POST EXAMINATION MEDICAL REPORT

DATE

For whom it may concern, regarding Mr. XXXXX (DOB: XXXX): The patient has been suffering from chronic headaches since the age of five years old. The symptom is described as a deep ache and pain that was, initially, situated in the temple region (undulating between left and right), but has spread to the eyes, occiput, neck, and even the chest. He states that he fell and hurt his head hard as a child, approximately four years old, and suspects that the symptoms started after this. He also says that his parents brought him to an “old lady” who performed some sort of cervical manipulation and “lengthening” as his neck appeared “short” – this, at the age of approximately three years old. Nonetheless, as it appears, shortly after these happenings the patient developed the headache problem. The patient has undergone numerous medical consultations and imaging studies for his malady, but so far, to no avail. He says that his symptoms are better in the morning and evening but worse mid-day. He also says that he will know how the day will turn out, pain wise, if he wakes up with a headache. He says that now, at adult age, the symptoms directly follow his neck pain. Elaborated, he says that it starts with a supraclavicular pain / tension that progresses into the lateral chest following the long thoracic nerve, chest, upper back, and finally the head (occipital, temporal and orbital zones). The pain patterns are ipsilateral, but as stated, can fluctuate side to side from day to day. He says that if he falls asleep lying on his arm, on his side, then he is much more likely to develop ipsilateral headache the coming day. Salty foods make his headache worse. He also says that running or lying down flat without a neck pillow can improve his symptoms intermittently, but they return afterward. Finally, he states that the symptom-ipsilateral eyelid shows ptosis when he feels quite ill. I had an hour’s long consultation with the patient to review his case. He requested this report on his own initiative.

I have reviewed his imaging. Magnetic resonance imaging of the neuraxis forelies, including angio- and venograms of the head. All of these images were determined normal from the radiology centers. However, some findings may suggest underlying craniovascular aberrancy, consistent with his seemingly vasculogenic headaches / migraines. Firstly, the lateral ventricles appear narrow (fig. 1), which is a common sign in cases of elevated head pressure (1,2). The internal jugular veins are mildly stenosed at the skull base (fig. 2), approximately 40%, but I am not so sure that this is clinically relevant. There is also transverse sinus stenosis bilaterally (fig. 3), which might be clinically relevant. The left sinus carries a low signal overall. There is mild depression / concavity of the pituitary gland (fig. 4), which is another common sign in head pressure cases, albeit a very subtle one in this case. FLAIR T2 white matter hyperintensities are also seen adjacent to the posterior horns of the lateral ventricles (fig. 5), which is yet another common sign of head pressure. Finally, there is evidence of several pseudomeningocele (fig. 6). Although none of these signs are blatant *proof* of pathology, I think they are subtle unions of suggestive craniovascular pathology. I believe that this is also suggested by his symptoms. Pertaining to the spine, fairly normal findings are present. There is some straightening of the cervical spine with multilevel disc degenerative disease, but there is no significant prolapse and certainly no emerging root or central canal stenosis. In other words, clinically insignificant other than

suggesting some room for postural improvement. The peridural veins are dilated (fig. 7), and this can sometimes be seen in cases of CSF leak. However, I am not so convinced that this is the problem here.

It is interesting, and, in my opinion, important to note that the patient's headache correlates with his neck symptoms. The tandem of occipital pain / hemicranial headache that is directly associated with supraclavicular, chest and arm symptoms is a common constellation of symptoms that is seen in thoracic outlet syndrome, which I believe the patient developed, progressively so, after the neck manipulation and falls that he underwent as a child. Thoracic outlet syndrome is a known instigator of migraine headaches (3,4,5,6,7), although the pathomechanism is poorly understood. However, as we showed in our 2020 paper (3), TOS – due to obstruction of the subclavian artery distal to the carotid and vertebral branches – has the potential to cause flow retrogradation and hyperperfusion of the cranioarterial system. It has been our experience that this frequently results in migraine headaches, and especially occipital headaches, as documented to be present in as much as 86% of TOS patients according to renowned TOS researcher and surgeon Dr. Richard Sanders (8). I believe that the patient's symptoms are of craniovascular origin.

Upon clinical examination, the patient demonstrates an immediately positive costoclavicular test, causing pain into both arms as well as creeping pain into the right shoulder. Roos' test was positive in approximately 15 seconds. Morley's test reproduces his lateral chest symptoms within approximately 15 seconds (ie. a Tinel's sign). We proceeded with several vascular tests: The Valsalva maneuver increased his headache, and noted progressive worsening of headache after the test's completion. The Queckenstedt's test for manual venous compression (IJV) was negative. We moved on with the cervical retraction test (3), but it was not conspicuously positive. However, the patient did develop an aggressive migraine that lasted for the entire next day. It is still too early to know if this is, or not, directly caused by the provocative maneuvers that we performed, but I expect that it was.

Now, could the patient's symptoms be secondary to a subtle case of idiopathic intracranial hypertension or craniovenous hypertension? It is possible, but 1. It does not explain the correlated neck and upper limb symptoms and 2. The Queckenstedt's test was negative. Moreover, the optic nerve sheaths appear perfectly normal. I suggest a trial of 250mg of acetazolamide for 7 days, and *if* non-responsive or worse on this drug, then cessation, followed by propranolol 20mg every 12 hours, especially before bedtime (5). If the acetazolamide *contrary to my expectations* causes improvement of symptoms, then cessation followed by a lumbar puncture might be warranted.

I have also requested ultrasound duplex imaging of the carotid and vertebral arteries, as well as a fundus exam. As shown in my paper on TOS CVH, fundus and doppler imaging is useful because it permits a qualitative assessment of the intracranial vasculature. The carotid artery exam shows slightly pulsatile vertebral artery waveforms, but the changes are subtle (fig. 8). For the right internal carotid artery, somewhat pulsatile waveforms are also seen, but with a normal systolic upstroke (fig. 9). This image (study) is of poor quality, unfortunately, but I was forced to include it seeing as the right ICA was absent in the good study that I was

provided with. It does show turbulence, ie. filling of the spectral window, but because the images are so dirty, it is impossible to know whether or not this is legitimate turbulence or simply poor machine configuration; probably the latter. For the left ICA, however, the waveforms clearly change: We see slowed systolic upstrokes with accelerated post-systolic delination (ie. the initial diastolic segment), “pyramidal waveforms” (3) (fig. 10). These are typical TOS CVH waveforms. The jugular veins demonstrate clear color doppler flow in the caudal direction with normal phasic waveforms (fig. 11); this once again reduces the likelihood of the patient’s problem being significantly venogenic. As for the fundus images, fairly normal circumstances are observed, except for mild arterial tortuosity with, perhaps, some increased arterial light reflex (figs. 12,13). This, as shown, only present on the side that is also abnormal on the doppler.

