A DIFFICULT CASE

Ever missed a diagnosis? Then take another history!

We all worry about missing a diagnosis. Not so much because we may land up in jail or because our ego is put into question, although both may hurt. It is because we know that getting our patients better depends first on a correct diagnosis. We re-learn this every few weeks when an elderly patient in stupor arrives in hospital with laboured breathing, and the family is ready to let him or her die: this can cause prolonged suffering on all sides if one does not establish a basic diagnosis and understand the underlying cause. Another reason why making the right diagnosis helps is that neurologists have become quite good at secondary prevention – preventing another epileptic seizure, another relapse of multiple sclerosis, or another stroke. Not making the right diagnosis prevents us from preventing, and yes, we often do feel incompetent, untrustworthy, and a bad doctor if our patients come back with worsening disease because we made the wrong diagnosis.

THE STORY

A 30-year-old police officer had a first, generalized epileptic seizure off-duty at his home, witnessed by his wife. On arrival at our emergency department 45 min later, he was well, but had amnesia for the event. When the experienced neurology resident first took the history from the patient and his wife, the only unusual feature was that the seizure had occurred about

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Figure 1 DWI MR scan showing ischaemic lesions in the right superior cerebellum and left thalamus.

2 minutes after his daily bodybuilding session, which the patient had had to stop because of suddenly feeling unwell when lifting weights in the supine position. He denied excessive alcohol consumption, lack of sleep, or taking any licit or illicit drugs. The neurological examination 60 minutes after the seizure was normal, and there was no bitten tongue. Head-CT with contrast and basic laboratory work-up were normal. We decided to hospitalize the patient for 24 h to do an EEG and to get a better idea of what was going on – a first seizure in an otherwise healthy man is a somewhat traumatic experience.

When taking the history again the next morning, the patient now recalled that he had had some mild neck pain when getting up from the weight-lifting bench, followed by 'blurry vision' while he was walking over to the living room before the seizure. An EEG showed mild right-sided temporal abnormalities, and the epileptologist suspected hippocampal sclerosis. After discussing lifestyle and driving issues we started him on carbamazepine, sent him home and organized an outpatient MRI to look for temporal sclerosis.

Because something seemed not quite right – the story with the body-building, the mild neck pain, and the blurry vision – we decided to get the MRI soon after discharge. Did he have some orthostatic hypotension constricting his visual

fields when getting up from body-building, or a migraine turning into a seizure – certainly an unusual combination? Vertebral artery dissection would be likely linked to minor trauma, and the patient did not have any signs of cerebral ischaemia on neurological examination immediately after the seizure.

The next day, the radiologist called us to say that he had found two small lesions on MRI – one in the left anteromedian thalamus, and the other in the superior right cerebellum. What a nasty surprise! These are not the sites we usually look out for in seizure patients. We asked the radiologist to add diffusion-weighted (DWI) and angiographic sequences. He called us again an hour later – the two lesions were in fact compatible with acute ischaemia, according to the DWI (Fig. 1). In addition, there was an embolic occlusion of the top of the basilar artery – another nasty surprise (Fig. 2)! Had we sent a patient home with acute basilar occlusion? On carbamazepine? A young patient? These young patients are the very ones we should invest in most, because we can save the most 'quality adjusted years', are they not? Don't basilar artery occlusions carry one of the worst prognoses amongst cerebrovascular diseases (Caplan & Tetteborn 1992)? How many acute basilar occlusion patients have you sent home? We must be some of the worst doctors around ...

Has anyone else
seen a similar
combination of
bodybuilding, a
seizure, muscle-
building powder
and a top-of-the
basilar TIA?

Figure 2 MR angiogram showing occlusion at the tip of the basilar artery (arrow).

