What are the connections?

Dr CH Ng

ICU, KWH

23 March 2010

A Cardiologist with Guillian-Barre Syndrome

- 一名58歲私家男醫生，平安夜接種H1N1甲型流感（人類豬流感）疫苗後下肢乏力，懷疑患上「吉巴氏綜合症」，衛生署　專家小組召開會議研究事件。
- 專家在會後指，患者臨床徵狀暫與「吉巴氏綜合症」符合，但暫未發現與接種疫苗有關，衛生防護中心　會繼續跟進事件。
- 他又強調，全球已注射8000萬針甲流疫苗，只有50宗「吉巴氏綜合症」，危險性少於百萬分之一。

Case Presentation

F/67

NSND

Independent ADL

Past Health:

- HT on Adalat Retard and Atenolol, FU GOPC
- Hyperlipidaemia on diet
- Hysterectomy 10 yrs ago

P/E on admission

- Temp 38.4 ℃
- BP135/75, p 75/min regular, no murmur
- GCS 15/15
- Power UL: 5|5, LL: 5-|5-; areflexia
- No sensory deficit
- Chest: clear
- Abdomen: Unremarkable

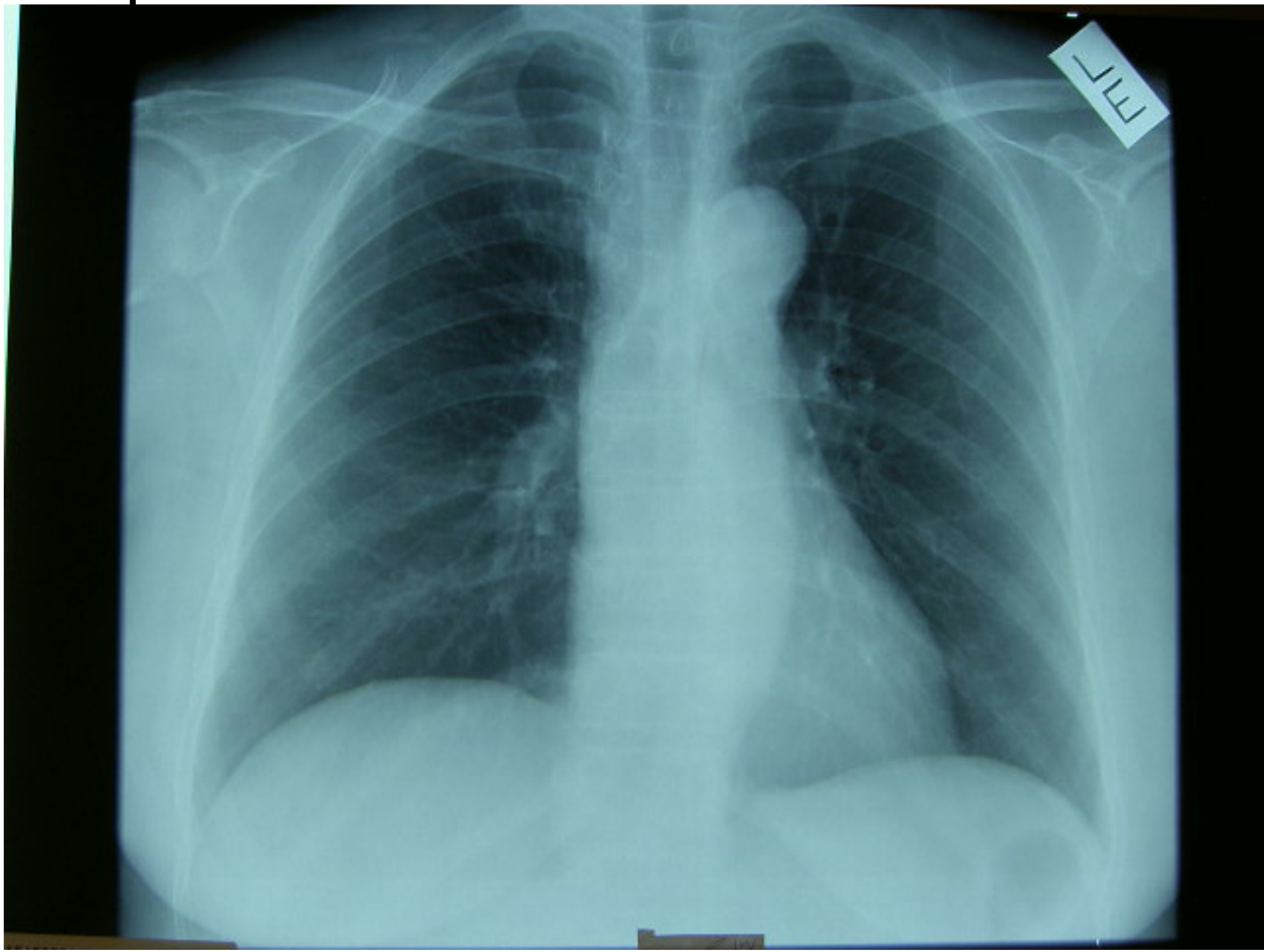
Provisional Dx:

? Guillain-Barré syndrome

Admission to Medical ward on 15/01/2010

- c/o generalized weakness x 3/7
- 4 limb weakness, with progressive worsening
- On and off colicky abdominal pain, nausea, fever, chills and rigors
- No diarrhoea, no SOB, mentally well
- TOCC-ve
- Received swine flu vaccination on 23/12/2009 in To Kwa Wan Clinic

From vaccination to onset of weakness: 25 days



- CXR: clear lung fields
- Sputum/urine/blood/stool culture
- CBP: WBC 9.5, Hb 13.8, PLT 156
- Normal liver and renal function tests