Summary

In conclusion, this seems to be a case of craniovascular hypertension secondary to cervical injury at very young age. I strongly suspect that the cervical manipulation that the patient underwent at 4-5 years of age has been the predominant contributor to this affliction, if not its direct cause.

Scalenus injury with resultant TOS is common in cases of iatrogenic cervical manipulation. In further support of this notion, the patient’s headache and, especially, occipital headache elicits in tandem with chest, upper back and arm symptoms which is absolutely typical for patients with TOS. Regarding triggers, the patient checks fewer obvious boxes, but we have been able to reproduce his symptoms with provocative scalenus strengthening. Finally, the vascular imaging, though not conspicuous, does show several signs that are compatible with a TOS CVH diagnosis.

The patient’s affliction, as such, seems to be somewhere within the lower echelon of moderate in severity. If it turns out that my diagnosis is correct, ie. that his symptoms are indeed second to scalenus injury and thoracic outlet syndrome, then he is absolutely within the spectrum of patients who have good conservative prognostic outlooks. I have suggested just that; conservative treatment for thoracic outlet syndrome. We will also rehabilitate other important muscles in the neck, such as the suboccipitals, splenius capitis and cervicis,trapezius, sternocleidomastoideus and levator scapulae muscles. The upper cervical rehabilitation will also improve venous drainage (10). His symptoms are not that aggressive, per se, but has been ongoing for 30 years. It is therefore too early to tell with regards to treatment time, but having experience with similar cases, again presuming that the diagnosis does turn out to be accurate, then I would expect somewhere between one and two years.

With regards,

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References:

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Attachments:

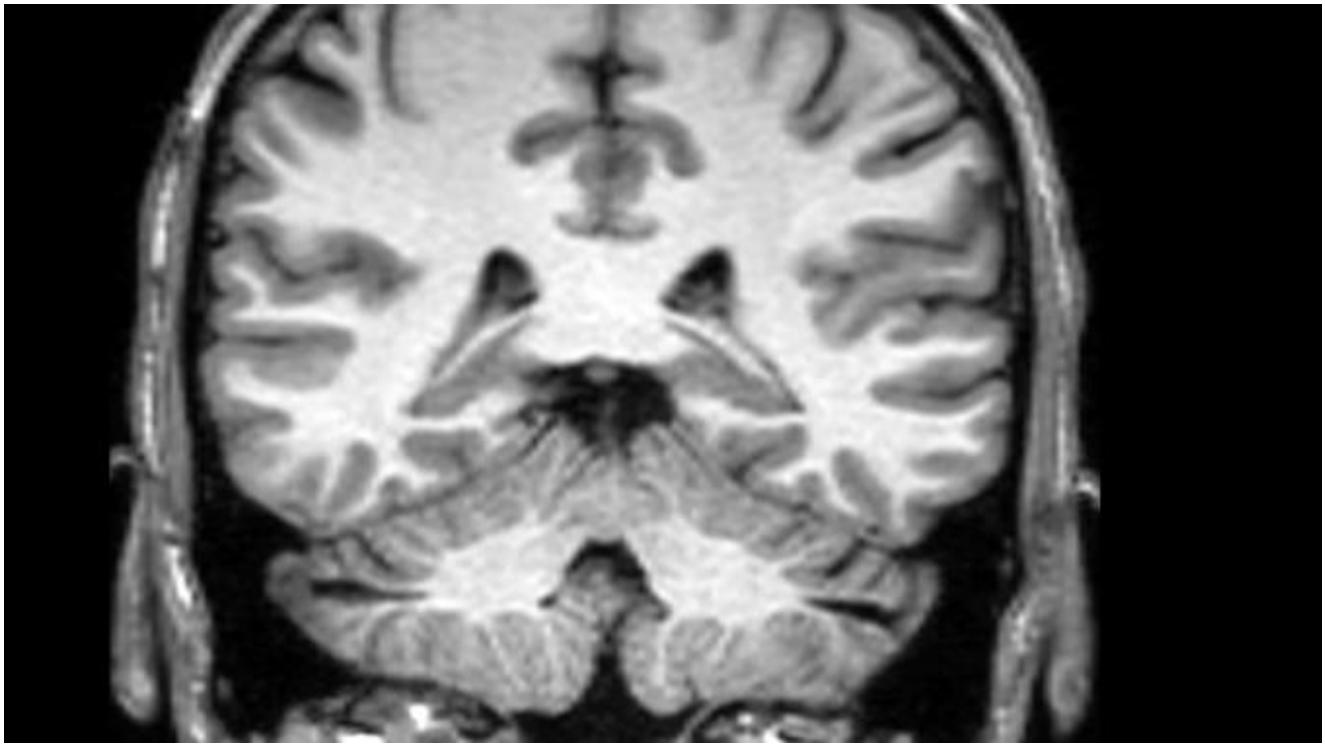

Figure 1: Ventricular narrowing. This can be a sign of elevated intracranial pressures, but is not – in my experience – limited to sole CSF hypertension.

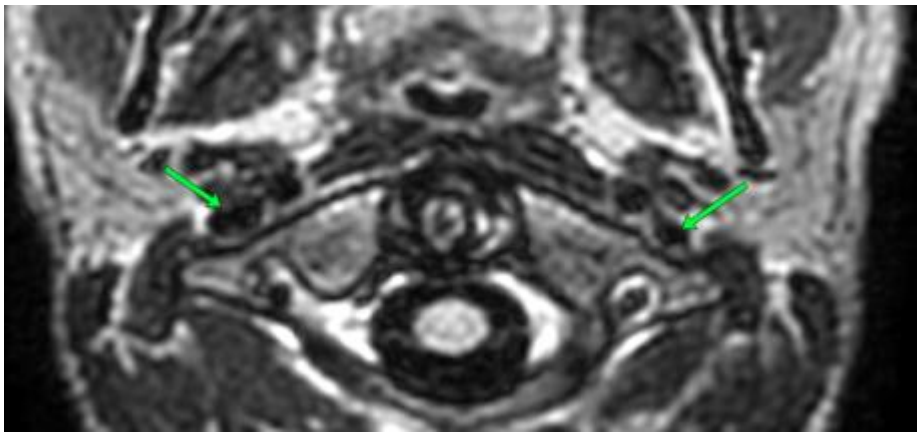

Figure 2: 40-50% stenosis is seen of the jugular veins at the skull base. This tends to get worse when the patient is upright, but seeing as the patient says that he feels better at the end of the day, I am not so convinced that this is a clinically important finding. There are some signs of CSF hypertension, though equivocal, and as stated in the main text, a trial on Diamox 250 may prove diagnostically helpful.

