Case conference

報告者:R1 施膺泰
指導者:VS吳柏衡
05.20.2014

The patient

- 55-year-old man
- 13:47 DAY1
- T/P/R: 36.7°C/73/19
  BP 151/77mmHg SpO2 98%
  E4VAM4
- Chief complaint: 意識程度改變
- Triage: 1

Present illness

- 今天回診追蹤甲狀腺機能亢進
- Sudden onset of dizziness
- Conscious change
- Vomiting(+)

Past history

- Hyperthyroidism
- NKDA

Physical examination

- E3V1M5
- Head and neck: not pale
- Chest: bilateral clear breathing sound
- Abdomen: soft
- Extremities: warm
- NE: pupil size (L)3+ (R)3+

Impression? What to do next?

Impression

- CVA r/o ICH
- 啟動tPA

13:50

- On monitor
- N/S run 60ml/hr
- CBC/DC/Plt
- Crea, AST, Tro-I, ammonia
- VBG7
- PT,aPTT
- Brain CT without contrast
- 啟動tPA

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VBG7	
pH	7.379
pO2	54
pCO2	42.8
HCO3	25.3
Na	143
K	3.9
iCa	1.28
Hb	16.7

15:20 (?)

- Bedside echo
  - No pericardial effusion
  - No AAA/ aortic flap
- Brain CT: no ICH

14:20

- Neurologist suggest brain CTA
  - 病人有hyperthyroidism兩年未用藥
  - 補單free T4, TSH, alcohol
- Neurologist tPA for suspected BAO
  - 家屬拒絕

Neuro consultation

S	Sudden onset of dizziness and conscious disturbance	
O	E1V1M4 Pupils: 2+/2+ Ocular dipping (bobbing?) VOR (-) Cough reflex (+)	Muscle power: >3/>3 DTR: no increase Babinski sign: bilateral withdraw Sensory: grossly normal Coordination, gait: cannot cooperate
A	Acute conscious disturbance R/O brainstem stroke R/O metabolic encephalopathy NIHSS=20	
P	家屬拒絕tPA治療 Bokey 3# PO ST MgO 1# PO ST Follow cortisol, iCa, Mg, urine toxin screen 急作brain MRI (only T2+DWI) Arrange TCD+ECD	

15:10

- Primperan 1amp IV ST
- NaHCO3 3amp IV ST
- Vena 1amp IV ST
- Brain MRI without contrast
- BZD, Mg, cotisol

Lab data

Hb	WBC (N/L)	Plt	PT (INR)	aPTT	
15.8	11100 (43/43)	313000	10.6 (0.98)	30.5	
AST	Crea	Troponin-I	Ammonia		
22	0.76	0.01	37		
Alcohol	TSH	Free T4	Mg	BZD	Cortisol
negative	<0.0025	3.07	2.0	<3.0	28.7

Brain MRI

Brain MRI

- Recent infarctions presenting as hyperintense DWI signal in left paramedium of pons.
- MRA shows high grade stenosis or occlusion of the basilar artery. Bilateral PCAs were perfused from the PComA.
- Impression：C/W occlusion or high grade stenosis of the basilar aretery with infarct at pons.

17:30

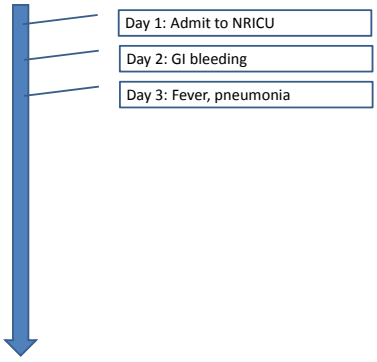
17:37

- CxR
- ECG
- Admit to NRICU

18:00

- 入ICU
  GCS: E3V2M6 坐起來會暈 (罵髒話)
- 告知家屬目前尚不需插管
  但意識狀態若變差可能有需要

Hospital course



DISCUSSION
1.BASILAR ARTERY OCCLUSION
2.HYPERTHYROIDISM VS ENHANCED CT

Basilar artery occlusion. *Lancet Neurol*. 2011;10:1002–1014.

BASILAR ARTERY OCCLUSION

Bisilar artery occlusion

- 1% of all strokes
- 8% of patients with symptomatic vertebrobasilar territory ischemia

Etiology

- Atherosclerosis 26-36%
- Embolism 30-35%
- Dissection 6-8%
- Undetermined 22-35%

Clinical manifestation

- Prodromal symptoms: vertigo, headache
- Medial pontine tegmentum- reticular activating system
- Paramedian tegmentum- oculomotor fibers
- Paramedian pontine base- long motor tracts, crossed cerebellar tracts

Differential diagnosis

	Ancillary investigations
Subarachnoid haemorrhage	Head CT, CT or MR angiography, CSF
Non-convulsive status epilepticus or postictal state	Clinical findings, history, EEG
Hypoglycaemic or other metabolic coma, intoxication	Clinical findings, history, blood chemistry, EEG
Hypoxic-ischaemic encephalopathy	Clinical findings, history, ECG, EEG, diffusion-weighted MRI
CNS infection (meningitis, encephalitis)	Clinical findings, cerebrospinal fluid, MRI
Bilateral hemispheric stroke	Head CT or MRI, carotid ultrasound, cardiac ultrasound
Cardiogenic or haemorrhagic circulatory shock	Haemodynamics, ECG, chest radiography, cardiac ultrasound
Guillain-Barré syndrome or cranial neuritis, Miller-Fisher syndrome, Bickerstaff encephalitis, botulism, myasthenic crisis	Clinical findings (reflexes), CSF, nerve conduction studies, serum antibodies
Basilar-type migraine	History, clinical course, MRI

MR=magnetic resonance. CSF=cerebrospinal fluid. EEG=electroencephalogram. ECG=electrocardiogram.

Table 3: Differential diagnosis of basilar artery occlusion and ancillary investigations, by disorder

Neuroimaging

- CTA
- MR
- Transcarnial Doppler
- Angiography

Management

- Antiplatelet agents
- IV thrombolysis
- IA thrombolysis
- Mechanical endovascular treatment
- Bridging therapy (abciximab/alteplase)

UpToDate

IODINE-INDUCED HYPERTHYROIDISM

Jod–Basedow effect

- 2-12 weeks after iodine exposure
- Risk factor:
  - Regions of iodine deficiency
  - Nodular goiter
  - Grave's disease
  - The elderly

Incidence?

- Only 2 of 788 unselected patients from an iodine deficient area developed hyperthyroidism within 12 weeks after coronary angiography
- In a prospective study of 73 patients (mean age 65.7 years), only two developed hyperthyroidism after exposure to radiographic contrast

Risk of iodine-induced thyrotoxicosis after coronary angiography: an investigation in 788 unselected subjects. Eur J Endocrinol. 1999;140(3):264.
A prospective study of the effect of nonionic contrast media on thyroid function. Thyroid. 1996;6(2):107.

THANKS FOR YOUR ATTENTION!!