In the name of god

Morning report

Neurology department

Razi hospital

94.4.31

۲۶مردان

اقای ۵۴ ساله کشاورزکه ۱۱ روز قبل حین فعالیت در حال ایستاده به طور ناگهانی دچار ضعف و کرختی اندامهای راست وانحراف صورت و اختلال تکلم شده بود و از عصر همان روز دچار اختلال بلع.سیر علایم ثابت بود .روز قبل از بستری دچار سردرد فشارنده ژنرالیزه و استفراغ شده بود

history

SH:Smoking 20 p/y

PMH: _

DH:Nortriptylin

Phenytoin

Piracetam

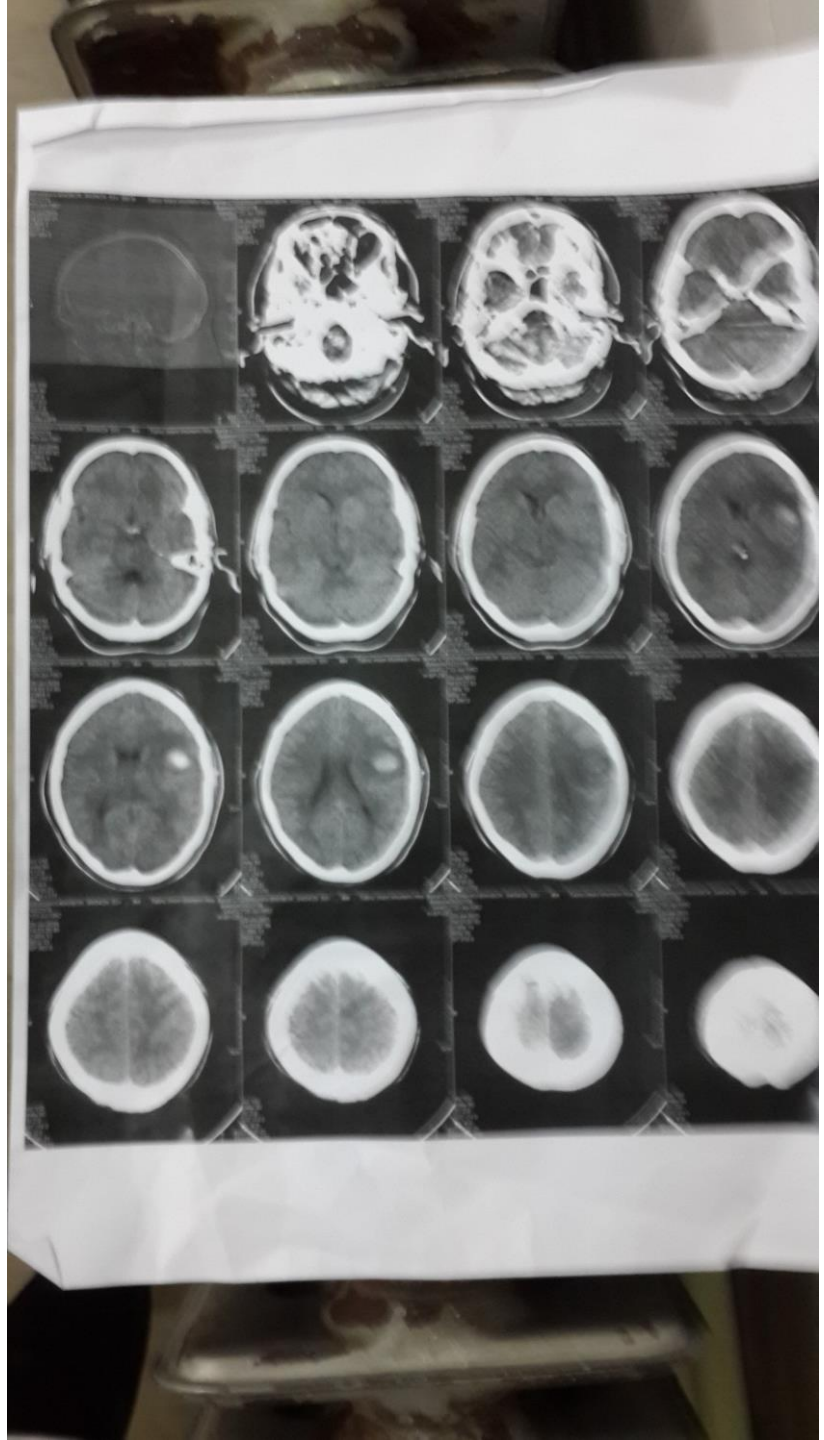
Phisycal exam

Bp:130\80 Pr:81 RR:14 BT:36.7
General exam :nl rythm:sinus cardiac:nl urticarial+
neurologic exam: awake /aware/
Dysartria+/ RCHFW +/Gag : -
MF Right side: 4/5 + DTR:2+ Double flex

Problem list

اقای ۵۴ ساله که ۱۱ روز قبل حین فعالیت در حال ایستاده به طور ناگهانی دچار ضعف اندامهای راست وانحراف صورت و اختلال تکلم شده بود و از عصر همان روز دچار اختلال بلع.