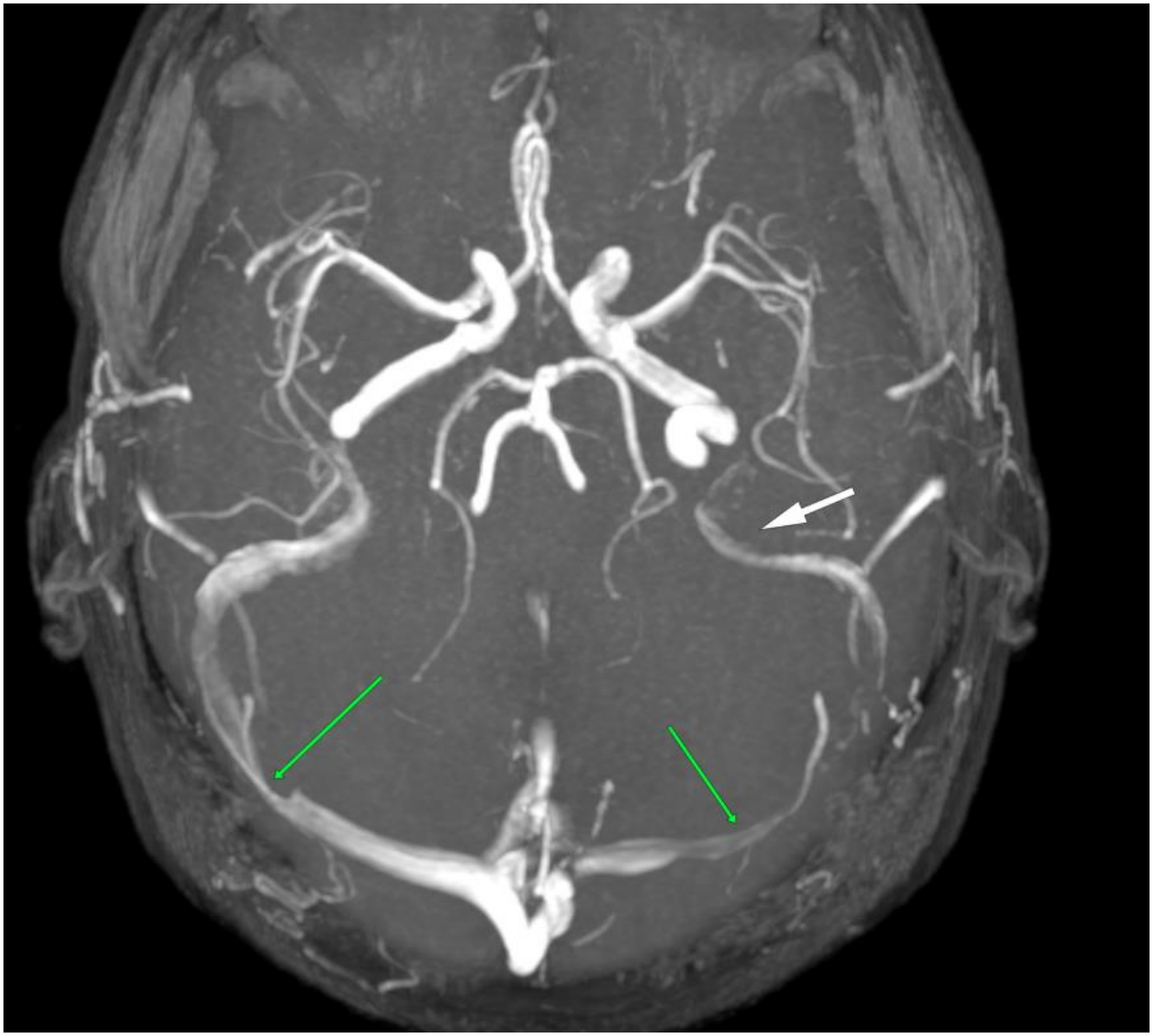

Figure 3: The MR venography does demonstrate stenosis of both the hypoplastic (left) and dominant (right) transverse sinuses. It is possible that this, in tandem with the jugular vein problem, can cause problems. A doppler volume flow exam of the jugular veins can be helpful in proving whether or not there is normal outflow volumes. Flow outflow rate should be no less than 250mL/min per vein, and no less than 700mL combined (5,9).

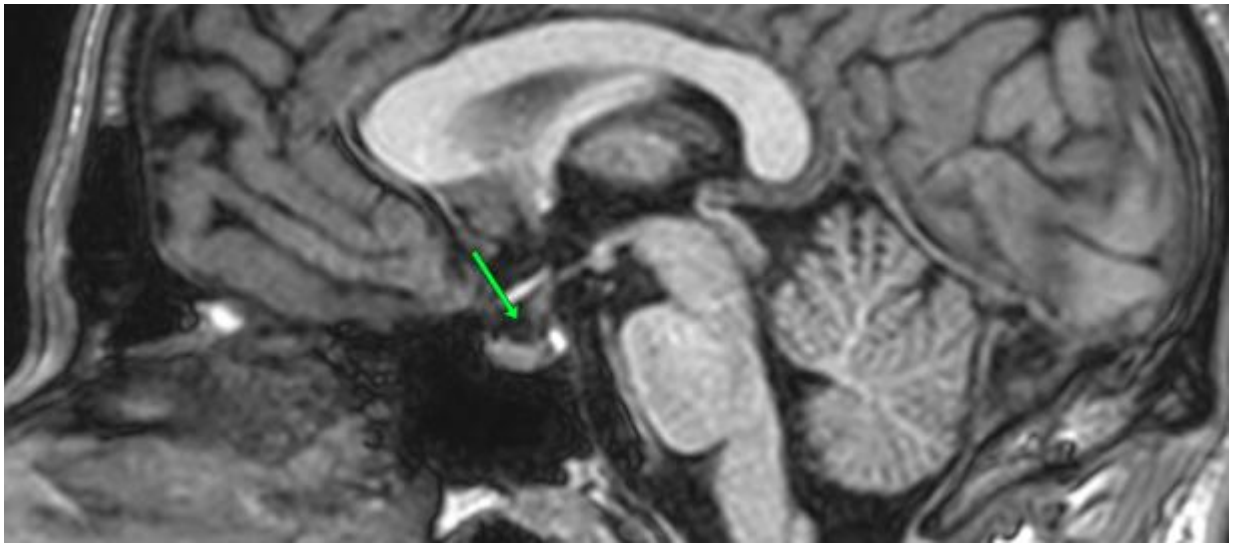

Figure 4: Subtle concavity is seen of the pituitary gland. This is not a rock solid finding, but is yet another sign that may suggest raised intracranial pressures.