Rx: oral Augmentin empirically

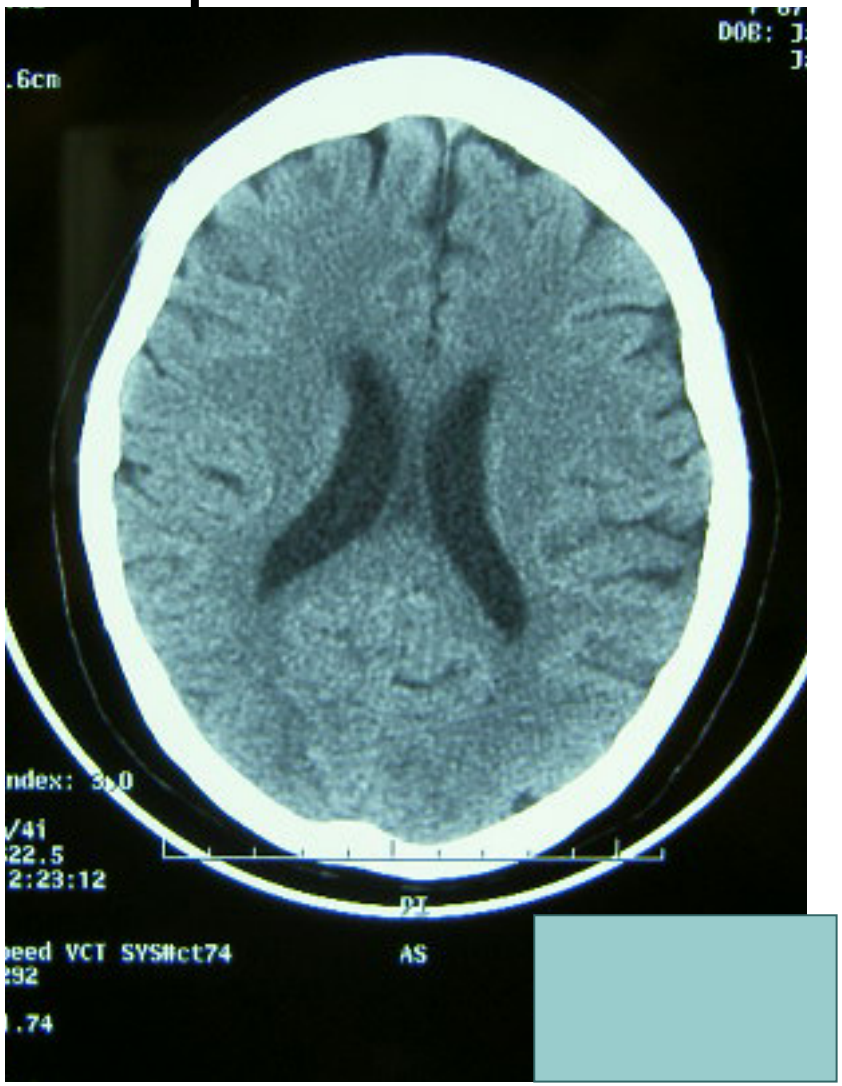
Same day of admission

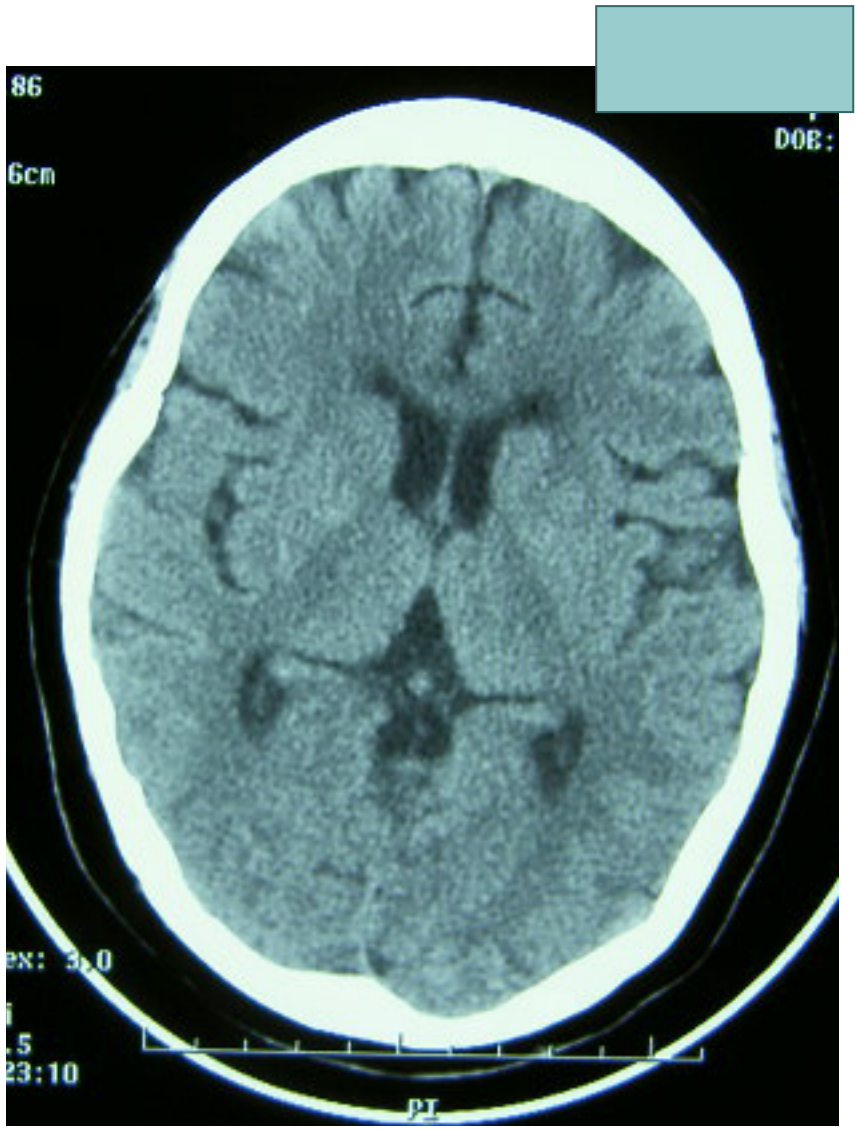
- NCT

  Absence of R. sural SNAP. Mild slowing of R. ulnar sensory conduction. Satisfactory R. ulnar motor and median sensori-motor conductions. Satisfactory R. peroneal and tibial conductions. No nerve conduction blocks were detected. Prolonged F-waves of R. tibial nerves
- Conclusion: the findings were not conclusive of GBS. Suggest to repeat NCT next week

- Persistent fever, vomiting+, changed to IV Augmentin

Jan 16





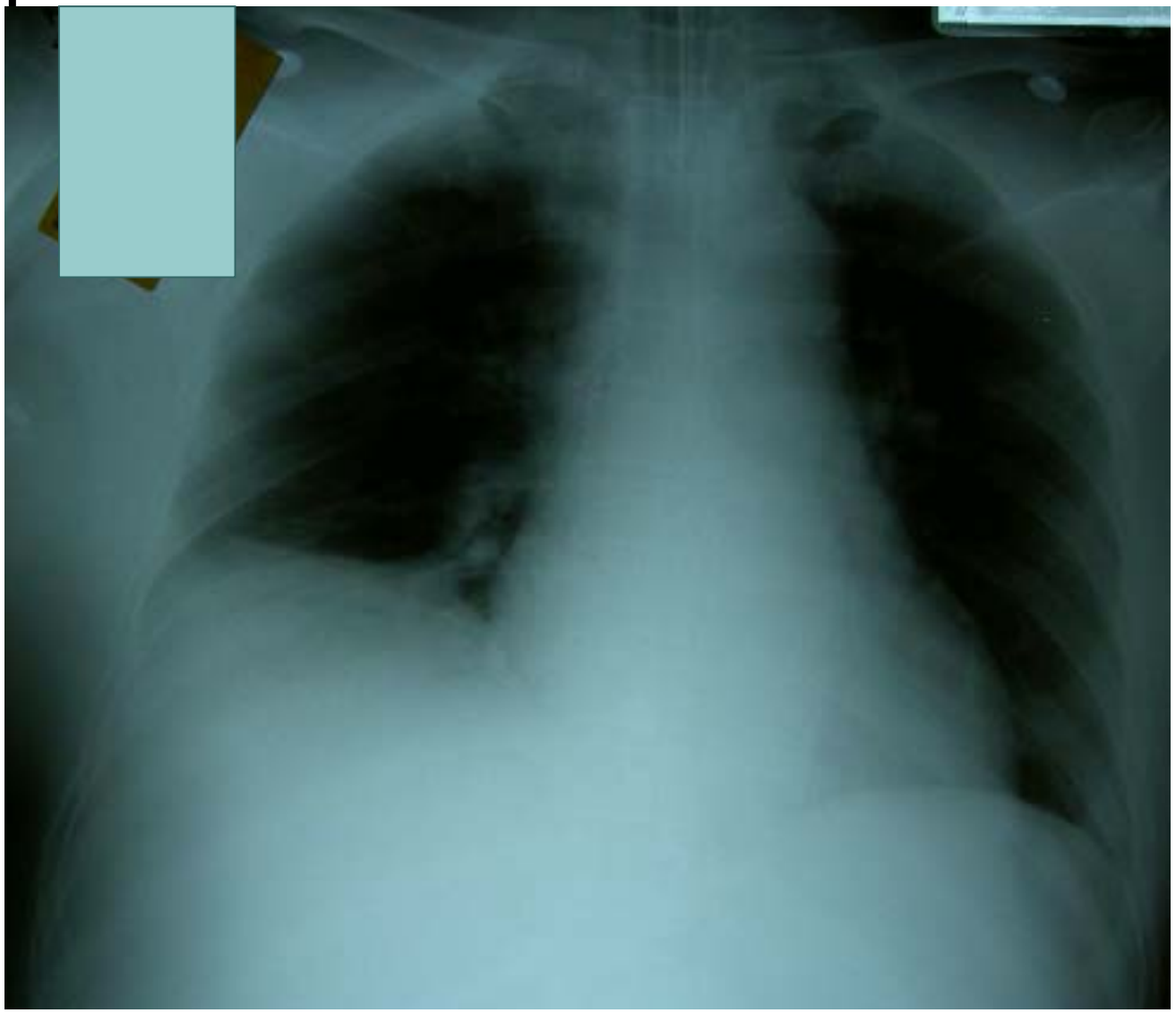
Power UL: 4|4, LL: 4|4

Worsening of muscle weakness

- Fever 39 ℃ , WBC 31.2
- Dyspnea & increasing O2 demand
- Limb power: UL: 2|3, LL: 2|2, areflexia, plantars equivocal
- Started Tazocin and Clarithromycin
- LP:
  - Opening pressure 5.5cmH2O, WBC 9/ml (N: 15%, L: 85%), no organism, culture –ve; CSF protein: 0.27g/L, glucose 12.7 (H'stix 22)
- Desaturation and confusion, slurred speech
- Elective intubation
- Low BP, requiring fluid resuscitation, Dopamine and Adrenaline

ICU admission (18/1)

- Clinically, in warm shock
- CXR: RLZ segmental collapse
- High inotropic supports
- Oliguric renal failure, started on CVVH
- Bedside echocardiogram: prominent RV, fair biventricular systolic function; USG: normal kidneys
- Discussion with neurologists, suspected GBS with rapid deterioration, started on IVIg



- Repeated NCT
  - Sensory nerve conduction could not be performed due to interference from machines. Motor nerve conduction of R. median, ulnar, PTN and CPN were within normal limits.
  - Not suggestive of GBS
  - Stop IVIG (after 400mg/kg x 2 days)

