The Importance of a Thorough History Review: A case study of a Patient with Acute Rapidly Progressive Encephalopathy.

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Introduction

In the age of modern medicine, the use of a multitude of laboratory tests, the ability to collaborate inter-departmentally, as well as the use of advanced imaging modalities have become essential for patient management.

Despite this, rudimentary information such as a thorough history and examination can be overlooked and obfuscated within the myriad of results, consults, progress notes, and lists of possible diagnosis.

One paper found that in 76% of cases, history-taking lead to the final diagnosis compared to laboratory investigations leading to only 11% of final diagnoses, supporting the concept that most diagnosis are made from the medical history.

In addition, electronic health record systems have timesaving features such as auto-populated problem lists, diagnoses and results as well as copying previous notes that are designed to streamline the documentation process, but also do not guarantee that clinicians have thoroughly reviewed findings. This allows perpetuation of potentially misinterpreted information and can allow clinicians to forgo performing their own thorough examination and evaluation and instead rely on existing patient documentation. Such cases allow patients with causes that can be more easily diagnosed with a thorough medical evaluation to be complicated from over-assessment or even miscommunication.

Case presentation

A 54-year-old male with history of epilepsy s/p VNS, history of prostate cancer s/p resection, history of paraneoplastic retinopathy, and diabetes mellitus type II presented to the emergency room with approximately 4 weeks of rapidly progressive cognitive and functional decline. Approximately, one month prior to admission he was independent in all ADL's but was admitted after increase seizure frequency (16 generalized convulsions in 24 hours) which prompted increase in Clobazam to 80mg/day. On arrival to emergency department approximately 4 weeks later, the patient was unable to perform ADLs, had decrease oral intake, was minimally verbal, and non-ambulatory. Home AEDs included clobazam 80mg/day, perampanel 8mg/day, lacosamide 400mg/day, lamotrigine 750mg/day.

- Initial vital signs were within normal limits.
- On exam, he was lethargic, making incoherent sounds, unable to follow commands, able to move all limbs but appeared to have increase tone.
- CMP was normal, WBC normal. No signs of systemic infection identified.
- NM PET Brain showed diffuse cortical hypometabolism.
- EEG monitoring revealed occasional right frontocentral epileptiform discharges and diffuse slowing of consistent suggestive of a moderate encephalopathy but no electrographic seizures.
- MRI was unable to be performed due to concerns about over-heating of VNS in an encephalopathic patient.

Differential Diagnosis: neurodegenerative disease vs. autoimmune/paraneoplastic encephalitis vs CSF infection (i.e CJD).

Hospital Course

Day 1: Perampanel was stopped given mental status.
Day 2: Clobazam decreased to 60mg/day.
Day 5-9: 5 day course of empiric IVIG was given due to presumed autoimmune/paraneoplastic encephalitis.
Day 8: Level of alertness improved but was not able to follow commands.
Day 9: Clobazam decreased to 50mg/day.
Day 10: Clobazam decreased to 40mg/day.
Day 11: Clobazam decreased to 10mg/day.
Day 12-13: Patient was planned to receive Rituximab for suspected autoimmune encephalitis but the plan was delayed when he experienced an acute hypoxemic respiratory failure secondary to aspiration pneumonia and was transferred to MICU.
Day 19: Patient able to follow simple commands intermittently.
Day 22: Patient more alert and following commands. Speech mostly unintelligible. Decision to hold off Rituximab as potential side effect of AEDs was considered. Clobazam stopped.
Day 27: MRI Brain w/wo contrast showed changes consistent with right mesial temporal sclerosis and overall stable when compared to MRI completed 3 years prior.
Day 28: Speech much clearer. He consistently followed commands. He was able to participate with physical therapy. He asked for food.

Due to his improvement, rituximab therapy was not initiated. After 30 days, he was discharged with homehealth.

Further Laboratory and Diagnostic Workup

FIG. 1 ***Serum and CSF analysis***

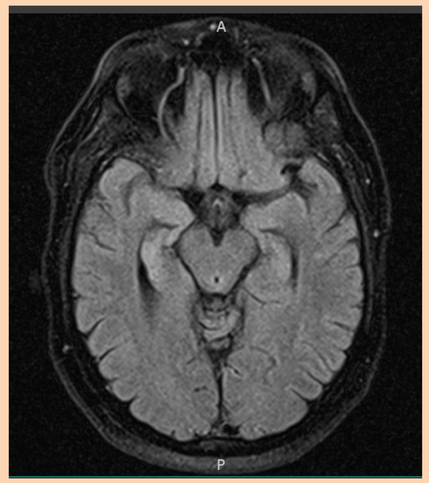
Tests	Results	Tests	Results
WBC, CSF	20	M. Tuberculosis DNA by PCP, CSF	Negative
RBC, CSF	10000	T. Gondii by PCP, CSF	Negative
Color, CSF	Red	EBV, CSF	Negative
Protein, CSF	54	Autoimmune Encephalopathy Panel, CSF	Negative
Glucose, CSF	75	Serum paraneoplastic panel	Negative
Enterovirus RNA by PCR, CSF	Negative	HIV	Negative
VDRL	Nonreactive	TSH	Within normal range
West Nile Virus by PCP, CSF	Negative	RPR	Nonreactive
Herpes Simplex by PCR, CSF	Negative		

FIG. 3 Vagal Nerve Stimulator Parameters

Tests	Normal	Autostim	Magnet
Output	1.5mA	M. Tuberculosis DNA by PCP, CSF	1.75mA
Frequency	30 Hz		
Pulse Width	500 microsec	500 microsec	500 microsec
On Time	30 sec	30 sec	60 sec
Off Time	1.1 min		
Duty Cycle	35%		
VDRL	Nonreactive		
West Nile Virus by PCP, CSF	Negative		
Herpes Simplex by PCR, CSF	Negative		

FIG. 2 ***Antiepileptic Drug Levels***

Tests	Result
Lacosamide	7.3
Clobazam	228.0
Desmethylclobazam (Active metabolite)	18440.0
Lamotrigine	6.1



MRI Brain FLAIR Sequence: increased T2 signal and subtle volume loss of right hippocampus similar in appearance to imaging done 3 years prior.

Conclusions

Leveraging medical record gives physicians access to more details about patient's healthcare but we should pay careful attention and avoid auto-populating data without independent and detailed history and assessment. At discharge, the cause of the patient's encephalopathy was thought to be more consistent with adverse effects of antiepileptic medications after extensive workup although an immune-mediated encephalopathy could not be completed excluded. The consideration of medication side effects became higher on differential after obtaining negative serum paraneoplastic and CSF encephalopathy antibody panels and stable MRI Brain. The delayed recovery in this case was thought to be due to the long half-life of clobazam and it's active metabolite, instead of treatment with IVIG. This case is an outstanding example of how medication side effects can disguise as complicated clinical presentations. In order to prevent such outcomes, we recommend to thoroughly review the details of cases especially in patients who have had a prolonged clinical course and several teams have been involved in patient care and to re-evaluate the case once more data becomes available.

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