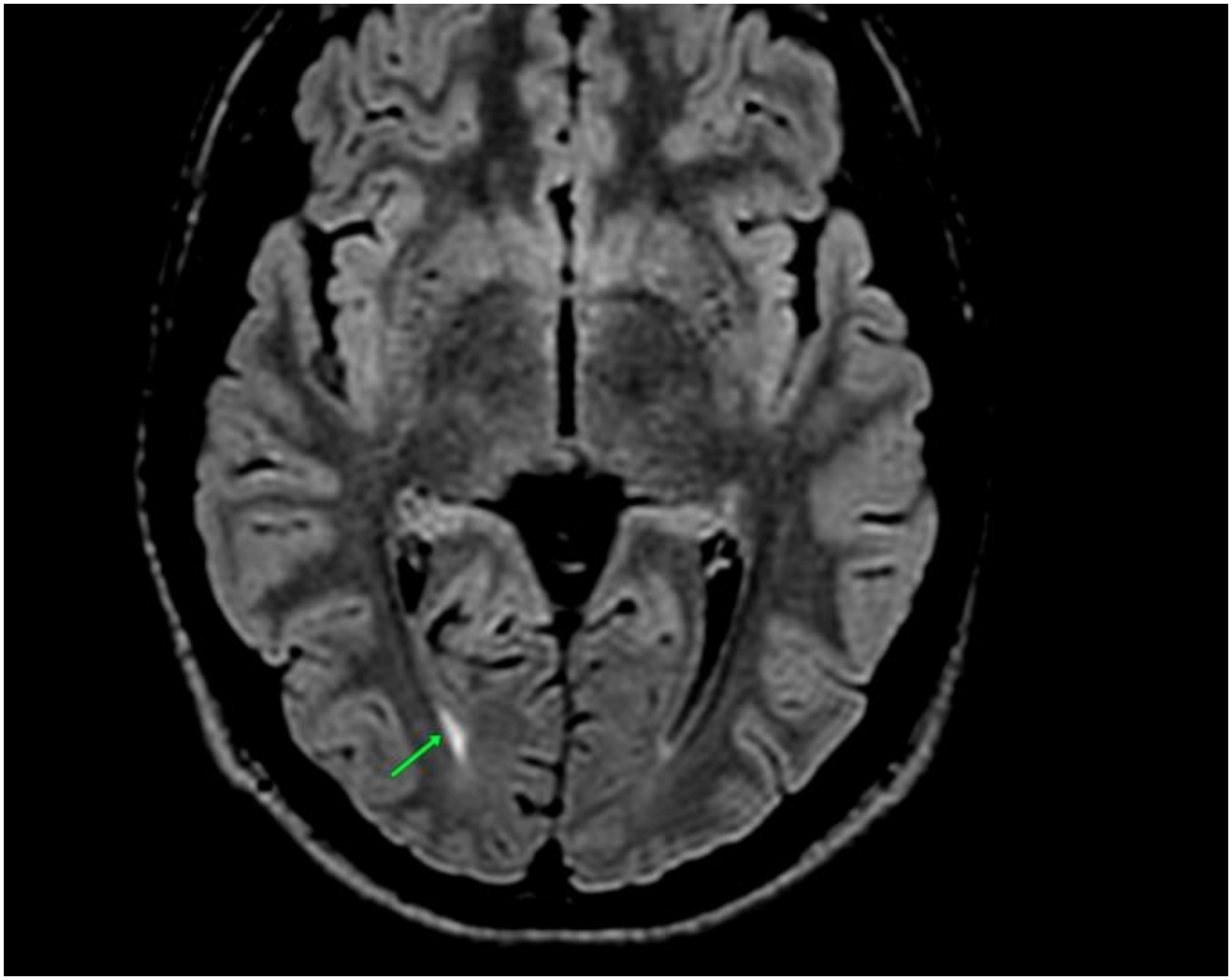

Figure 5: It is not unusual to see periventricular and ependymal swelling (T2 FLAIR hyperintensities) in cases where the intraventricular pressure is elevated.

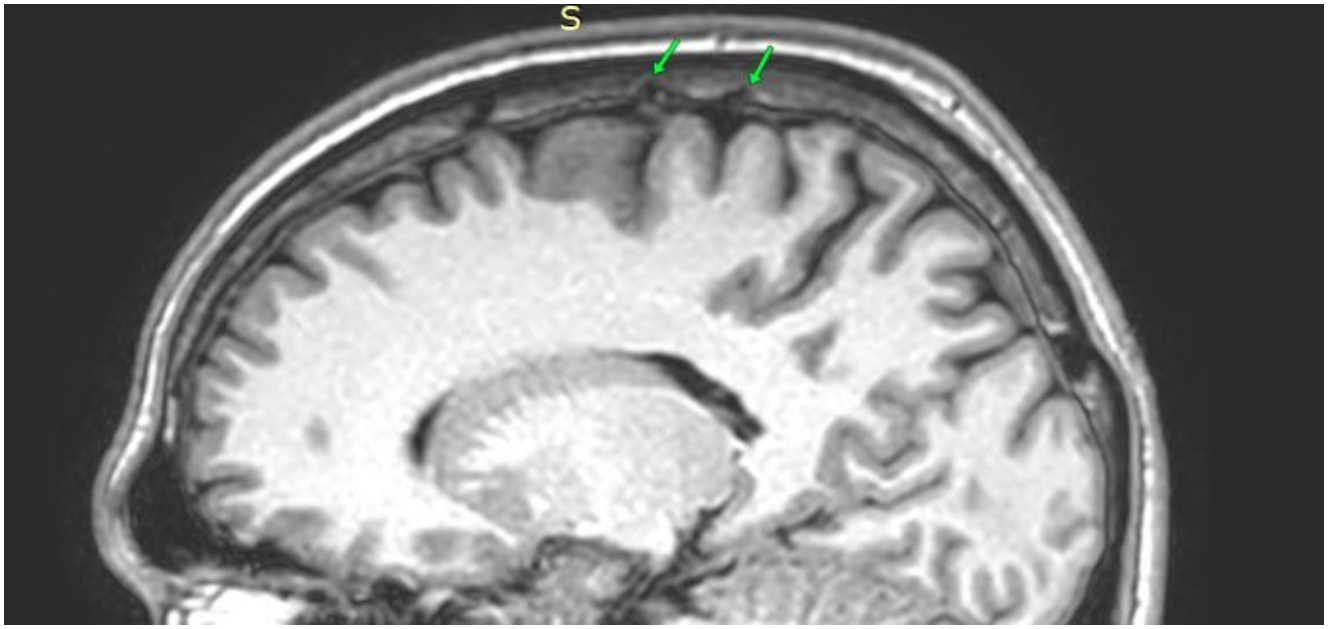

Figure 6: Finally, small protrusions into the porous parietal bone is a common finding in patients with a history of elevated intracranial pressures.