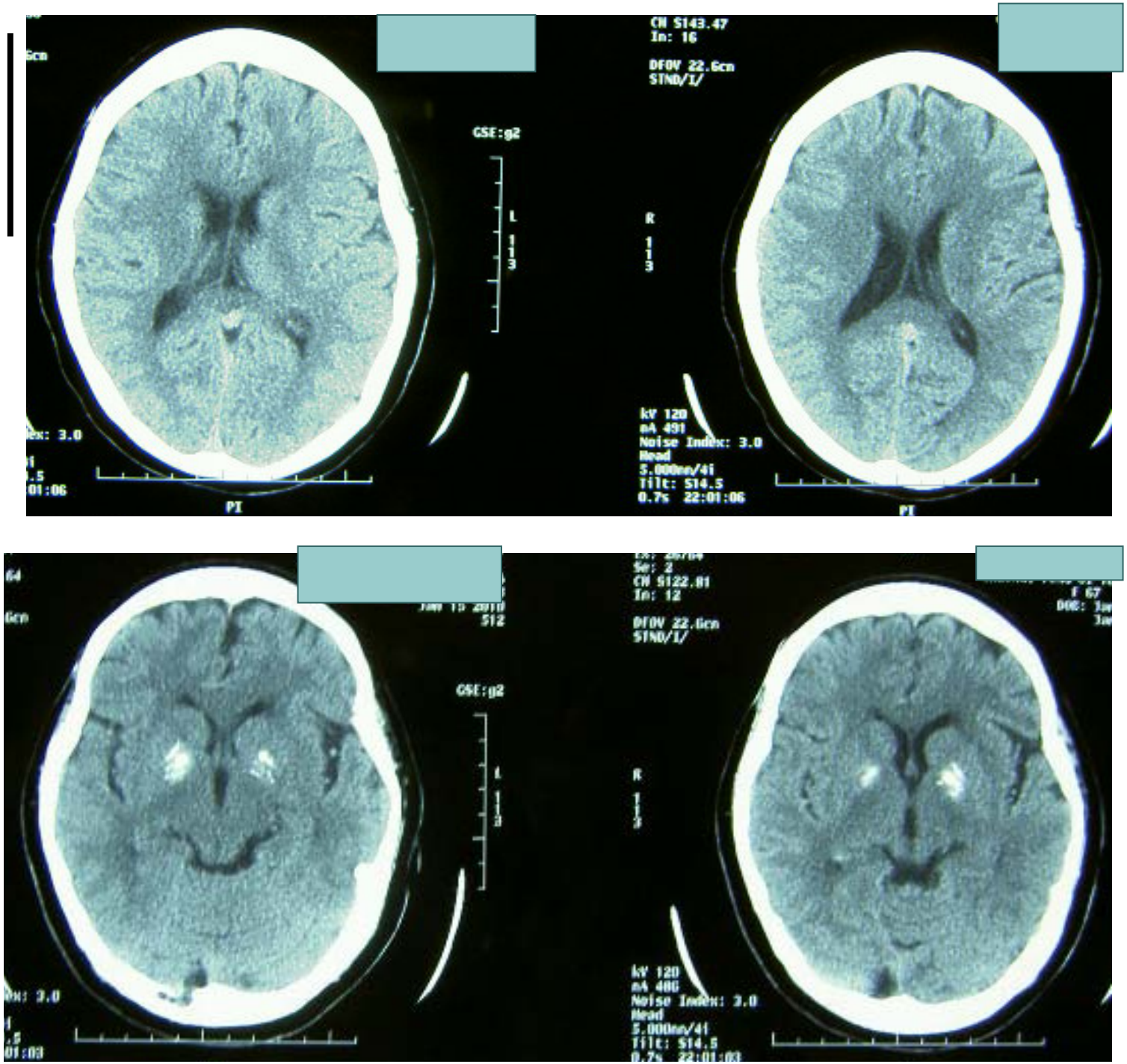
D2/3 in ICU

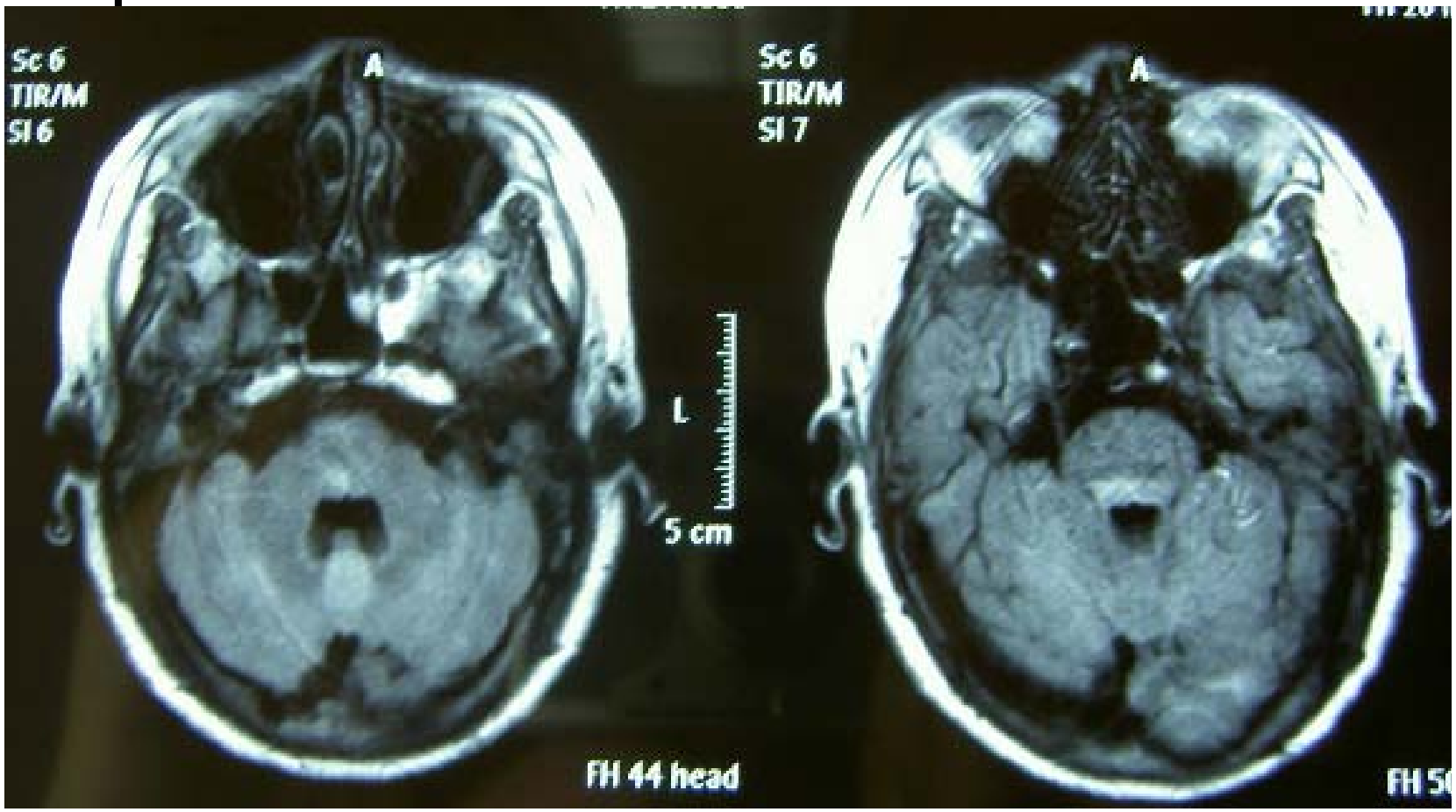
- Improving limb power and brisk jerks
- On ventilator with sedation, mentally still communicable (E2M5Vt)
- Fibreoptic bronchoscopy for RLL toileting, no microbial isolated in BAL
- Ventilator asynchrony, required sedation
- Dialysis dependent
- Hemodynamics improving & inotropes weaned off