دیس ارتری اختلال بلع فلج فاسیال همی پارزی راست۴/۵+



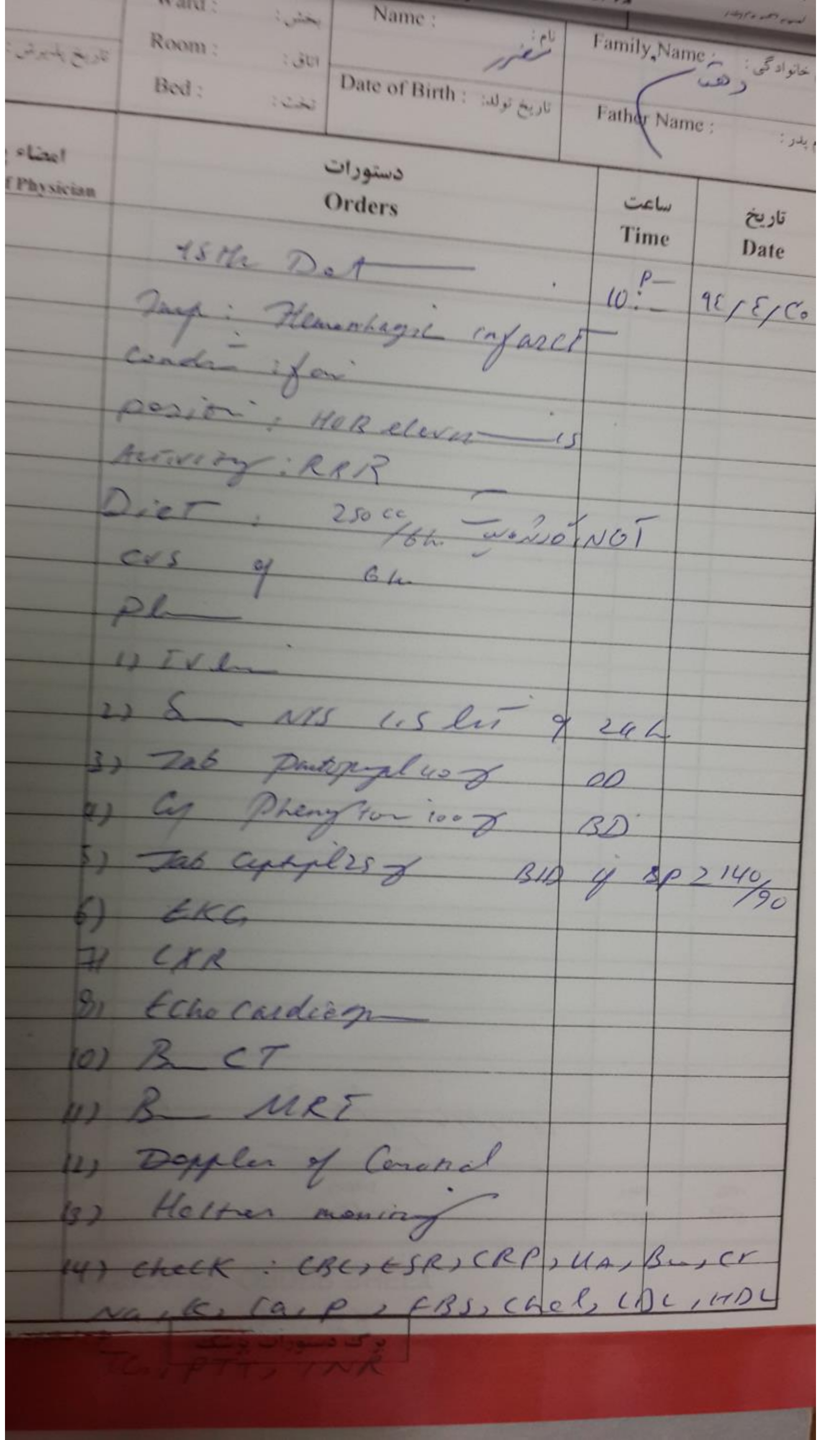
Ddx:

Embolic ischemic stroke (red infarct)

Sol(apoplexy of tumor)

AVM

Lobar ich


Ward : بخش :
Room : اتاق :
Bed : تخت :
تاریخ پذیرش :
Name : نام :
Date of Birth : تاریخ تولد :
Family Name : نام خانوادگی :
Father Name : نام پدر :

امضاء پزشک Signature of Physician	دستورات Orders	ساعت Time	تاریخ Date
	4SMr Dat	10 P	۹٤/٤/۲۰
	Imp: Hemorrhagic infarct		
	Condition: fair		
	position: HOB elevation 15		
	Activity: RRR		
	Diet: 250 cc/6h آب گوشت و میوه NGT		
	C&S of 6h		
	plan		
	1) IV line		
	2) Serum N/S 1.5 lit / 24h		
	3) Tab Pantoprazole 40 mg QD		
	4) Cap Phenytoin 100 mg BD		
	5) Tab Captopril 25 mg BID if BP ≥ 140/90		
	6) EKG		
	7) CXR		
	8) Echocardiogram		
	10) Brain CT		
	11) Brain MRI		
	12) Doppler of Carotid		
	13) Holter monitoring		
	14) check: CBC, ESR, CRP, UA, Bun, Cr, Na, K, Ca, P, FBS, Chol, LDL, HDL, PT, PTT, INR		

برگ دستورات پزشک

۱۶زنان

خانم ۵۶ ساله که ۱ هفته قبل از صبح دچار تهوع و استفراغ شده و از شب دچار سردرد شده که طی مراجعه سر پایی و درمان علامتی مختصری بهبود یافته.از فردای همان روز بیمار بیحالی و سستی ژنرالیزه داشته وازروز بعد دچار خواب الودگی شده است.در تمام این مدت از سردرد تمپورال راست شاکی بوده و تهوع گهگاهی داشته.سرگیجه گهگاهی را ذکر میکرده.ضعف اندام ها-تاری دید- تشنج نداشته