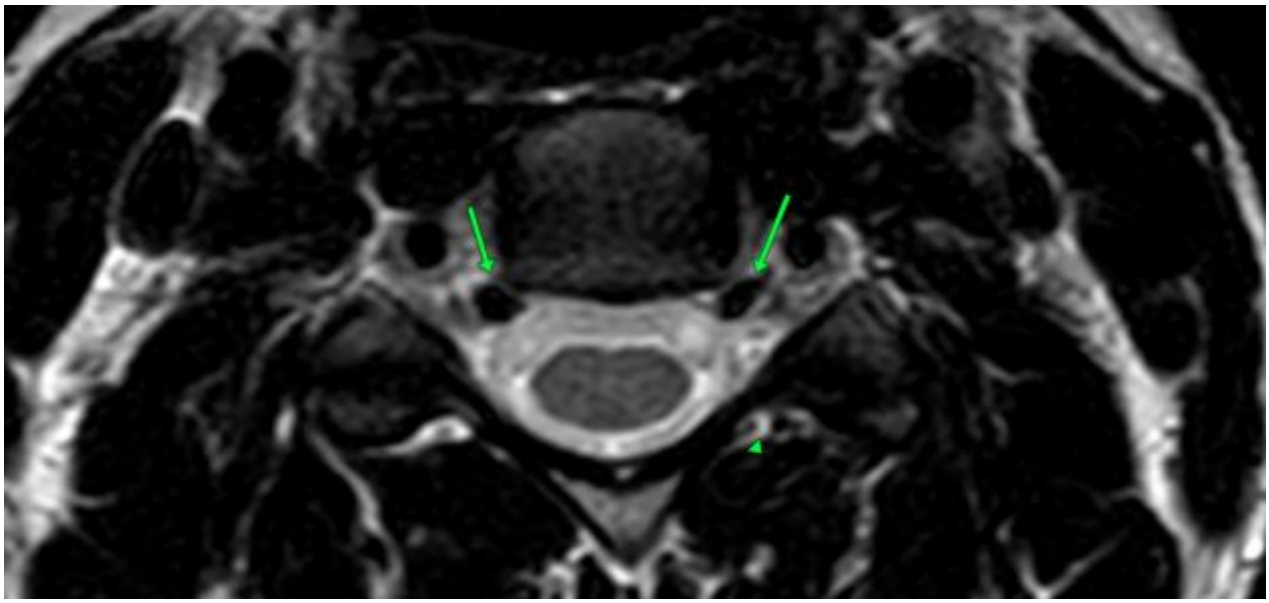

Figure 7: Peridural venous dilation is seen in the spinal canal. This is a common finding in patients with either reduced CSF pressure or elevated accessory venous outflow. The latter makes more sense than the former. It can also be an asymptomatic / coincidental finding.

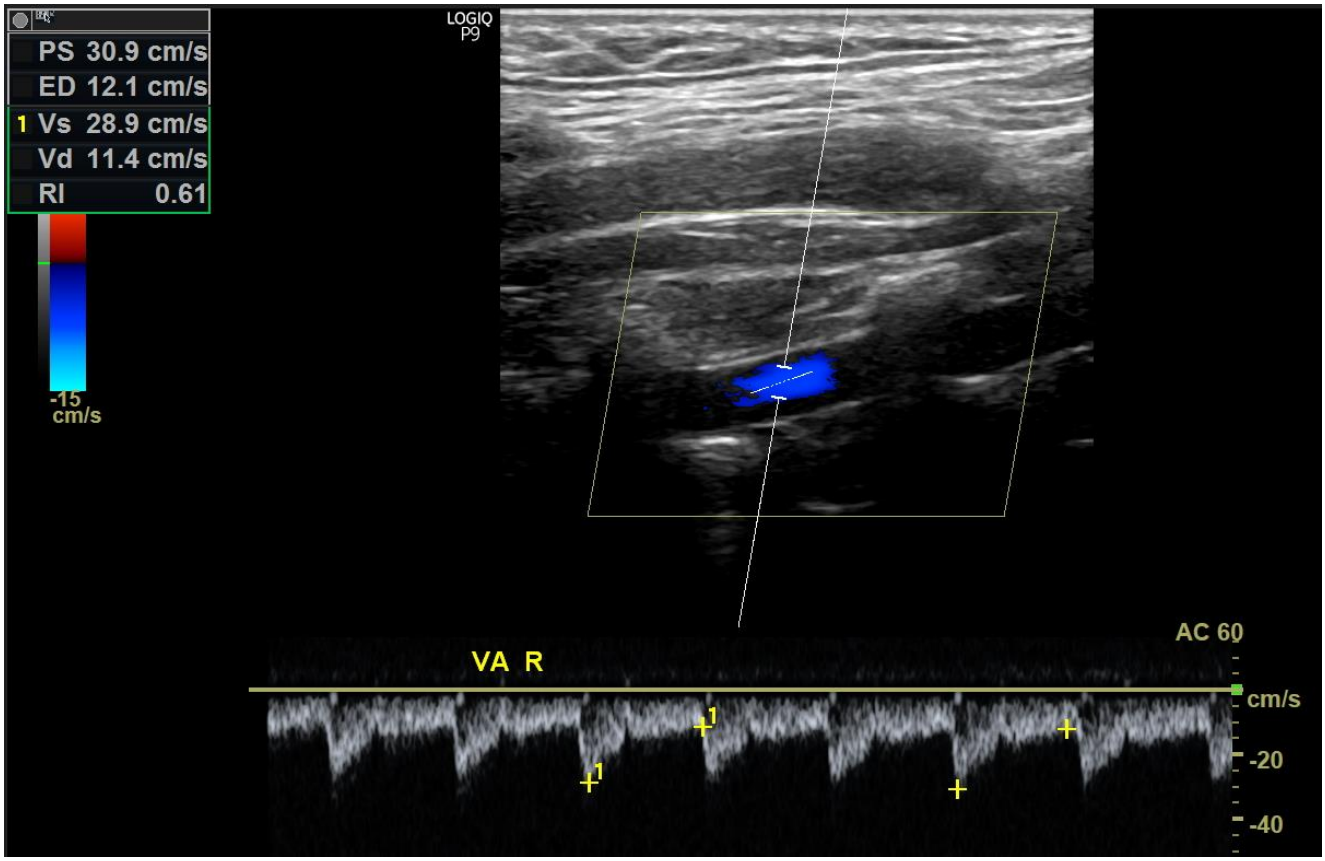
PS 30.9 cm/s
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Figure 8