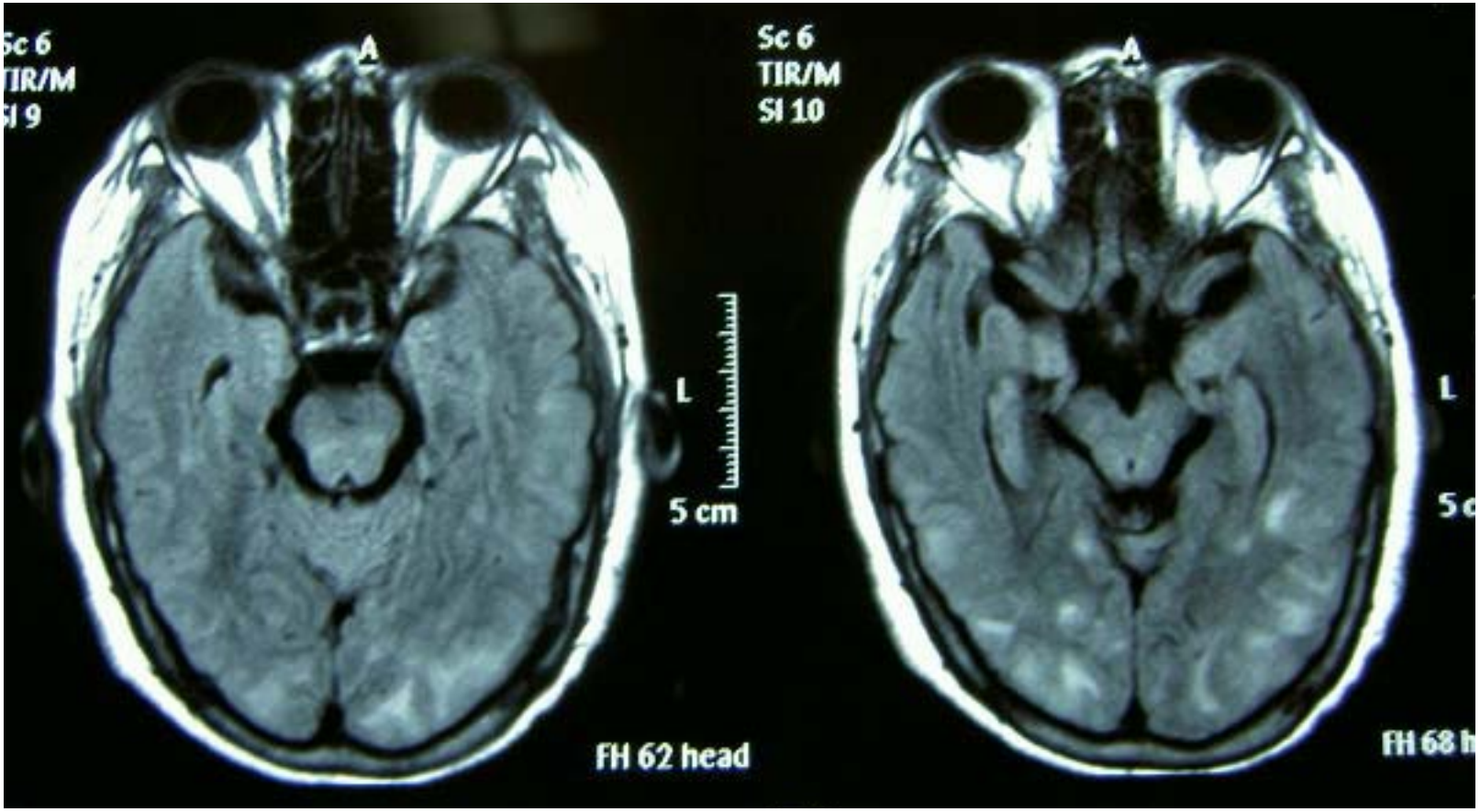
A week later…

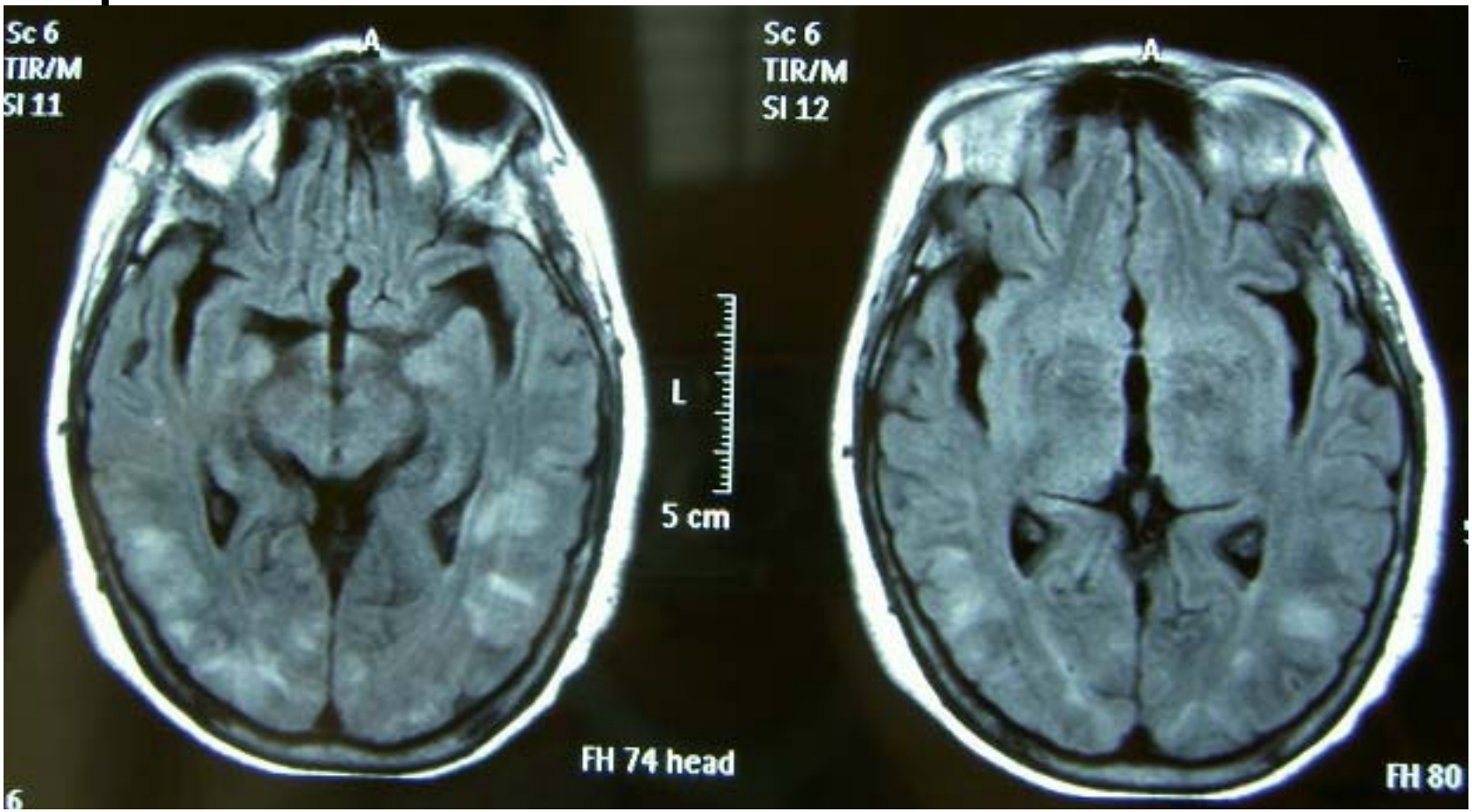
- Depressed sensorium despite cessation of sedation for a week
- Best GCS arousable but hypersomnolent
- E2M4Vt
- CT brain: unremarkable
- LP: nil WBC, CSF protein 0.34, glucose 7.7 (plasma 8.3)
- Plasma ammonia: 14 umol/L (10-47)

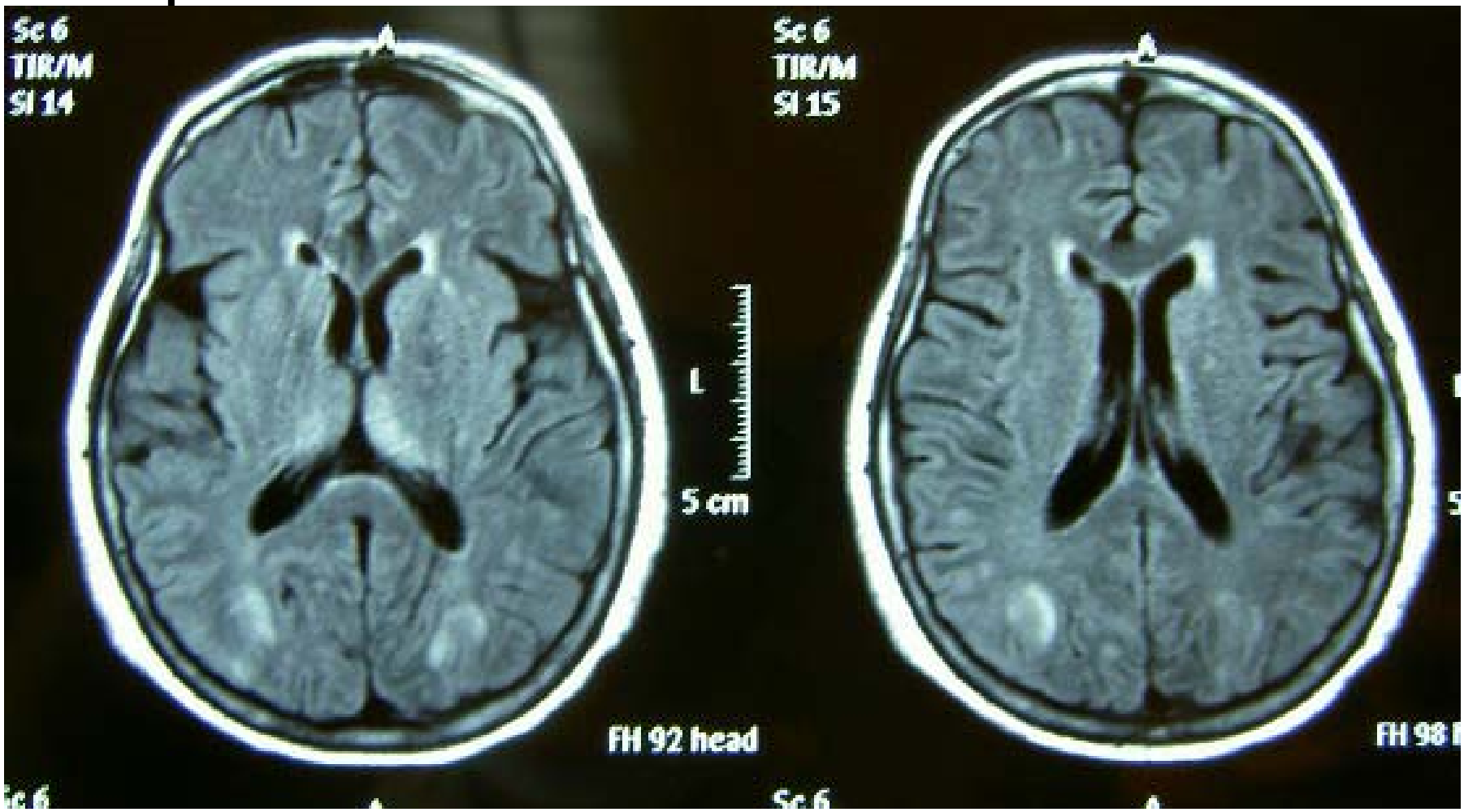
- MRI brain and cervical spine done

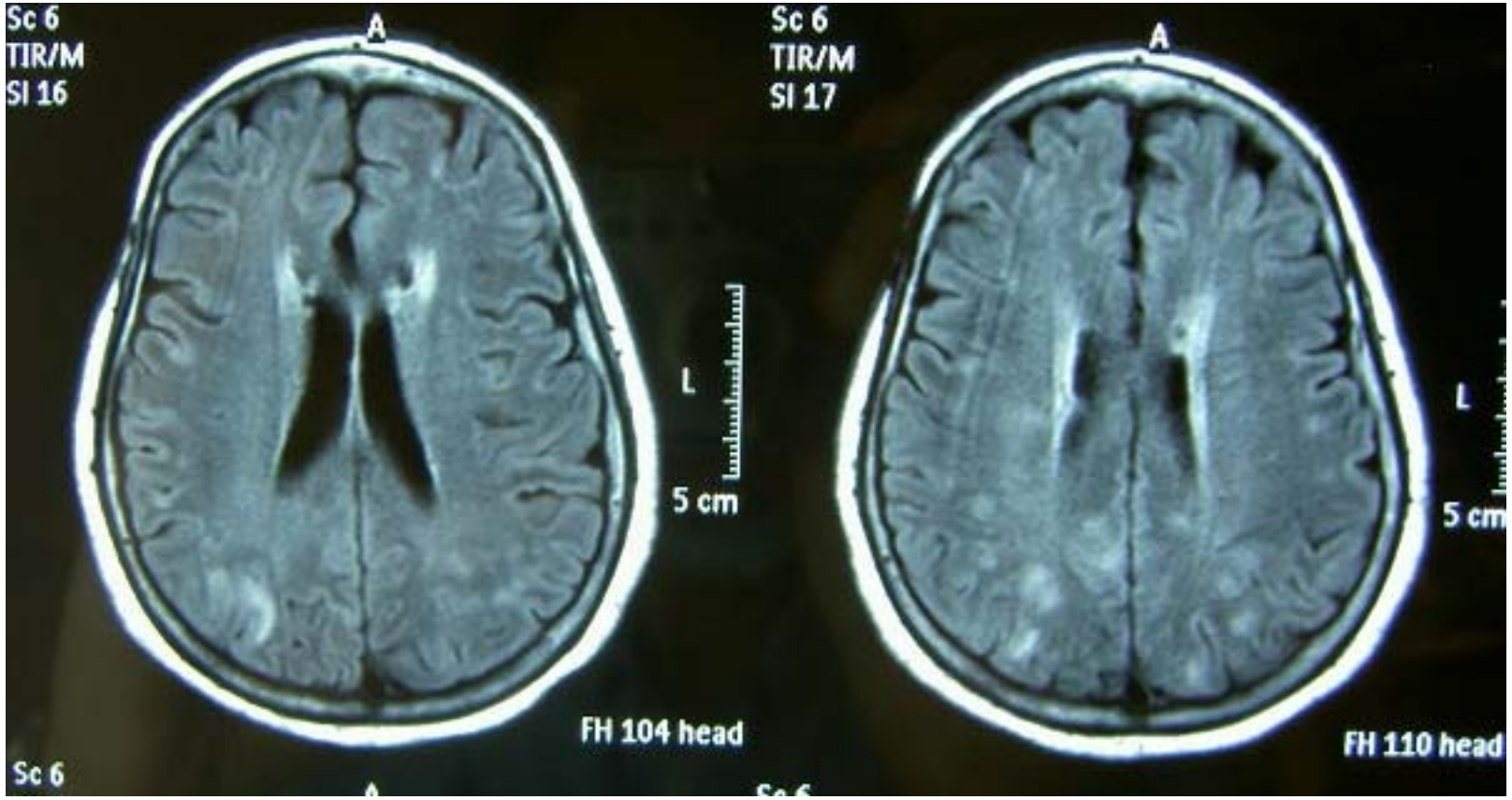

GSE:g2
PI
DFOV 22.6cm
STND/I/
kV 120
mA 491
Noise Index: 3.0
Head
5.000mm/4i
Tilt: S14.5
0.7s 22:01:06
PI
GSE:g2
DFOV 22.6cm
STND/I/
kV 120
mA 486
Noise Index: 3.0
Head
5.000mm/4i
Tilt: S14.5