history

PMH : -

DH : OCP

FH: -

SH: -

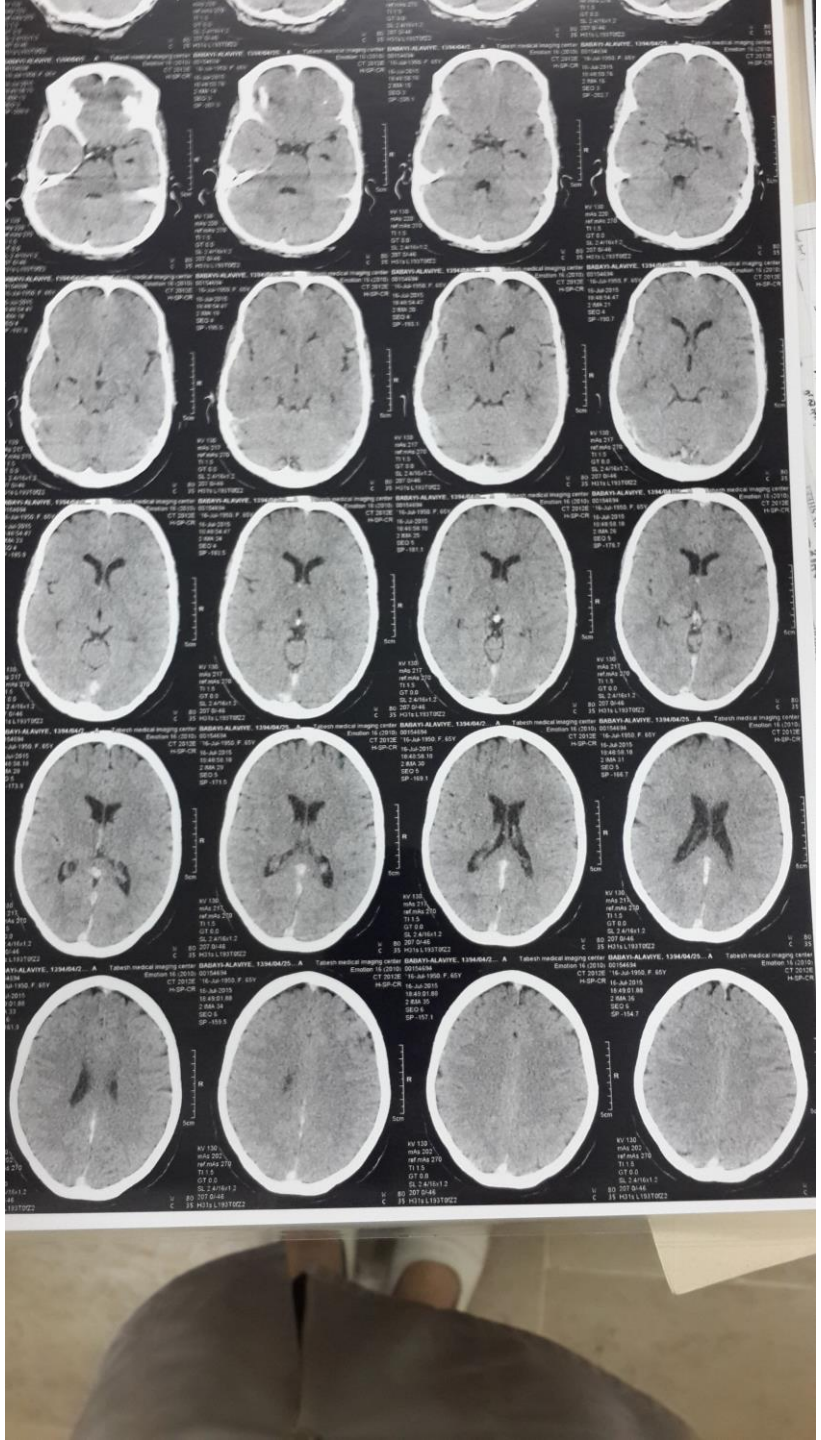
Physical exam

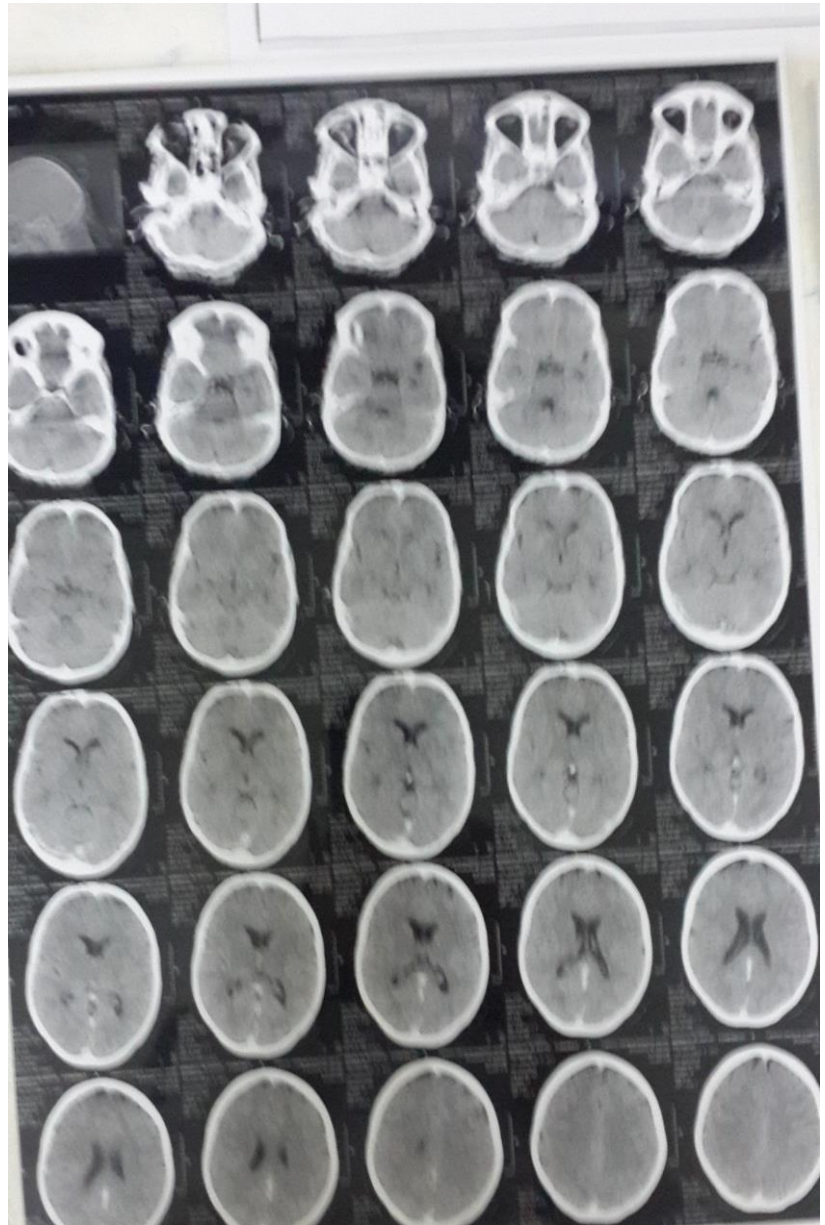