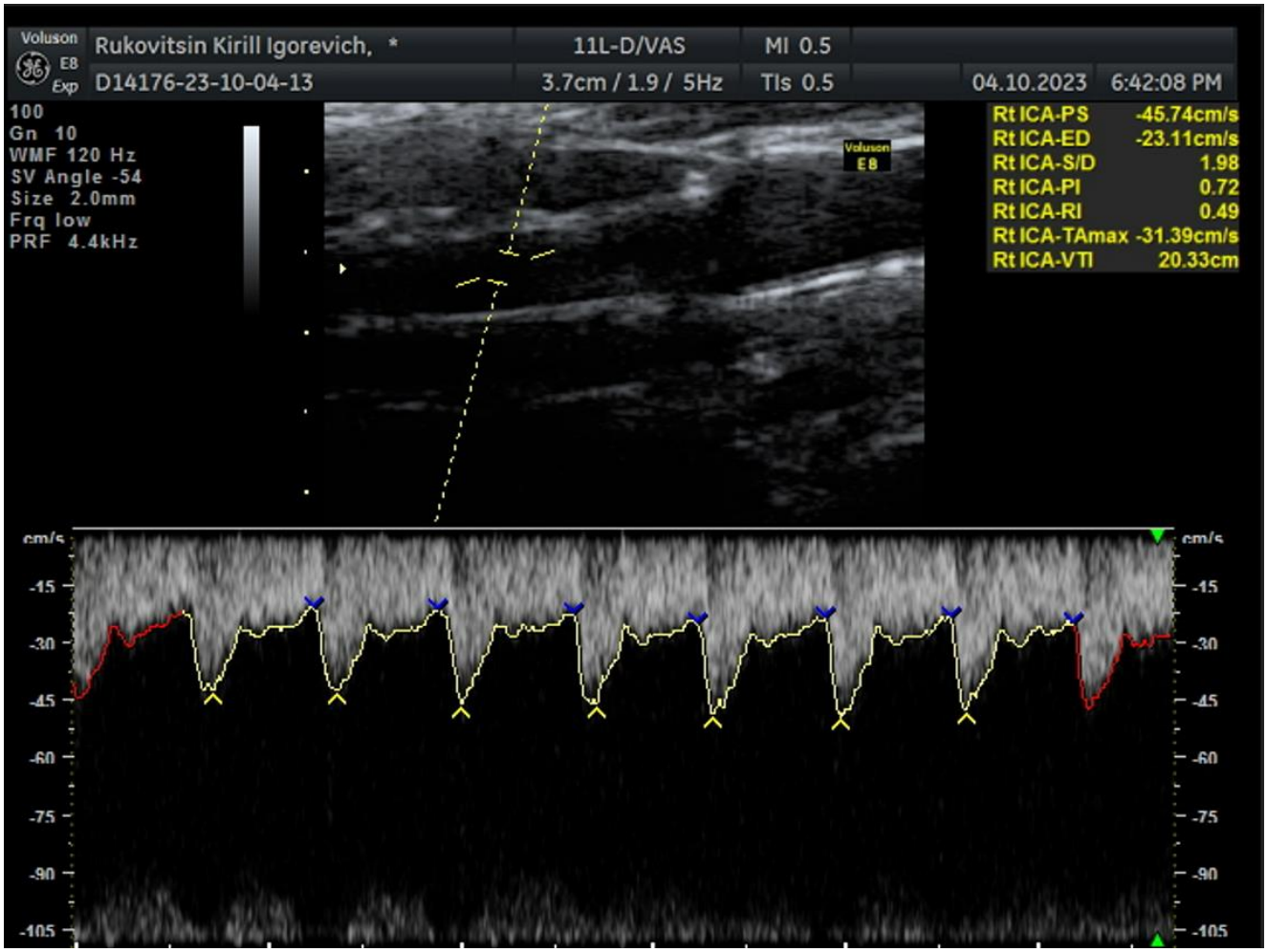

Figure 9: For the right ICA, we only have this suboptimal image. The waveforms are probably quite accurate, although the doppler angle correction is inaccurate. It is also overgained, and there is venous interference. That said, there is no delay in systolic upstroke, but some increased acceleration of beginning of diastole is seen (raised pulsatility). The best place to measure this is usually 1-2 cm distal to the carotid bulb, but in this case we are at the distal ICA.

