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Temp	35.1 °C
Pulse	59 bpm
Resp	19/min
BP	**211/108**
SpO2	99%

These were the vital signs for one of my most interesting patients in the ED.

HX:
“46 YO AA M with h/o HTN, current smoker, on lisinopril but not amlodipine as prescription was lost, works as cement truck driver and sets up truck to pour cement. Two days ago around 1500 was working and began feeling lightheaded, clammy, sweating profusely. Reports somewhat hazy recollection of events, reports co-workers told him he was confused (no dysarthria) and pt went into truck, turned up AC to max. Reports he may have had a brief episode syncope; he is rather unsure. As air cooled he felt better and was able to drive home though he notes some blurred vision L eye (no amaurosis, no eye pain) and throbbing L temporal headache. The following day he went back to work, took ibuprofen, drank lot of liquids, headache seemed to improve but never resolved then toward evening intensified, was unable to sleep overnight so came in for evaluation. Emesis x 2 on the first day, NBNB, in early evening; none since. No fevers. No CP, no SOB, no abd pain. No arm or leg weakness. Sig photophobia, pt wearing sunglasses, +phonophobia. Had a similar event in July though more of a R-sided headache. Pt reports some slight numbness to entire L face but not to arms or legs, reports slight blurred vision and transient spots in L eye visual fields –Attending Note with Resident Edits

PE:
“Gen - alert, NAD
Normal gait
CN 2-12 intact but endorses some slight numbness L c/w R side of face although he can feel me touching both, it feels "different"
Nl finger nose b/l, No pronator drift
Str 5/5 throughout
Lungs CTA
Abd Soft NT
No meningismus
Slight tenderness temporal artery”

While our work-up included tests and questions that attempted to rule out temporal arteritis vs. migraine/heat stroke vs. stroke vs. SAH, our initial gestalt of the patient was one of stability, due to the improving nature of the patient’s symptoms that began two days prior to presentation. However, the elevated BP, possible continuing neurologic components, episodes of vomiting, with possible syncope were concerning for a serious

etiology of our patient's complaints. Our non-contrast CT Head brought back the following read:

"1.New subarachnoid clot over surface of left parietal lobe. The source of this bleeding is not evident on unenhanced CT but possibilities include a peripheral mycotic aneurysm or dural AV fistula which would be better evaluated by CTA or catheter angiography."

This case prompted me to delve more into some of the clinical presentations and evolution of SAH. Two of the most important risk factors my patient had for a SAH was his **smoking** history, which can increase his risk of SAH by four to five times, and his **uncontrolled hypertension**, which can double it. While the notes written during our patient's hospital stay, by neurosurgery and neurology, paint the picture of a thunderclap headache, the story reported by the patient in the ED was not so clear-cut. What was significant was that the patient's **headache felt different from headaches in the past**, which warrants the consideration of SAH on initial evaluation. According to Tintinalli's EM, only 11-25% of SAH is diagnosed by the classic description of a "thunderclap headache."

Other co-existing symptoms with headache, which typically present with ischemic stroke, include nausea and vomiting, altered mental status, and photophobia, all of which our patient had at one point in time. What was difficult to ascertain from the patient, was whether or not he did have an episode of dysarthria at the time of onset or true syncope minutes after sitting in his vehicle for environmental temperature relief. Though it did not change our initial management, it did, at first, challenge the severity of suspicion for our patient's condition.

Of note, the patient arrived to the ED greater than 48 hours after having symptoms. "The sensitivity of modern CT in diagnosing subarachnoid hemorrhage is highest shortly after symptoms begin and is estimated to be **98% when performed within 12 hours** of the onset of symptoms." As expected, the **sensitivity continues to decline as the hours pass** until, an estimated ten days post-symptom onset, when the SAH has probably been completely resorbed.

If the **CT is normal, then a lumbar puncture** is indicated. The most important tests of CSF for SAH are **xanthochromia and RBC count**. It is important to note that it **takes up to 12 hours** for xanthochromia to develop and may persist in the CSF for up to three weeks. In addition to this timing effect, a traumatic rupture could produce xanthochromia within two hours of CSF sample retrieval. The **amount of RBCs has not been clearly defined** by the literature, in both cases of true SAH and the normal amount considered in a traumatic puncture, and can be difficult to tell the difference even when using the RBC count in the fourth or final CSF tube. What is reliable for ruling out a SAH is the combination of a negative head CT, negative xanthochromia, and an RBC cell count of $<5 \times 10^6$ RBCs/L.

According to Tintinalli, "Intracerebral and extracerebral complications of subarachnoid hemorrhage include **vasospasm, rebleeding, cerebral infarction, cerebral edema, hydrocephalus, intracranial hypertension, fluid status and electrolyte abnormalities, respiratory failure, myocardial dysfunction, thromboembolism, and sepsis**." The greatest risk of rebleeding can happen in the first 24 hours and, with adequate blood pressure control, can be significantly reduced (this is controversial). We

initiated a nicardipine drip. We wanted to lower the blood pressure, with recommended MAP goal is < 130 mm Hg, to where his baseline was, without lowering the rate and a CCB was also used to prevent vasospasm. **Vasospasm can occur anytime between 2 days and 3 weeks of symptom onset**.

Follow-up imaging studies were done on our patient including head/neck MRI/MRA, but did not find a conclusive cause for our patient's subarachnoid bleed. Both neurology and neurosurgery agreed that the most likely cause of SAH was probably CSF hypotension, after etiologies such as AVF and tumors were considered, though a leak was not specifically identified.

SAH Sources: Tintinalli's Emergency Medicine