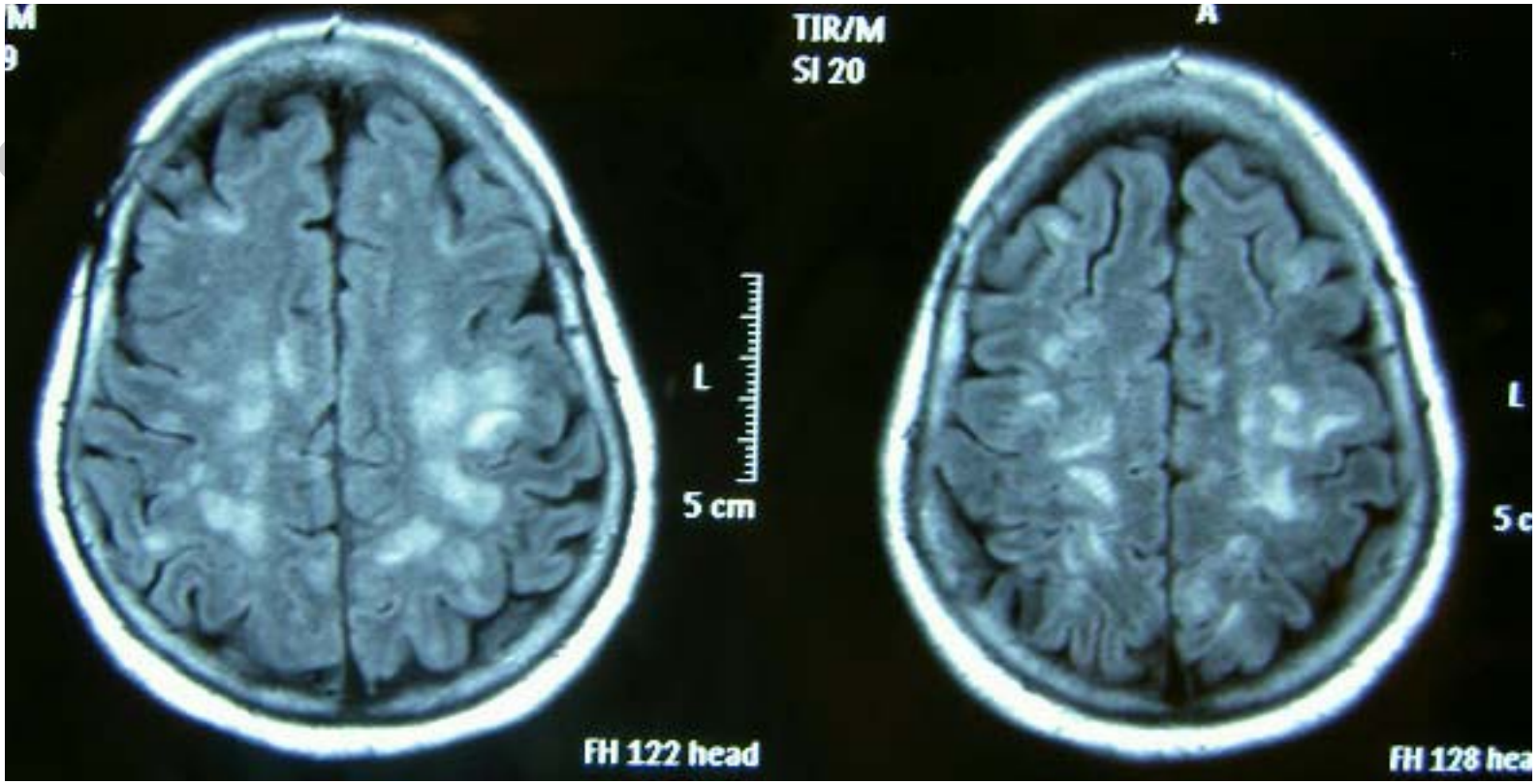

Sc 6
TIR/M
SI 6
A
L
5 cm
FH 44 head
Sc 6
TIR/M
SI 7
A

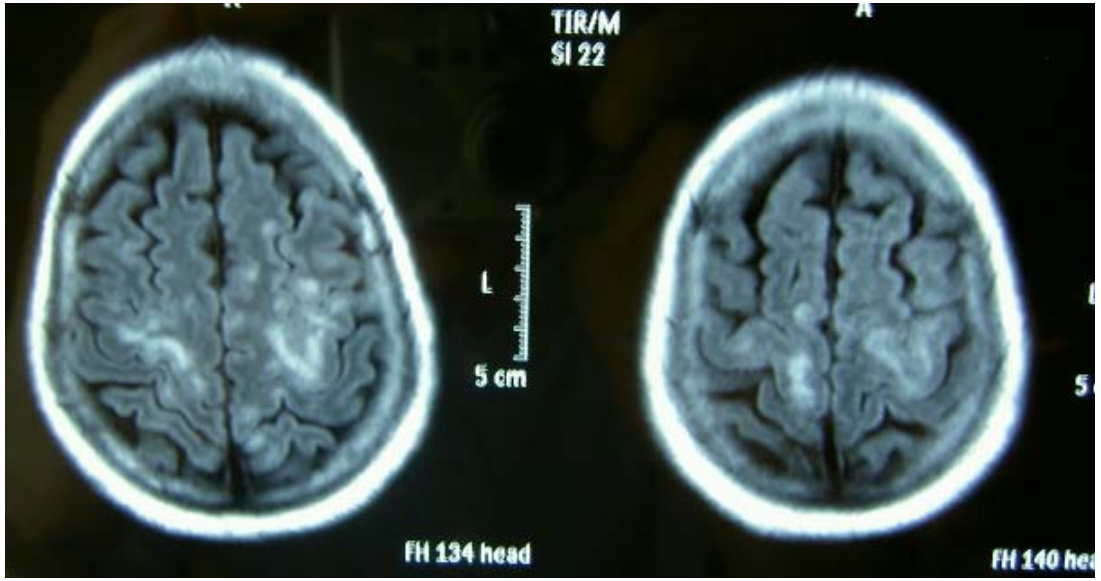

Sc 6
TIR/M
SI 9
A
L
5 cm
FH 62 head
Sc 6
TIR/M
SI 10
A
L


Sc 6
TIR/M
SI 11
A
L
5 cm
FH 74 head
6
Sc 6
TIR/M
SI 12
A
FH 80


Sc 6
TIR/M
SI 14
L
5 cm
FH 92 head
Sc 6
TIR/M
SI 15
Sc 6


Sc 6
TIR/M
SI 16
A
L
5 cm
FH 104 head
Sc 6
TIR/M
SI 17
A
L
5 cm
FH 110 head


TIR/M
SI 20
L
5 cm
FH 122 head
FH 128 hea


TIR/M
SI 22
L
5 cm
FH 134 head
FH 140 hea

MRI report

- Abnormal T2 hyperintense signals involving asymmetrically in bilateral cerebral hemispheres, mainly over periventricular white matter in bilateral frontal horns and subcortical white matter/U-fibers predominantly in parietal and occipital regions. Bilateral thalami and pons were also involved. No definite area of restricted diffusion was shown on DWI. No abnormal parenchymal or leptomeningeal enhancement was observed after contrast injection
- MRI cervical spine: unremarkable

Radiologist's opinion:

- **Demyelinating disease** e.g. acute disseminated encephalomyelitis was one of the differentials.
- Other DDX: vasculitis, multi-infarcts and less likely infection
- Clinical correlation was required

EEG Report

- Eyes opened spontaneously sometimes, did not obey commands, occasional LL tremors were observed
- Background was grossly symmetrical, mainly 5-6 Hz theta activities, voltage 20-70uV over both hemispheres. Some irregular 2.5-3 Hz slow activities, more prominent over frontal region was noted. *No definite epileptic discharge was detected.*
- Findings nonspecific, could be compatible with **encephalopathy of any cause**

Lab results

- HIV 1&2 serology: -ve
- ANCA –ve
- ANF 80, anti-ds DNA 5 (<50)
- C3 0.49(0.79-1.52), C4 0.17(0.16-0.38)
- NPA and Trach asp x swine flu PCR –ve, viral culture -ve
- Urine Legionella serotype 1 antigen –ve
- Tracheal aspirate: AFB smear –ve
- Blood cultures –ve
- ASOT <200

CSF study : Negative

- Viral culture
- PCR herpes simplex 1 & 2,
  enterovirus
- CFT measles and varicella zoster
  Ab titres <4
- AFB smear, cutlure
- Bacterial C/ST Fungal C/ST
- IgG 5.1mg/dL

- Oligoclonal band not increased

Paired Serum (18/1 & 9/2):

Influenza A	**40**	**20**
Influenza B	**20**	**10**
Adenovirus	**20**	**10**
Chlamydia gp	**<10**	**<10**
Mycoplasma pneumoniae	**10**	**20**
Parainfluenza type 1,2 & 3	**<10**	**<10**
RSV	**10**	**10**
Enterovirus	**<10**	**<10**
Varicella-zoster virus	**<10**	**<10**
Herpes simplex virus	**20**	**120**
Japanese encephalitisvirus	**<10**	**<10**
Spotted fever gp rickettsia	**<128**	**<128**
Typhus gp rickettsia	**<128**	**<128**
Orientia tsutsugamushi	**<128**	**<128**

Treatment

- IV pulse methylprednisolone 1g daily x 3
- followed by 4/52 oral prednisolone

Acute
Disseminated
Encephalomyelitis

ADEM

- Acute immune-mediated demyelinating disorder of CNS characterised by multi-focal white matter involvement
- In developed countries most commonly follows non-specific URTI
- In developing countries most commonly follows common childhood illnesses e.g. measles (1 in 1000), chickenpox
- It can also occur post vaccination
- The incidence rate is ~4-8/1,000,000 people per year

ADEM

- Though it occurs in all ages, most reported cases are in children and aldolescents, with the average age of ~ 5 to 8 yrs old
- When the patient suffers more than one demyelinating episode, it is called Recurrent Disseminated Encephalomyelitis/Multiphasic Disseminated Encephalomyelitits (MDEM)