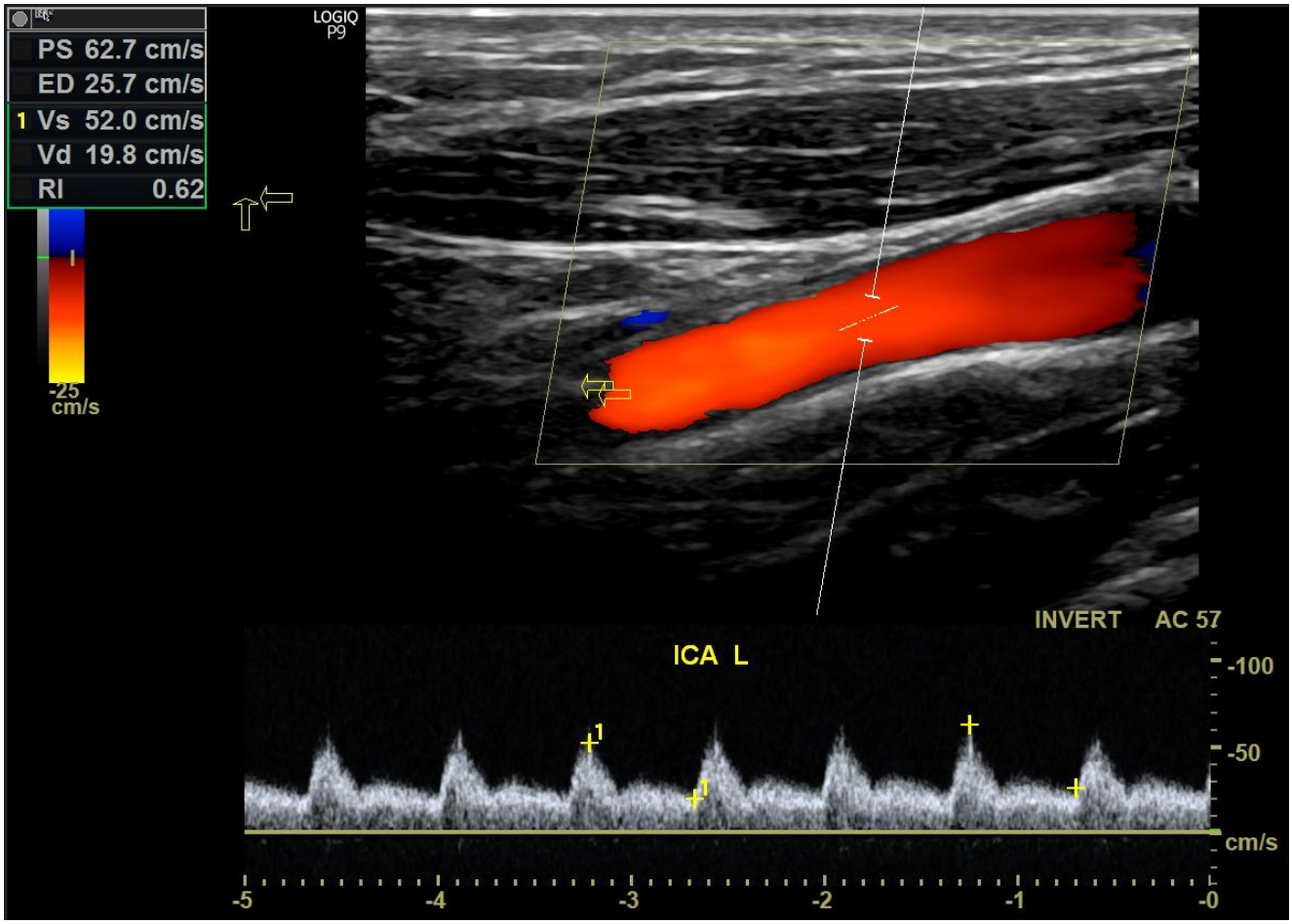

Figure 10: In the left internal carotid artery, "pyramidal" waveforms (3) are seen, suggesting distal arterial congestion. Due to intracranial hypersaturation, the systolic peaks delay in reaching velocity (the slowed systolic upstroke) and also decline at a greater acceleration than expected in stereotypically "low pressure vessels" such as the ICA. This is a typical waveform seen in TOS CVH. Also note the filling of the spectral window; turbulence.

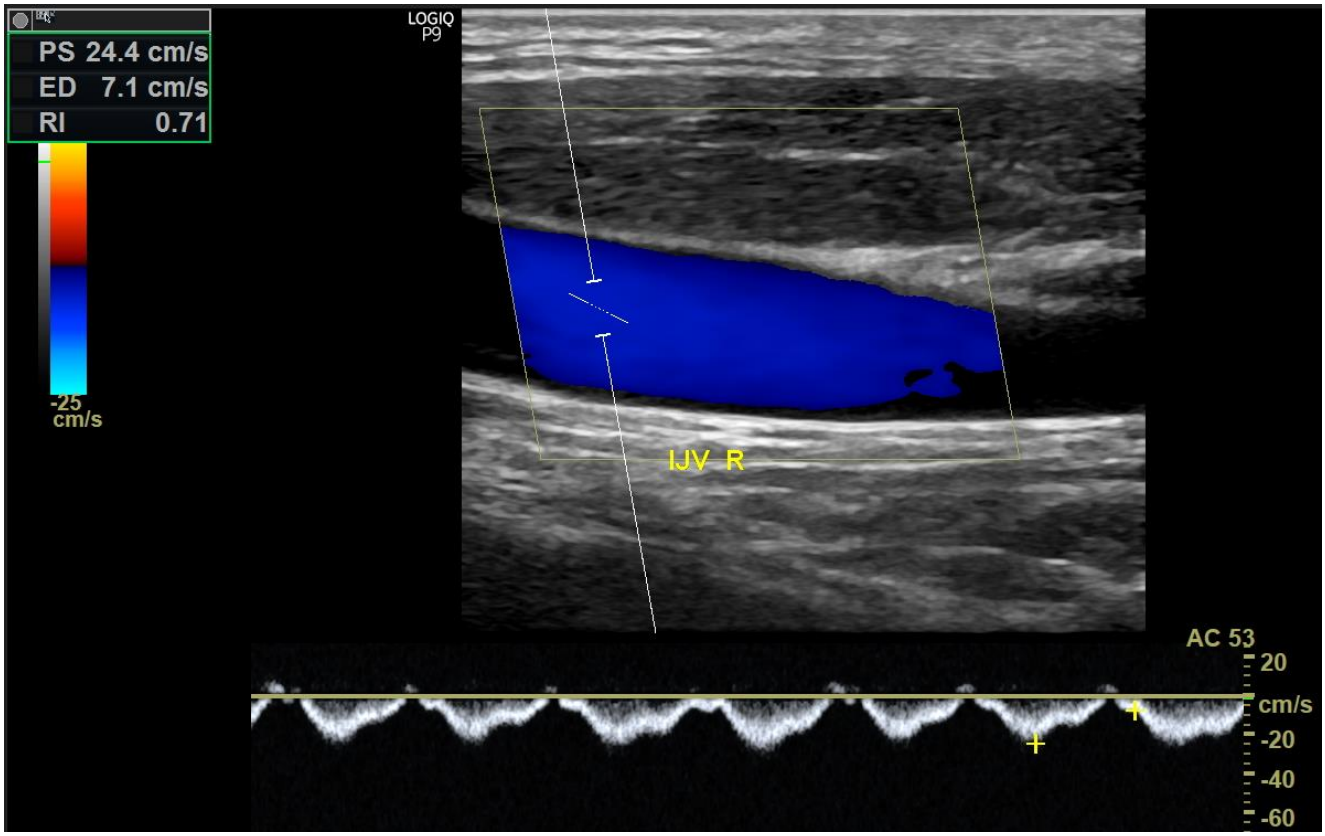

Figure 11: Normal venous phasic flow, and good luminal color filling.