BP:150/90 PR: 85 RR:13 BT:36.5

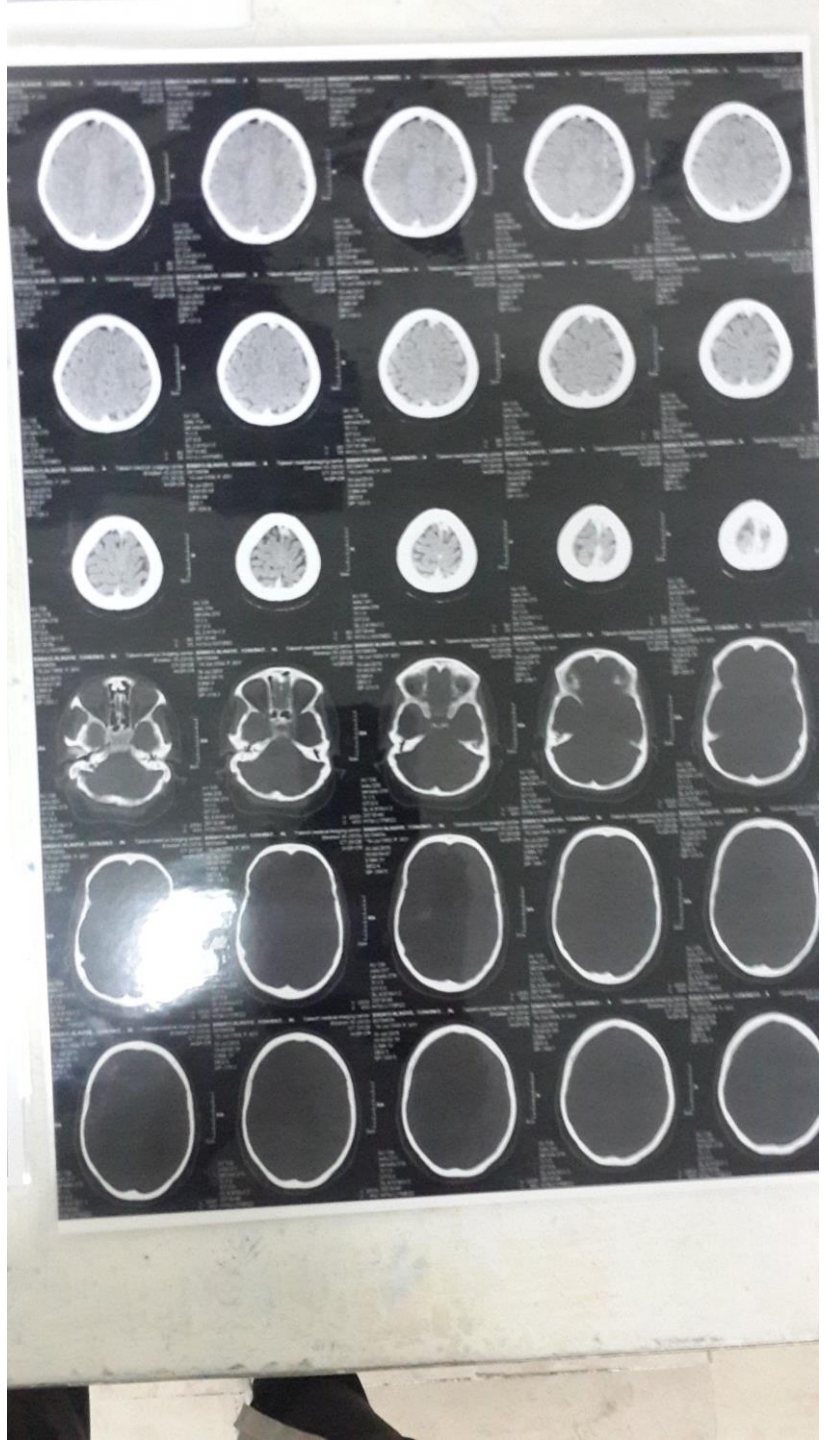
Lethargic

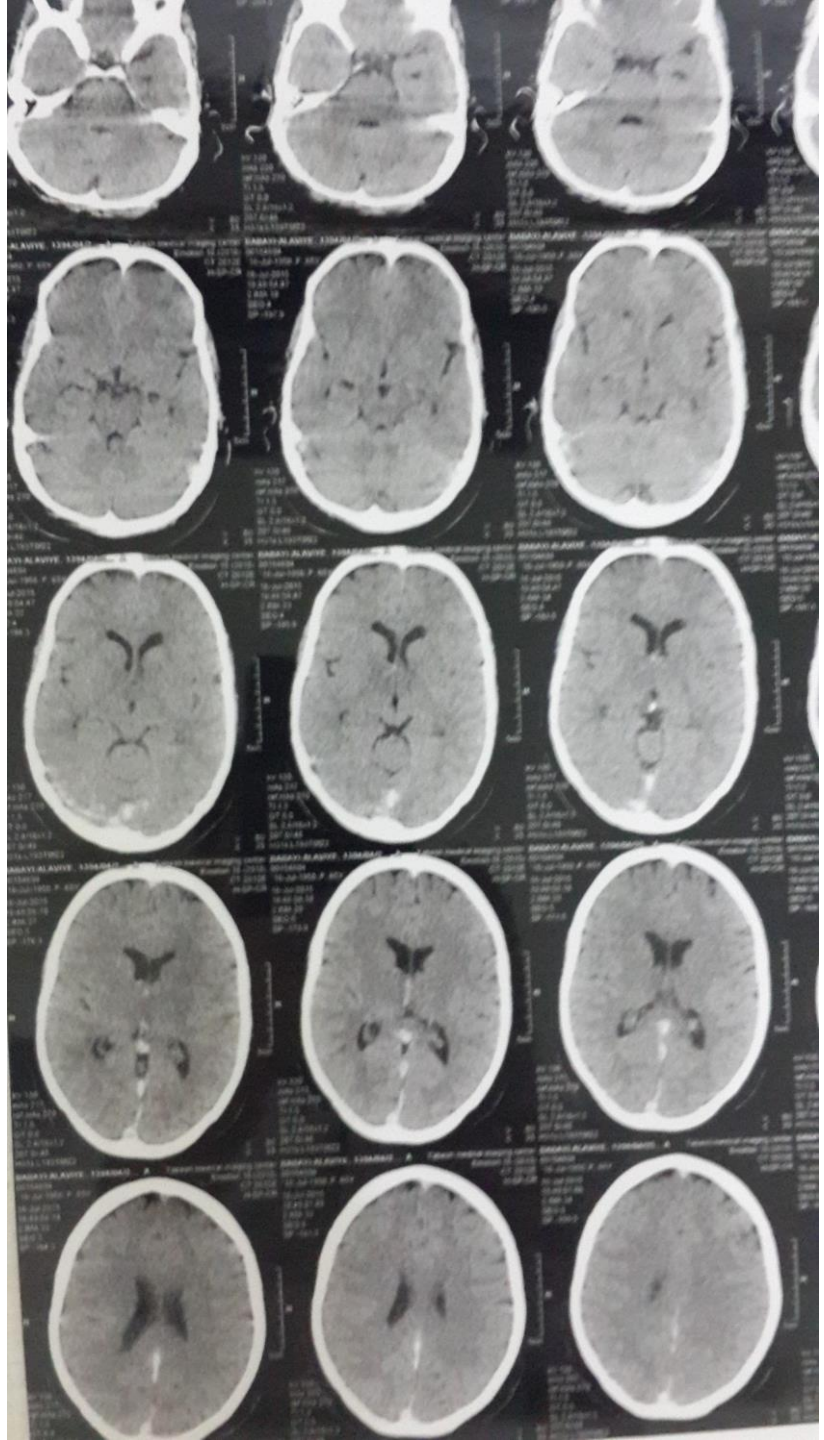
Papil edema grade 2