ADEM

- Clinical features:
  - Nonspecific symptoms (e.g. fever, malaise, myalgia, headache, nausea and vomiting) often precede neurological features, beginning 4-21 days after precipitating event

Clinical features

Neurological features:

- Focal or multi-focal
- Rapid onset over few days
- Early features:
  - Encephalopathy ranging from lethargy to coma
  - Hemiparesis
  - Cranial nerve palsies
  - Paraparesis with cord lesion often complete
- Meningism
- Ataxia
- Movement disorders
- Fits in severe cases
- Optic neuritis

Post-infectious encephalomyelitis

- Viral infections:
  - Measles
  - Mumps
  - Influenza A & B
  - EBV
  - HHV-6
  - Dengue virus
  - Coronavirus
  - Varicella-zoster (1/10,000)
  - Rubella (1/20,000)
  - Hepatitis A & C
  - HIV
  - HSV
  - Coxsackie B
  - Nonspecific URTI

Post-infectious encephalomyelitis

- Non-viral infections:
  - Gp A B-haemolytic Streptococci
  - Legionella pneumophila
  - Salmonella typhi
  - Leptospirosis
  - Plasmodium falciparum
  - Mycoplasma pneumoniae
  - Rickettsia rickettsii
  - Borrelia burgdorferi

Postvaccinal encephalomyelitis

- Postvaccinal ADEM:
  - Rabies
  - - Neural type (Semple) vaccine (1/300 -1/7000)
  - - Non-neural (Human Diploid Cell) vaccine (<1/75,000)
  - Measles
  - Japanese encephalitis virus (0.2/100,000)
  - Oral poliovirus
  - Tetanus toxoid (0.9/100,000)
  - Influenza
  - Chickenpox (3/665,000)
  - Hepatitis B recombinant vaccine (10 cases)
  - Tick-borne encephalitis

Journal of Infection (2009) 58, 321e328

Pathology of ADEM

- Multiple inflammatory lesions in the brain and spinal cord, particularly in the white matter
  - Usually these are found in the subcortical and central white matter, and cortical gray-white junction of both cerebral hemispheres, cerebellum, brainstem, and spinal cord
  - But periventricular white matter and gray matter of the cortex, thalami, and basal ganglia may also be involved

ADEM

- Pathogenesis:
  - Non-immune
    - CNS demyelination as a possible result of direct neurotoxicity of a neurotropic virus
    - Disruption of the BBB sustained after direct viral CNS infection → leakage of CNS autoantigens into circulation → emergence of a self-reactive T-cell response
  - Immune-mediated
    - Molecular mimicry by which pathogens might lead to autoimmue responses

ADEM

- DDx:
  - Since there are no standardised international diagnostic criteria for ADEM, Dx of ADEM is based on the clinical history, neurological and neuroimaging findings, and CSF analysis
  - Acute viral encephalitis and multiple sclerosis are principal diseases to consider

ADEM

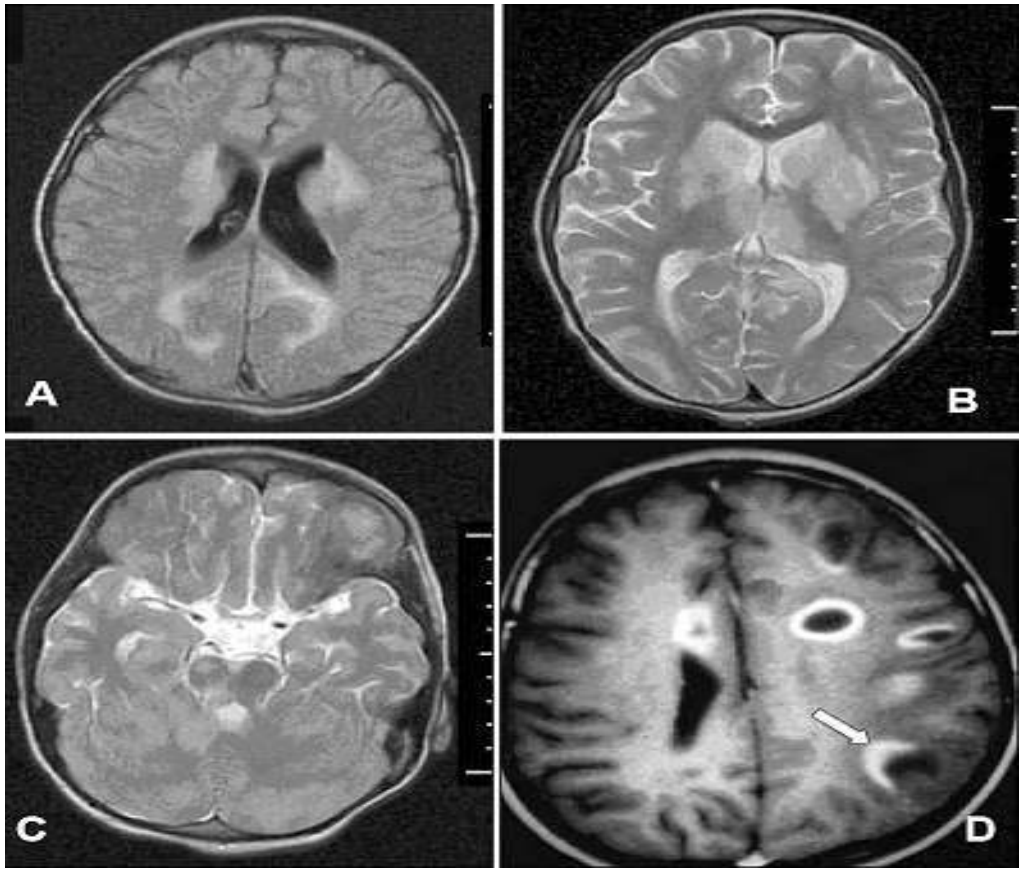
- Investigations:
  - CSF analysis by lumbar puncture and
  - Microbiological and serological tests
    - CSF typically reveals an increase in pressure, a lymphocytic pleocytosis (up to 1,000/ml) but may be polymorphs in early states, and raised protein levels (usually < 1g/dL) +/- raised gammaglobulin, and myelin basic protein
    - A CSF oligoclonal band is less common in ADEM

ADEM

- Investigations:
  - EEG
    - Non-specific abnormalities common
  - CT
    - Usually normal at onset, but 5-14 days later becomes abnormal with typical appearance showing low attenuation, multifocal lesions in subcortical white matter

ADEM

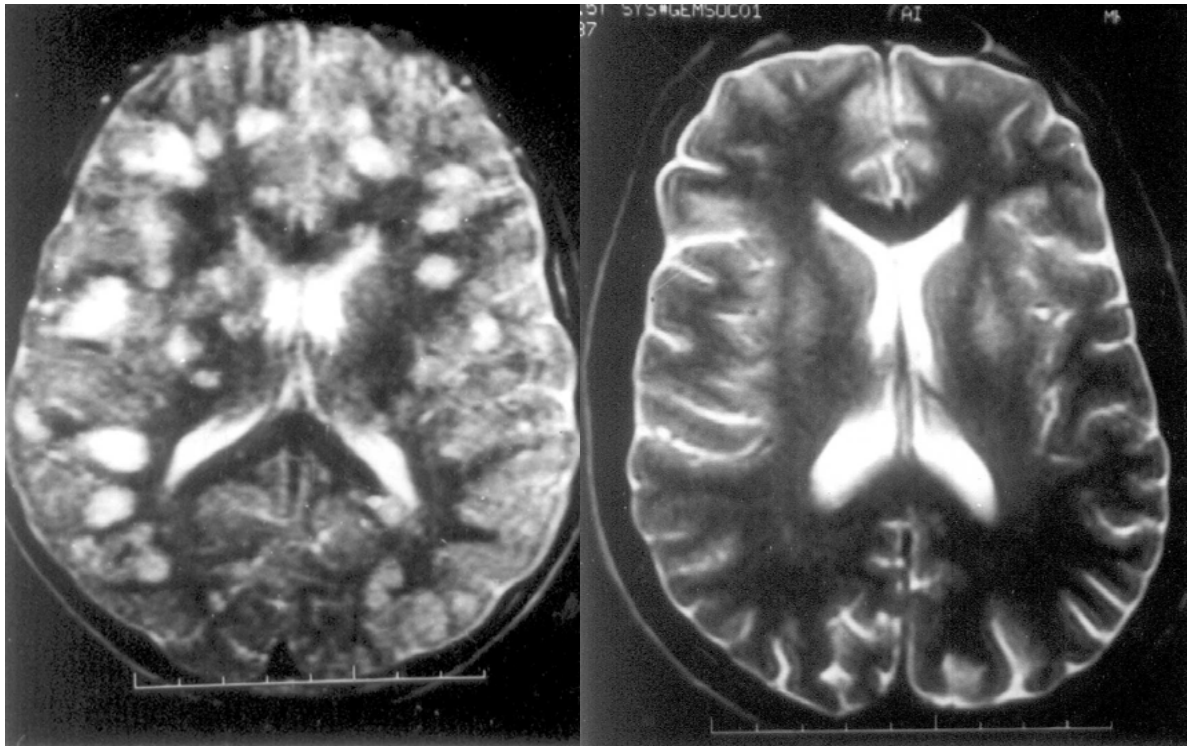
- MRI (choice of Ix modality)
  - Demyelinating lesions without mass effect scattered throughout white matter of posterior fossa and cerebral hemispheres, appearing as patchy areas of increased signal intensity on conventional T2-weighted images
  - Although there is no pathognomonic MRI appearance for ADEM, in one study cortical involvement or lesions in the basal ganglia were present exclusively in patients with ADEM as compared to MS
  - Can be normal at initial presentation and delays between 5 and 14 days from symptom onset



- Acute disseminated encephalomyelitis. Notice the contrast enhancement which is characteristic of acute lesions. Also notice that many lesions are situated at the junction of deep cortical gray and subcortical white matter which is characteristic of ADEM

ADEM

- Investigation:
  - Follow up MRI scans after a minimum interval of 6 months is recommended to establish or confirm the Dx of ADEM at which time there should be a resolution, partial or complete, of old lesions and no new lesions, and the appearance of new lesions is strongly suggestive of MS



