record that Dr. RB was alive to the possibility of a central lesion as evidenced by his final assessment of Mr. AS: "I do not see any evidence of a brainstem infarct or Horner's Syndrome". While being cognizant of this possibility, it is unfortunate that he did not direct his history or physical examination in such a way as to make a central cause of the vertigo less likely. In addition, the patient's vascular risk profile was extremely high which should have placed stroke higher on the list of diagnostic possibilities.

Investigations

The investigations ordered were appropriate. The only significant abnormality on his bloodwork was an elevation in his blood sugar without evidence of an acidosis or a hyperosmolar state. This abnormality was certainly not a cause for his acute vestibular syndrome. Some physicians would not have ordered a CT scan but I think the majority would order a CT if available, particularly in the context of headache and vertigo. Dr. RB correctly interpreted the CT scan as being normal, as did the radiologist the following day. Unfortunately, a negative CT scan in this particular clinical scenario does not rule out cerebellar infarction or brainstem stroke. CT will be positive in only 15-20% of these patients when performed early in their illness. From a clinician's perspective this means that a normal CT in a patient with an acute vestibular syndrome will miss as many as 85% of patients with a stroke as the etiology for their disorder. Diffusion weighted MRI is a much more sensitive test and is regarded as a gold standard in imaging. However, even MRI will miss 5-10% of brainstem and cerebellar strokes when performed early in the clinical course.

Need for Further Investigations or Consultation

At the time of Mr. AS's discharge Dr. RB was quite definite that "this patient has almost certainly developed labyrinthitis". In his mind, neither consultation nor further imaging were necessary as the cause of the vertigo had been established as peripheral, not central. There is a point of interest to be raised here, that of whether or not a headache was present. The two nurses who initially assessed Mr. AS both noted headache and dizziness as the chief complaint whereas Dr. AS specifically states "no headache" in his initial assessment. The radiology report states specifically "no findings to explain headache". This implies that whoever filled out the requisition for the CT listed headache as one of the reasons for the CT. I am unsure from the medical record as to whether it was Dr. RB or a nurse who completed the requisition. If the former, then his reason for CT and his clinical history are at odds. There are many reasons why a history taken by two individuals may be contradictory including how the question is posed, comprehension on the part of the patient either cognitively or language related, and the clinician's understanding of the answer. If a severe headache was part of the clinical presentation it would raise several diagnostic possibilities such as a cerebellar hemorrhage (excluded by CT), vertebral artery dissection, vertebral basilar migraine (a diagnosis of exclusion and unlikely without a prior history of migraines), isolated labyrinthine infarction (rare) and lateral medullary infarction which can present with severe facial pain. If headache was deemed to be a significant feature then many clinicians would proceed to a CT angiogram to rule out vertebral artery dissection. If CTA was unavailable then either neurology referral or transfer for imaging would have been appropriate.

Outcome if Diagnosis Had Been Made Earlier

If the diagnosis of vertebral artery dissection or medullary infarction had been made at the time of his initial emergency encounter then Mr. AS would likely have been transferred to Hospital C for assessment and treatment, both medical and possibly surgical. Standard medical therapy consists of anticoagulation with heparin and medical management of blood pressure, hypercholesterolemia, and diabetes. To my knowledge anticoagulation has never been studied prospectively in the context of carotid or vertebral artery dissection. Despite this, it remains the current standard of care. I cannot state definitively that early initiation of anticoagulation and blood pressure management would have either prevented his stroke from progressing or reduced the severity of the stroke if it did occur. A neurologist or neurosurgeon would be capable of knowledgeably commenting on this issue. What I can state definitively is that there was a potentially avoidable 60 hour delay between symptom onset and diagnosis. During that time Mr. AS did not have access to specific treatment.

HINTS (Head Impulse, Nystagmus, and Test of Skew) Testing and Standard of Care

The HINTS examination was first described in an article in the journal Stroke in 2009. The abstract is as follows:

HINTS to Diagnose Stroke in the Acute Vestibular Syndrome

Three-Step Bedside Oculomotor Examination More Sensitive Than Early MRI Diffusion-Weighted Imaging

Jorge C. Kattah, MD; Arun V. Talkad, MD; David Z. Wang, DO;
Yu-Hsiang Hsieh, PhD, MS; David E. Newman-Toker, MD, PhD

Background and Purpose—Acute vestibular syndrome (AVS) is often due to vestibular neuritis but can result from vertebrobasilar strokes. Misdiagnosis of posterior fossa infarcts in emergency care settings is frequent. Bedside oculomotor findings may reliably identify stroke in AVS, but prospective studies have been lacking.