Mf: 5/5 DTR:2+ double flex









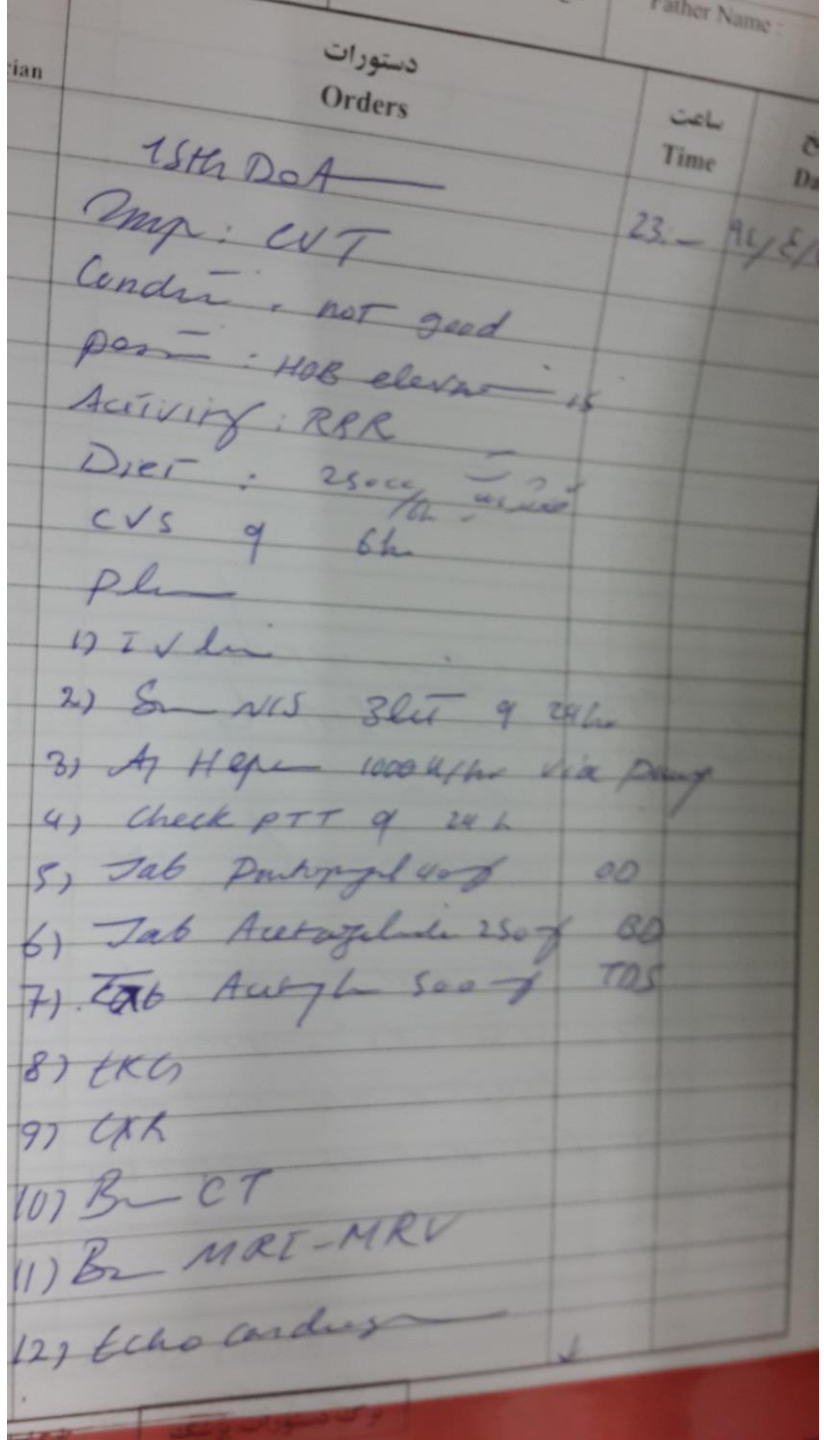
DDX:

CVT

SAH

IIH

Comlicated migrane



دستورات
Orders

ساعت
Time

Father Name :