We re-hospitalized the patient immediately, this time in the stroke unit, and continuously monitored his neurological and vital signs – all normal. When we examined him again very carefully, especially looking for memory problems, vertical gaze paralysis, ataxia, and sensory abnormalities, we still couldn't detect anything abnormal. So we took another history, and the patient then said that in fact he hadn't been able to see anything when he had got up after bodybuilding – he had found his way around his living room by touching the walls with his hands. Yes, he was cortically blind before the seizure!

THE 'QUESTION EVOKED MNESIC POTENTIAL' – QEMP

Why did he not tell us about this before? Rarely can you blame the patient for an incomplete history. If the history is non-informative, changing or contradictory, the patient usually has a confusional state, a cognitive problem or a psychiatric disorder, not bad intentions. In this case, one possible explanation is that the seizure or the thalamic stroke or both caused some retrograde amnesia that resolved as time went on. Another possibility is that visual symptoms are often not reported spontaneously by patients. Specific

questioning can reveal a whole range of symptoms, however, such as formed and unformed hallucinations, metamorphopsia, palinopsia and others that don't have an interesting name yet. And maybe patients don't tell us about odd visual symptoms because they fear being considered 'psychiatric', or don't believe in what they are seeing, or because they think that the symptoms are unimportant. Another possibility is that we, the doctors, did not ask the right questions. And finally, the memory of the visual loss might in fact have been intact but 'concealed', and only repetitive questioning over time revealed it. This last phenomenon is well known to, and most frustrating for, the residents who find themselves before a patient who adds several precise and important details to the same questions by the attending physician that he didn't reveal just one day earlier. This is also one of the reasons attending physicians take another history in front of the embarrassed resident – they know that priming of the patient's memory by the resident often evokes concealed memories later. One might call this the 'question evoked mnesic potential'. If you like abbreviations such as VEP or AEP, this would be the QEMP (although we wonder about its sensitivity and specificity).

One reason why revealing buried or concealed memories is so important in neurology is that – unlike most other organs in the body – malfunctioning of the brain can manifest itself immediately and often very dramatically. Think of hypoglycemia, TIAs, epileptic seizures, hallucinations, out-of-body experiences, migraineous auras and so on. The transient nature of many of these phenomena means that their structural, electrical or biochemical substrates have disappeared long before the patients can get to a diagnostic machine. And even if they do make it in time, many neurological phenomena are simply unmeasurable with current methods.

Luckily, the human brain often creates a detailed and long-lasting memory of fleeting events, which gives the physician a unique opportunity to use 'QEMPs' to study and diagnose neurological diseases.

BACK TO THE PATIENT

A thorough cardio-neuro-vascular work-up, including transthoracic and transoesophageal echocardiography with microbubbles, testing for prothrombotic states, repeat vertebrobasilar MR-angiography and Doppler of the neck vessels didn't reveal any cause for the stroke. We added aspirin and a statin for mild hypercholesterolaemia to the carbamazepine. We kept questioning the patient about performance enhancing drugs, erythropoietin and steroids. Strokes can be due to high haematocrit (with or without external erythropoietin administration), but this was not found in our patient. In athletes, strokes related to vasoactive substances (Foxford 2003) and to androgenic steroids (Frankle 1988) have been reported. Another bodybuilder we have seen had a generalized seizure 20 minutes after he had injected himself with an androsterone. Our patient again denied all these substances.

He eventually came back to the office with a protein-rich powder that he took once a day for muscle building. We had it analysed for stimulant and vasoactive drugs – negative. According to the label, it contained acesulfame (an artificial sweetener) and Aminogen® (a blend of enzymes that helps break down protein foods into their amino acids, that should make it easier to absorb), but we couldn't find any association with stroke in the literature.

Has anyone else seen a similar combination of bodybuilding, a seizure, muscle-building powder and a top-of-the basilar TIA? By the way, should we now call this a stroke rather

than TIA because of the imaging evidence of infarction (Albers 2002)? Have *you* sent home a patient with an acute basilar occlusion? We can't imagine you have, but if so, please let us know. It wouldn't make us feel better, but at least it would be an interesting surprise after all the bad ones.

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