Methods—The authors conducted a prospective, cross-sectional study at an academic hospital. Consecutive patients with AVS (vertigo, nystagmus, nausea/vomiting, head-motion intolerance, unsteady gait) with ≥1 stroke risk factor underwent structured examination, including horizontal head impulse test of vestibulo-ocular reflex function, observation of nystagmus in different gaze positions, and prism cross-cover test of ocular alignment. All underwent neuroimaging and admission (generally <72 hours after symptom onset). Strokes were diagnosed by MRI or CT. Peripheral lesions were diagnosed by normal MRI and clinical follow-up.

Results—One hundred one high-risk patients with AVS included 25 peripheral and 76 central lesions (69 ischemic strokes, 4 hemorrhages, 3 other). The presence of normal horizontal head impulse test, direction-changing nystagmus in eccentric gaze, or skew deviation (vertical ocular misalignment) was 100% sensitive and 96% specific for stroke. Skew was present in 17% and associated with brainstem lesions (4% peripheral, 4% pure cerebellar, 30% brainstem involvement; χ^2, P=0.003). Skew correctly predicted lateral pontine stroke in 2 of 3 cases in which an abnormal horizontal head impulse test erroneously suggested peripheral localization. Initial MRI diffusion-weighted imaging was falsely negative in 12% (all <48 hours after symptom onset).

Conclusions—Skew predicts brainstem involvement in AVS and can identify stroke when an abnormal horizontal head impulse test falsely suggests a peripheral lesion. A 3-step bedside oculomotor examination (HINTS: Head-Impulse—Nystagmus—Test-of-Skew) appears more sensitive for stroke than early MRI in AVS. (***Stroke*. 2009;40:3504-3510.**)

This article clearly states that the performance of three fairly simple physical tests can accurately differentiate between peripheral and central causes of vertigo in patients with an acute vestibular syndrome. In this case series HINTS testing was more sensitive than a diffusion weighted MRI performed at the time of initial presentation. The question then becomes how soon after the publication of a potentially practice changing study should physicians be expected to change their practice? This is a difficult question to answer as there are many variables. For most physicians CME is a composite of high quality journals relevant to the specialty, attendance at conferences and other CME activities, accessing online resources, participating and attending academic rounds, engagement with residents and medical students, and sharing knowledge with colleagues and consultants. Another rate limiting factor is that new knowledge often requires that one change one's clinical practice. In some instances, this knowledge will have both a cognitive and motor component. The HINTS exam is a perfect example. It is not adequate to be aware of the study, a clinician must also learn the appropriate physical examination techniques and incorporate them into practice when evaluating a patient with an acute vestibular syndrome.

I personally was not aware of the HINTS study and technique until several years after its publication. Certainly by 2011 the article was being discussed on various emergency medicine websites such as EM-RAP. I also recall that by 2011 HINTS was being discussed at some emergency medicine conferences. To my memory most emergency residents and academic emergency physicians were aware of HINTS testing by 2012 and had incorporated it into their practice. Certainly in the setting of a community hospital the uptake may be slower. Given that Dr. RB was working in an academic center and had frequent contacts with residents, other attendings, and consultants I would be surprised if he was not aware of the HINTS exam in January of 2014. I have included a list of references at the end of this report. The article from the British Medical Journal was published in 2009, the CMAJ review article was published in 2011, and the last edition of Rosen's textbook was published in 2013. Obviously, all predate the date of occurrence by several years. Each of these publications discuss the HINTS exam and its importance in evaluating dizzy patients. All things considered, I would say that the performance of the HINTS exam was the standard of care in January 2014. If Dr. RB had performed HINTS testing on Mr. AS and it was consistent with either a central or peripheral cause then I would not be writing this report.

Dr. RB provided compassionate and well intentioned care as evidenced by his frequent reassessments. Unfortunately, providing well intentioned care does not divorce one from the responsibility to either make a definitive diagnosis or, alternatively, to rule out potentially life and limb threatening conditions. It is not reasonable to expect that Dr. RB make a clinical diagnosis or vertebral artery dissection, however it is reasonable to expect that he accurately differentiate between peripheral and central vertigo. On balance, I do not feel that the standard of care, as it existed in January of 2014, was met in Dr. RB's evaluation of Mr. AS.

Ross Berringer MD D(ABEM) MCFP(EM)

References

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