15th DoA

Dmp : CVT

Condn : not good

pos : HOB elevation is

Activity : RBR

Diet : 250cc/hr

CVS q 6h

Plan

1) IV line

2) Sin NS 3lit q 24hr

3) Inf Hep 1000U/hr via pump

4) Check PTT q 24h

5) Tab Pantoprazol 40mg OD

6) Tab Acetazolamide 250mg BD

7) Tab Acetyl 500mg TDS

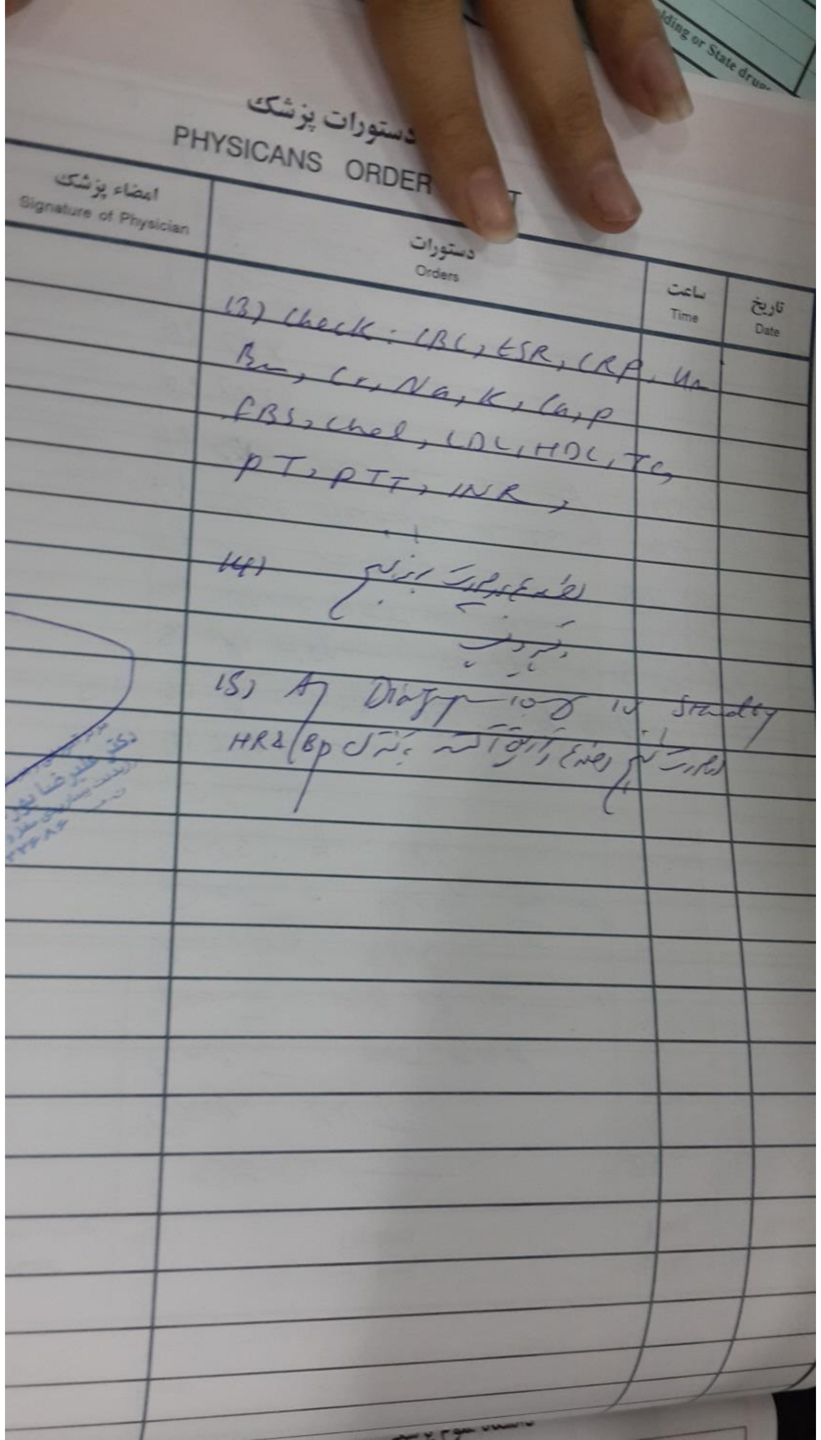
8) EKG

9) CXR

10) Brain CT

11) Brain MRI-MRV

12) Echo cardiography



دستورات پزشک

PHYSICANS ORDER [illegible]

امضاء پزشک Signature of Physician	دستورات Orders	ساعت Time	تاریخ Date
	(3) check: CBC, ESR, CRP, Ur, Bun, Cr, Na, K, Ca, P, FBS, Chol, LDL, HDL, TG, PT, PTT, INR,		
	(4) [illegible]		
	(5) [illegible] Diazepam [illegible] Standby		
[illegible]	HR & BP [illegible]		

۲۰ زنان

خانم ۵۱ ساله /اهل و ساکن بوکان/خانه دار/دارای ۳ فرزند

شکایت:بی ربط گویی

شرح بیماری

خانم ۵۱ ساله که ۱ هفته قبل دچارتب و لرز و تهوع و استفراغ و بی ربط گویی شده و از همانشب سردرد به علایم بیمار اضافه شده .از روز دوم تب و لرز تشدید یافته.سردرد فرونتال که به مسکن پاسخ نمیداده و علی رغم مراجعه متعدد و درمان علامتی سیر بیماری رو به پیشرفت بوده.بیمار به تدریج دچار عدم شناخت اطرافیان شده و از ۲روز قبل وسواس شستشو پیدا کرده.افت محتوای هشیاری سیر نوساندار داشته.سردرد و تهوع از ۳روز قبل قطع شده.تشنج و افت سطح هشیاری نداشته است.سرماخوردگی اخیر نداشته است