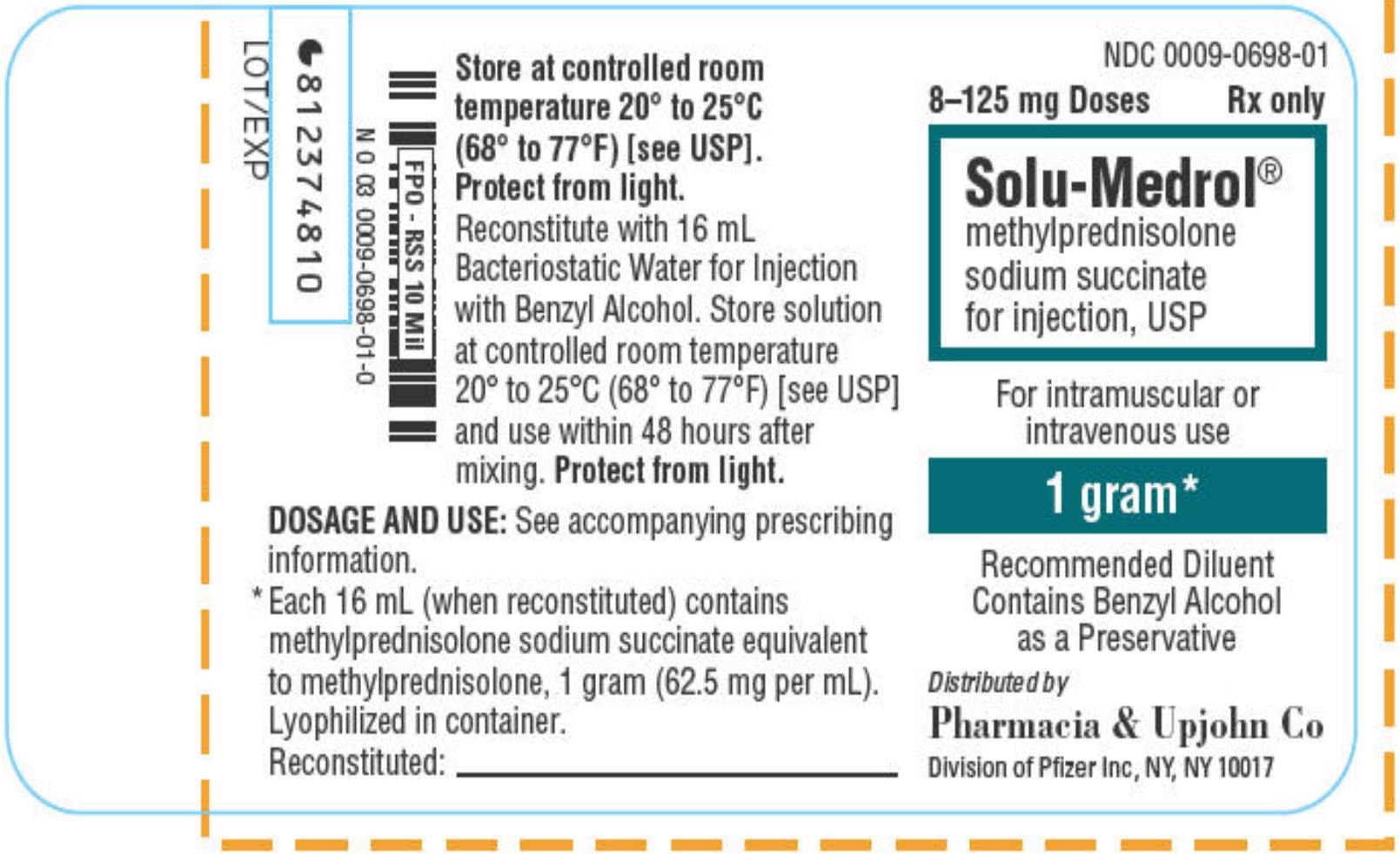
- ***Fig. 1. MRI brain T2 W image shows multiple hyperintense lesions in the subcortical white matter on both sides, right basal ganglia and internal capsule.***

- ***Fig. 2. MRI brain T2 W image after 3 months shows resolution of the lesions.***

ADEM

- Treatment:
  - There is a lack of controlled clinical trials and no proven standard treatment for ADEM
  - Most treatment options are based on empirical and observational evidence
  - Once ADEM is diagnosed and an acute infectious aetiology excluded, treatment should be instituted as soon as possible
  - Present treatments are centred on immunosuppression and immunomodulation
  - The options include corticosteroids, plasma exchange, and intravenous immunoglobulin (IVIg)

ADEM


LOT/EXP
81237481O
FPO - RSS 10 Mil
N 0 03 0009-0698-01-0
Store at controlled room temperature 20° to 25°C (68° to 77°F) [see USP]. Protect from light.
Reconstitute with 16 mL Bacteriostatic Water for Injection with Benzyl Alcohol. Store solution at controlled room temperature 20° to 25°C (68° to 77°F) [see USP] and use within 48 hours after mixing. Protect from light.
DOSAGE AND USE: See accompanying prescribing information.
* Each 16 mL (when reconstituted) contains methylprednisolone sodium succinate equivalent to methylprednisolone, 1 gram (62.5 mg per mL). Lyophilized in container.
Reconstituted: _____
NDC 0009-0698-01
8–125 mg Doses
Rx only
Solu-Medrol®
methylprednisolone sodium succinate for injection, USP
For intramuscular or intravenous use
1 gram*
Recommended Diluent Contains Benzyl Alcohol as a Preservative
Distributed by
Pharmacia & Upjohn Co
Division of Pfizer Inc, NY, NY 10017

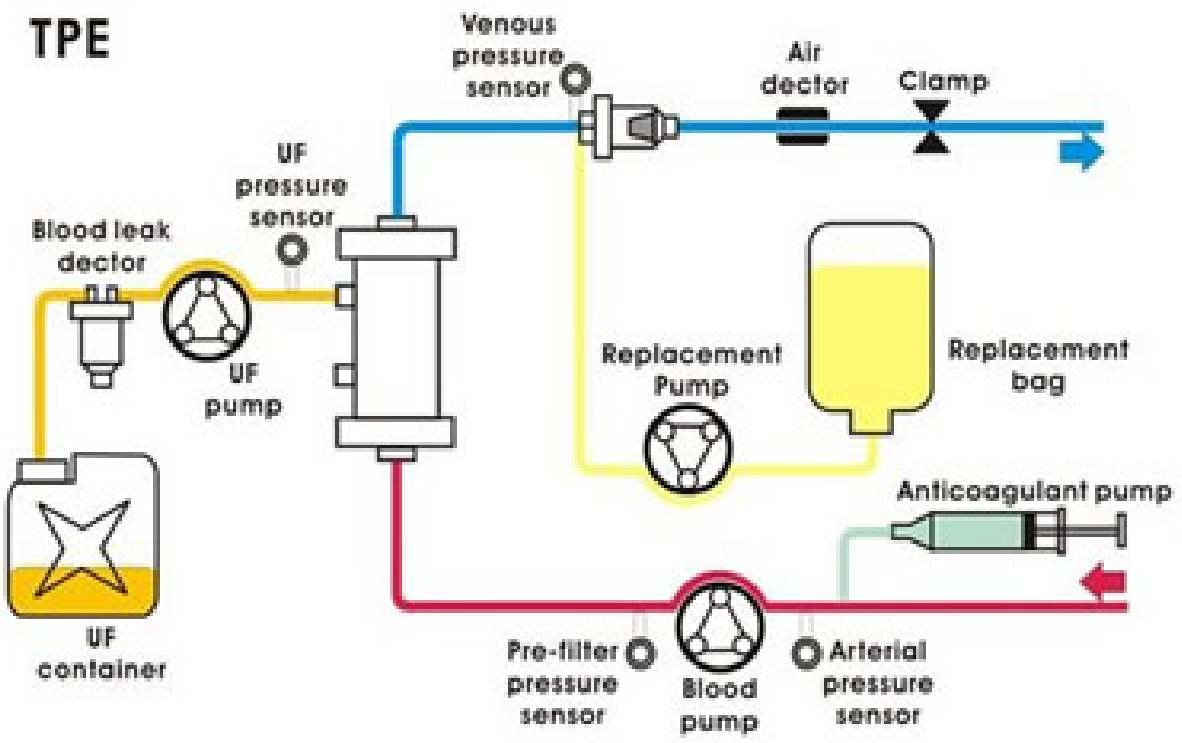
ADEM

- Corticosteroids:
  - Widely accepted as first line therapy for ADEM
  - The recommended treatment regime is IV methylprednisolone 30mg/kg/day (max. 1gm/day) or Dexamethasone 1mg/kg x 3-5 days, followed by a 1-2 month oral Prednisolone taper
  - 2/3 of patients might show a response
  - Shorter tapering might increase the risk of relapses
  - Patients treated with methylprednisolone showed better outcomes than with dexamethasone

ADEM

- Corticosteroids:
  - The effect is by their ability to reduce inflammation, reduce oedema, and seal the blood-brain-barrier, which should decrease the further influx of active immune cells and humoral factors, contributing to demyelination

ADEM


TPE
Venous pressure sensor
Air dector
Clamp
UF pressure sensor
Blood leak dector
UF pump
Replacement Pump
Replacement bag
Anticoagulant pump
UF container
Pre-filter pressure sensor
Blood pump
Arterial pressure sensor

ADEM

- Plasmapheresis:
  - It is recommended in patients who respond poorly to intravenous corticosteroids
  - The usual course involves 7 exchanges over 14 days with improvements frequently seen after the first session
  - This is perhaps through removal of antibodies that contribute to demyelination (like antibodies against myelin basic protein)

ADEM


CARIMUNE NF
Immune Globulin
Intravenous (Human)
Nanofiltered
ig

ADEM

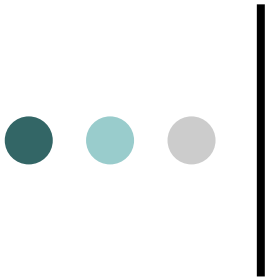
- IVIg:
  - It is reserved for ADEM patients who fail to respond to corticosteroid treatment and where plasmapheresis is contraindicated or difficult to access
  - IVIg may be preferred to plasmapheresis in cases of postvaccination ADEM
  - Certain subgroups of patients may benefit more from IVIg treatment, like those have both CNS and PNS involvements and evidence of polyradiculopathy

ADEM

- Others:
  - If the above treatments fail, several other therapies have been tried and used with anecdotal success, including intravenous cyclophosphamide and mitoxantrone

ADEM Prognosis

- The mortality rate may be as high as 30-80% in adults
- Full recovery, and in some cases spontaneous improvement, is the expected outcome in most cases, and is seen in about 50% to 70% with a higher proportion (between 70% to 90%) if minor residual deficits are considered
- The average time to recovery ranges from 1 to 6 months
- Suggested predictors of poor outcome include older age, female gender, degree of functional impairment at clinical onset, CSF protein level, spinal cord involvement, PNS damage, and poor response to corticosteroids
- Children tend to have more favourable outcomes than adults



What are the connections?