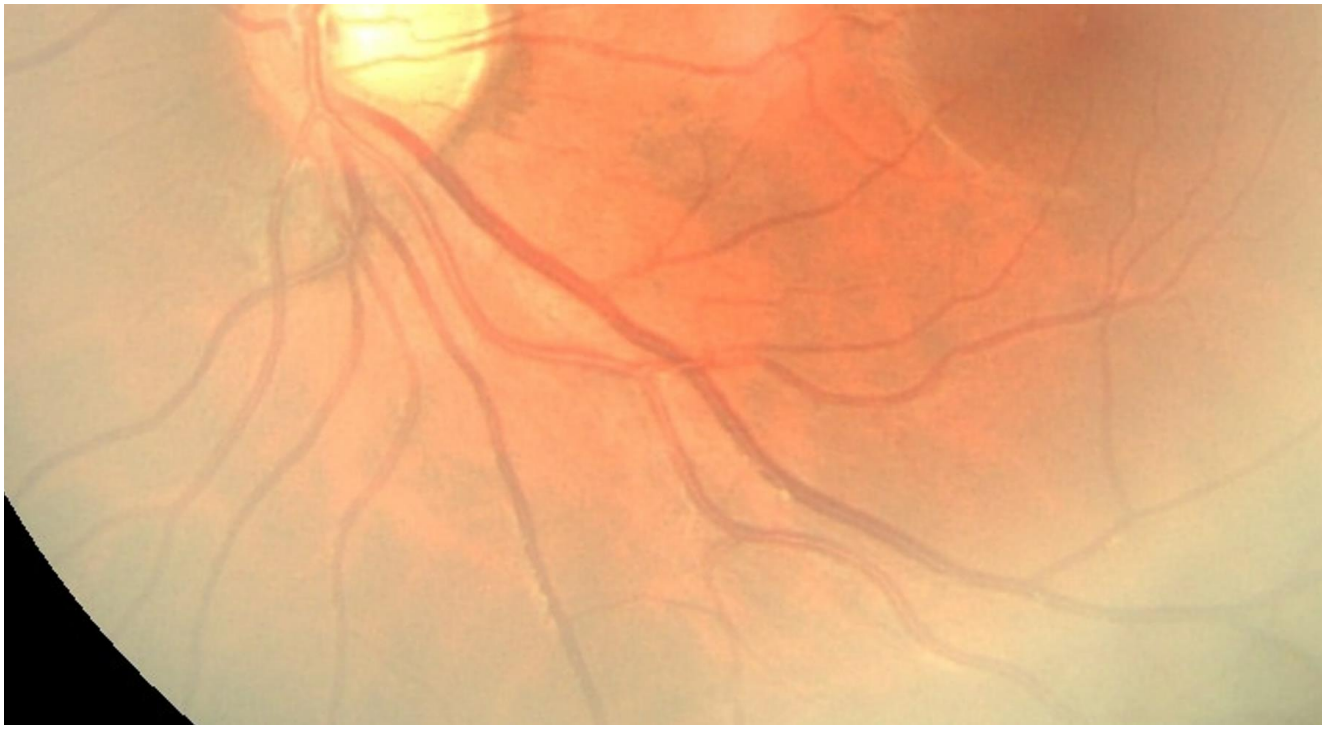

Figure 12: On the left retina, an increased arterial light-reflex is seen along with some tortuosity, which are signs of retinal hypertension. The point here is not to suggest impending eye disease, but rather suggestive of the cranioarterial status and quality. There is one site of very subtle arteriovenous nicking, but again, very subtle.

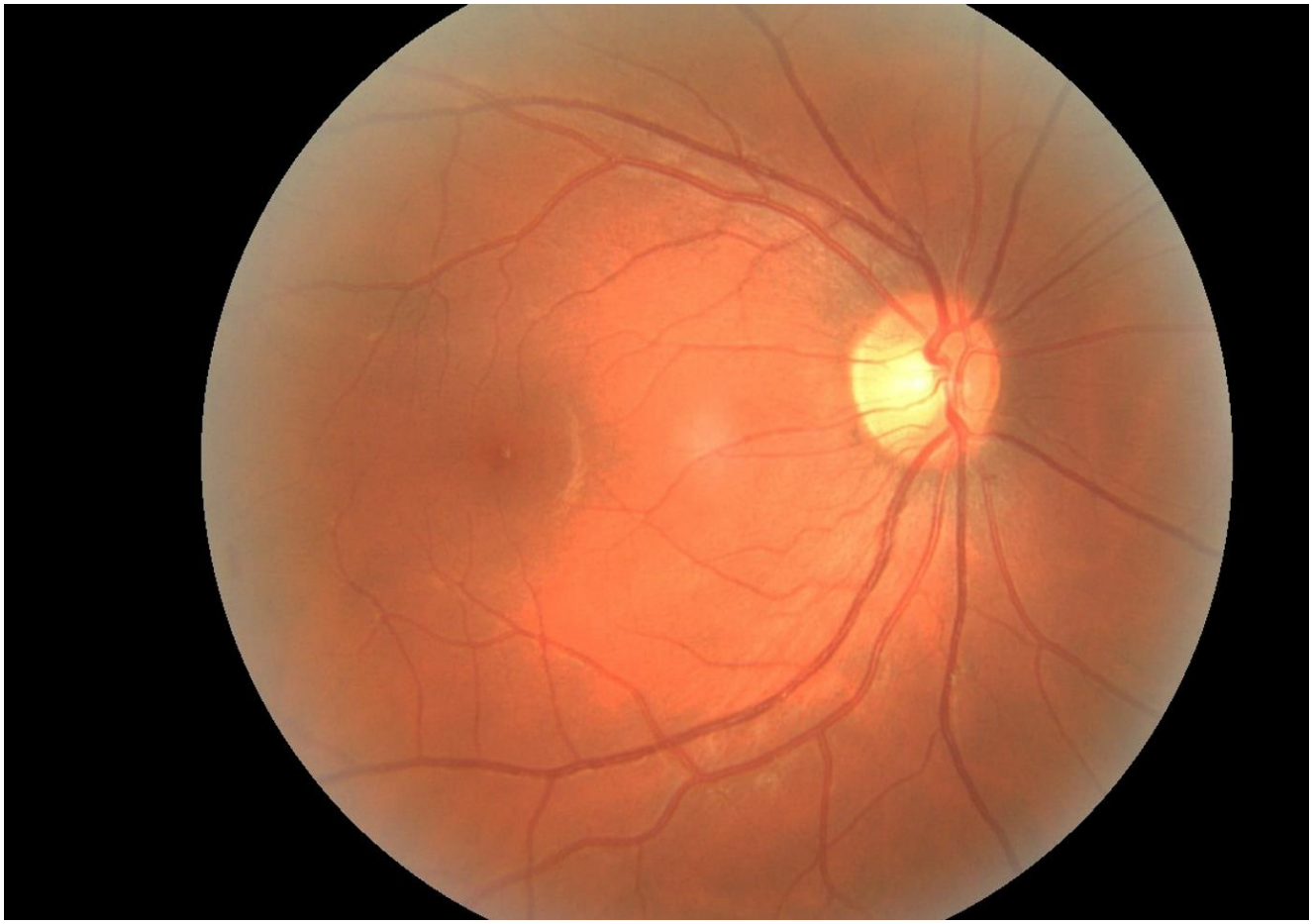

Figure 13: On the right retina, although there is mild increase in arterial light reflex, no tortuosity is seen and no evidence of AV nicking.