history

PMH: Asthma

DH: OCP

SPRAY SALBUTAMOL

FH: -

Physical exam

BP: 110/70 PR:150 RR:20 BT:37.2

GENERAL EXAM: cardiac :nl lung: weez

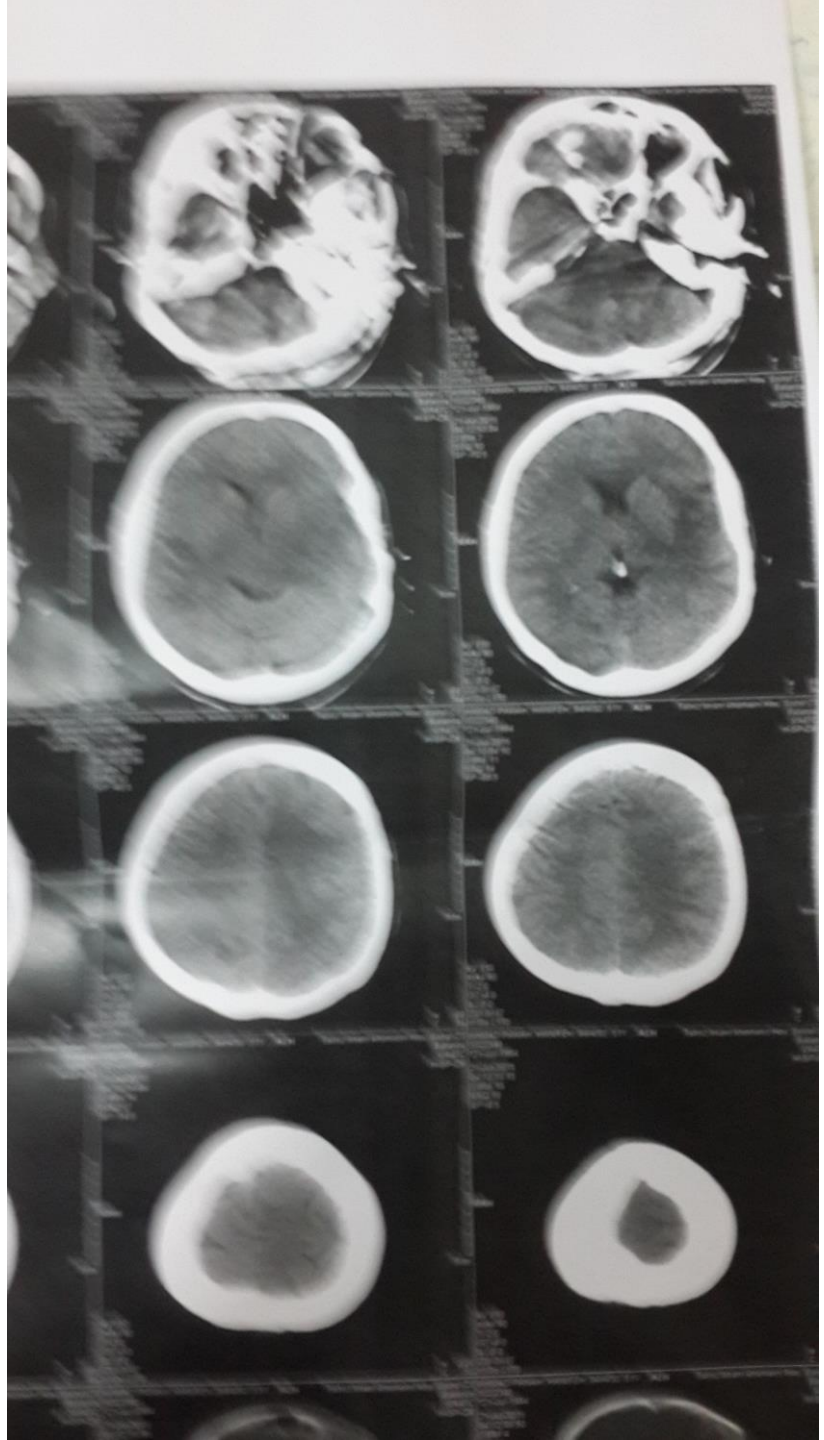
Arrytmia : - pulsation : 2+

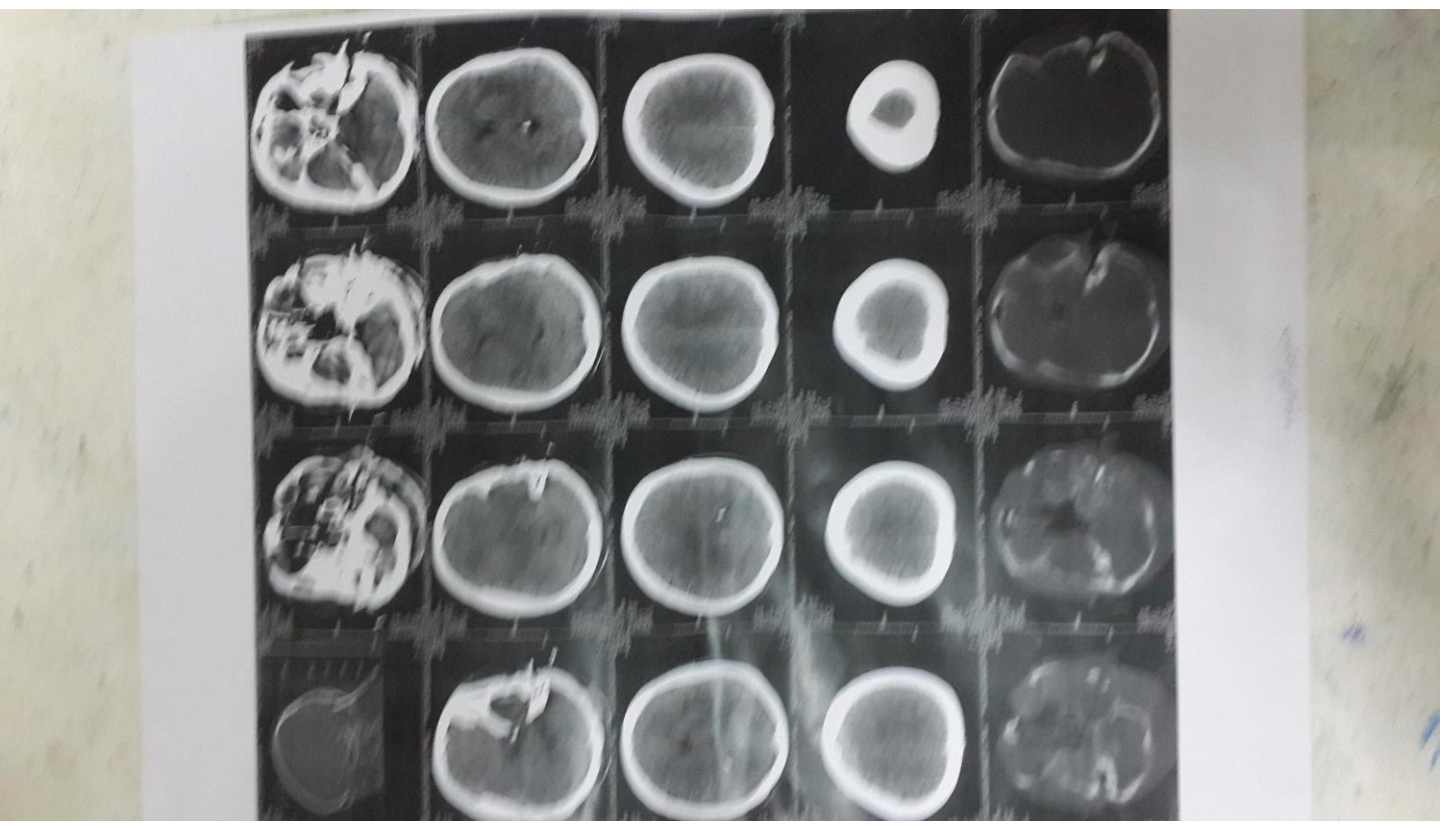
Neurologic exam

Awake/disoriented/
eye movement : nl/sharp disk venus puls +/
Pupils :4 mm reactive
Cranial : intact
MF:5/5 DTR:2+ DOUBLE FLEX
Nuchal rigidity +/-

خلاصه

خانم ۵۱ ساله که ۱هفته قبل دچارتب و لرز و تهوع و استفراغ و بی ربط گویی شده و از همانشب سردرد به علایم بیمار اضافه شده.بیمار به تدریج دچار عدم شناخت اطرافیان شده و از ۲روز قبل وسواس شستشو پیدا کرده.افت محتوای هشیاری سیر نوساندار داشته./تاکیکارد/دیس اریانته/ردور گردنی مشکوک



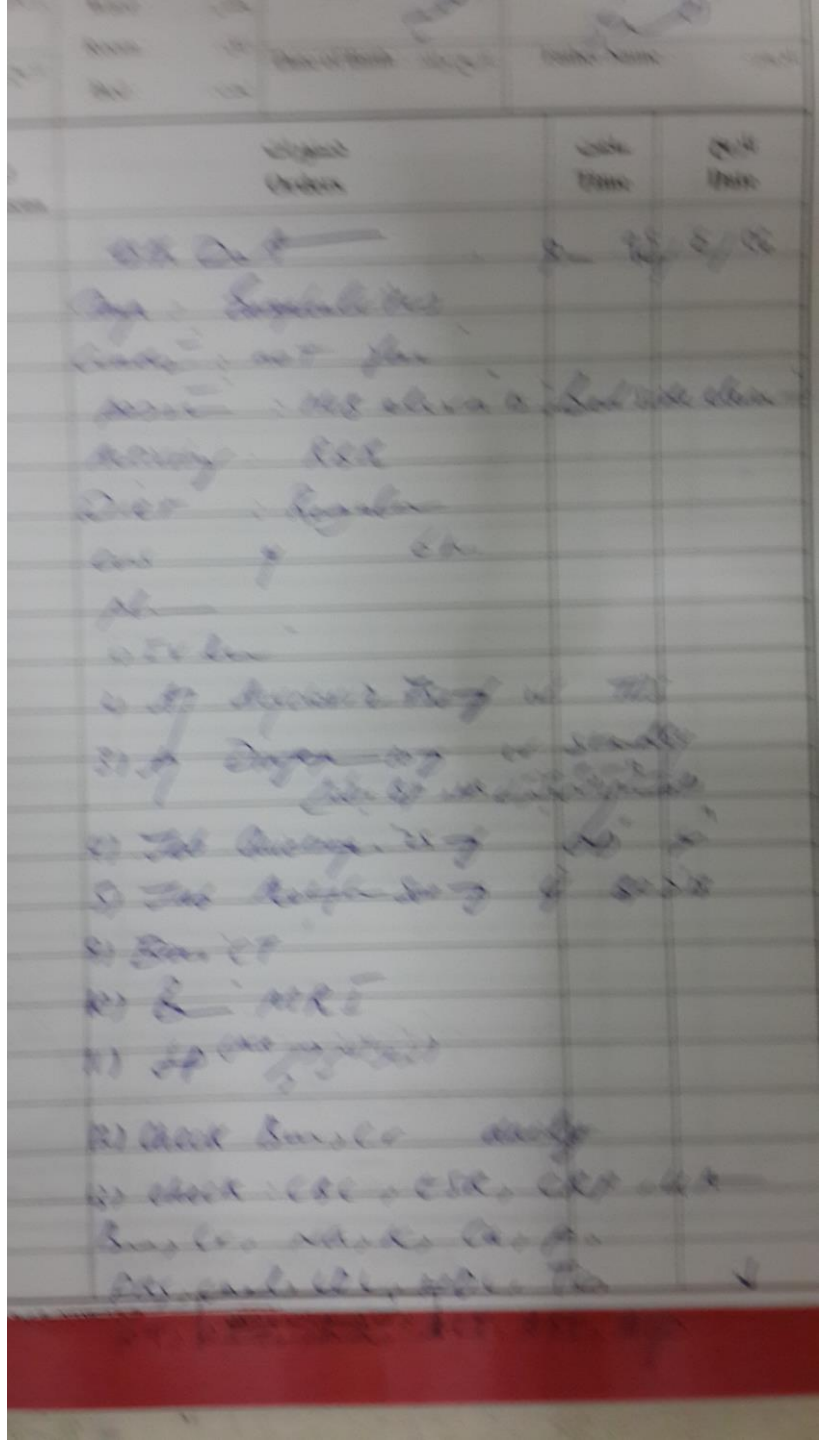


DDX:

Herpes encephalitis

ADEM

Drug toxicity



Herpes simplex virus type 1 encephalitis

INTRODUCTION — Herpes simplex virus type 1 (HSV-1) encephalitis is the most common cause of sporadic fatal encephalitis worldwide. The , clinical syndrome is often characterized by the rapid onset of fever headache, seizures, focal neurologic signs, and impaired consciousness

Epidemiology

HSV-1 encephalitis is the most common cause of fatal sporadic encephalitis in the United States, accounting for approximately 10 to 20 percent of the 20,000 annual viral encephalitis cases . The infection arises in all age groups, with one-third of all cases occurring in children and adolescents

PATHOGENESIs

immediate CNS invasion via the trigeminal nerve or olfactory tract following an episode of primary HSV-1 of the oropharynx; most patients with primary infection are younger than 18 years of age

murine model has shown that inoculation of HSV into the murine tooth pulp leads to an encephalitis primarily affecting the temporal cortex and limbic system

This manner of inoculation selectively infects the mandibular division of the trigeminal nerve, which is most common site of viral latency in humans with HSV-1 infections

Another possible mechanism of nervous system invasion is viremia. HSV has been identified by culture in the blood of neonates and immunocompromised patients and, by polymerase chain reaction

It is unclear whether the extent of CNS viral load is directly related to the severity of tissue damage. In one study of eight patients with HSV encephalitis, the level of HSV-1 viremia in CSF, as determined by PCR, did not correlate with the severity of clinical signs

CLINICAL FEATUREs

Symptoms and signs — Focal neurologic findings are usually acute (<1 week in consciousness, focal cranial duration) and include altered mentation and level of nerve deficits, hemiparesis, dysphasia aphasia, ataxia, or focal seizures Over 90 patients will have one of the above symptoms plus fever . Other percent of ,associated neurologic symptoms include urinary and fecal incontinence meningitis, localized dermatomal rashes, and Guillain-Barré

Later in the clinical course, patients may have diminished comprehension, paraphasic spontaneous speech, impaired memory, and loss of emotional control

Various behavioral syndromes have been reported in association with HSV-1 encephalitis including: Hypomania

Varying states of amnesia

HSV-1 CNS infection has also been implicated in cases of recurrent brainstem encephalitis

Laboratory abnormalities

Examination of the CSF typically shows a lymphocytic pleocytosis, increased number of erythrocytes (in 84 percent of patients), and elevated protein.

Repeat testing can be helpful whenthe clinical suspicion is high. Low glucose is uncommon and may suggest an alternative diagnosis

Imaging studies

Temporal lobe abnormalities on brain imaging areconsidered strong evidence for herpes simplex encephalitis. Temporal lobe lesions are predominantly unilateral and may have associated masseffect

MRI is the most sensitive and specific imaging method for HSV encephalitis, especially in the early course of the disease Brain perfusion

SPECT studies of patients with viral encephalitis and CSF antibodies to HSV have shown increased accumulation of radiotracer in the affected temporal lobe

EEG

(Electroencephalogram (EEG) — Focal electroencephalogram (EEG findings occur in >80 percent of cases, typically showing prominent , intermittent high amplitude slow waves (delta and theta slowing), and occasionally, continuous periodic lateralized epileptiform discharges in the affected region

It is extremely important that the diagnosis of HSV encephalitis be , entertained early in any patient who presents with suggestive signs symptoms, laboratory, and imaging studies since it is among the more treatable of the infectious etiologies of encephalitis

DIAGNOSIS

Polymerase chain reaction — The gold standard for establishing the

diagnosis is the detection of herpes simplex virus DNA in the CSF byPCR

Prior to the availability of PCR testing, brain biopsywas considered to be the only way to accurately diagnose herpes encephalitis

CSF antigen and antibody determinations are not helpful in the early diagnosis

TREATMENT

empirictherapy with IV acyclovir (10 mg/kg IV every 8 hours) should be initiated as soon as the diagnosis is considered

Early, aggressive antiviral therapy can prevent mortality and limitthe severity of chronic post encephalitic behavioral and cognitive impairments

PROGNOSIS

Untreated, the fatality in herpes encephalitis canapproach 70 percent and most of the survivors have serious neurologic deficits . Survivors can also have significant neuropsychiatric and neurobehavioral issues

Even with appropriate diagnosis and treatment, mortality may still be

as high as 20 to 30 percent

THANKS FOR YOUR ATTENTION