What are the connections?

- Symptoms and signs + CSF and MRI findings → ADEM?

Clinical features ~~> ADEM ?

- Fever, limb weakness
- Areflexia, reduced limb power
- Impaired consciousness (+/- sedation)→ fluctuated conscious level / hypersomnolence / tetraplegia
- Myoclonus (not seizure)
- CSF and MRI findings

What are the connections?

- 2. Post-infectious/post-vaccination?

Post-infection or post-vaccination ?

- ADEM in adults is rare
- Predominantly a paediatric disease with an incidence of 4/1,000,000
- Post-vaccination encephalomyelitis accounts for < 5% of ADEM
- **?** Incidence of post-vaccination ADEM in adults
- There is a temporal relationship with vaccination and a septic shock like picture
- No identifiable definitive infectious etiology
- **?** Idiopathic

What are the connections?

- 3. ADEM? or MS?

ADEM? or MS?

- 60 adults (aged >15) with an initial feature of ADEM from 1995-2005 in 13 French centres
- showed a 32% rate of conversion to clinically definite MS
- after a mean follow-up of 37 months (range, 1-10 years)

ARCH NEUROL / VOL 64 (NO. 10), OCT 2007

- ADEM in adults, which found a 35% rate of conversion to MS after a mean follow-up of 38 months

Neurology 2001;56(10):1313-1318

Any connection with MS ?

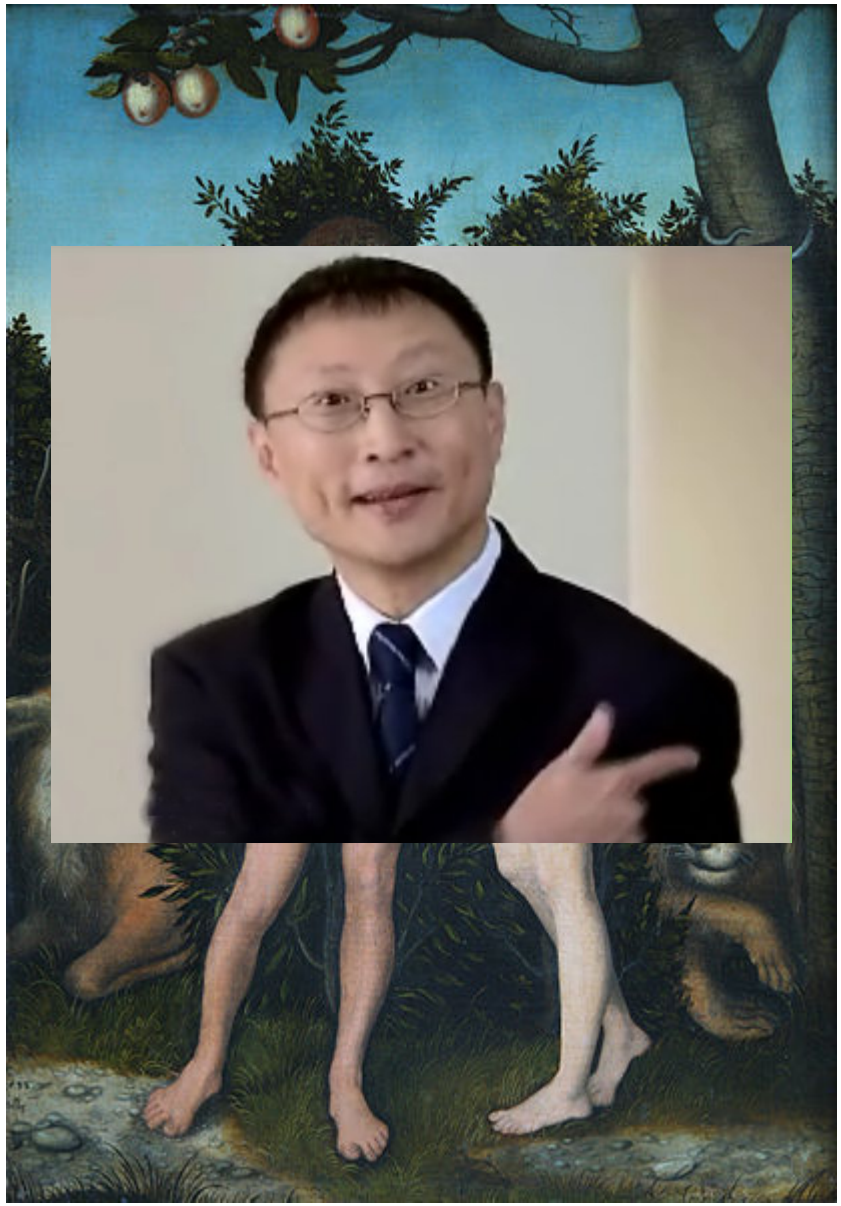
- Paediatric studies which found a rate of conversion to MS of approximately 15%
- A longer (?) follow-up of these patients is the only way to confirm that they do not eventually develop MS

Table 5. Proposed Criteria for Differentiation Between MS and ADEM

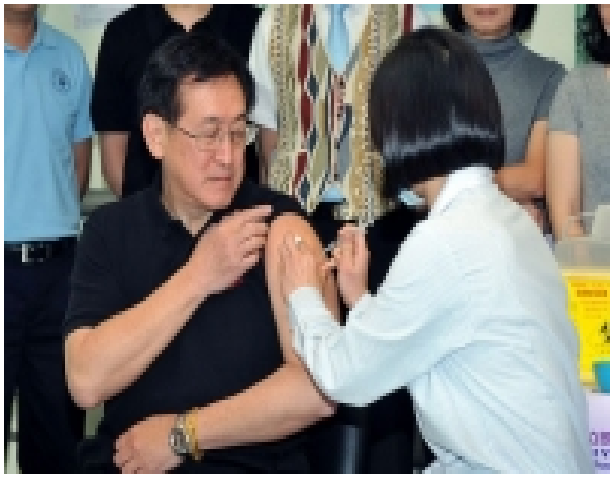
ADEM corresponds to patients with at least 2 of the following 3 criteria:
- Clinical atypical symptoms of MS. One or more of the following: consciousness alteration, hypersomnia, seizures, cognitive impairment, hemiplegia, tetraplegia, aphasia, or bilateral optic neuritis;
- Absence of oligoclonal bands in the cerebrospinal fluid;
- Gray matter involvement (basal ganglia or cortical lesions).

Twenty-nine of the 35 patients with ADEM (82.9%) at the end of the follow-up had 2 or more of these criteria and would therefore have been correctly classified, and 18 of the 19 patients with MS (94.7%) had no criteria or only 1 criterion and would also have been correctly classified.

Statistical data concerning the criteria for ADEM:
- Sensitivity = 82.9%
- Specificity = 94.7%
- Positive predictive value = 96.7%
- Negative predictive value = 75.0%
- Accuracy = 87.0%



- Have you got a jab?



廣州男童打流感針後染腦炎

- 廣州一名13歲男孩患上急性腦脊髓炎病例，其病前曾接種甲型H1N1流感疫苗。
- 專家初步診斷認為接種甲流疫苗引起的異常反應可能性大。市衛生局有關負責人表示，「這名13歲男孩的病因一旦明確診斷?接種甲流疫苗引起的異常反應，根據相關規定，政府將落實對異常反應病例的經濟補償。」
- 據了解，該病例為1名13歲的初中學生，其於去年12月21日出現發熱，其後病情加重到醫院診治，隨後轉至市婦女兒童醫療中心，期間出現意識障礙、下肢肌力變差等症狀。
- 專家會診考慮為「急性腦脊髓炎，呼吸衰竭」。經全力救治，目前患者已治癒，於1月21日出院。患者曾於去年12月16日在學校接種甲型H1N1流感疫苗

Vaccination safety?

- More frequently after primary vaccination compared to re-vaccination
- Vaccines produced in CNS tissue pose a higher risk of postvaccinal encephalomyelitis
- Encephalomyelitis or ADEM or any other adverse events – that follow administration of an inactivated component or live vaccine may be temporally associated with, but is not necessarily the result of administration of a vaccine

MMWR Morb Mortal Wkly Rep. 2009 Dec 11;58(48):1351-6.

Safety of influenza A (H1N1) 2009 monovalent vaccines - United States, October 1-November 24, 2009.

Centers for Disease Control and Prevention (CDC).

The Food and Drug Administration (FDA) licensed the first 2009 influenza A (H1N1) monovalent vaccines ("H1N1 vaccines") on September 15, 2009. The H1N1 vaccines are available as a live, attenuated monovalent vaccine (LAMV) for intranasal administration and as monovalent, inactivated, split-virus or subunit vaccines for injection (MIV). The licensure and manufacturing processes for the monovalent H1N1 vaccines were the same as those used for seasonal trivalent inactivated (TIV) or trivalent live, attenuated influenza vaccine (LAIV); none of these vaccines contains an adjuvant. Vaccine safety monitoring is an important component of all vaccination programs. To assess the safety profile of H1N1 vaccines in the United States, CDC reviewed vaccine safety results for the H1N1 vaccines from **3,783 reports** received through the U.S. Vaccine Adverse Event Reporting System (VAERS) and electronic data from **438,376 persons vaccinated** in managed-care organizations in the Vaccine Safety Datalink (VSD), a large, population-based database with administrative and diagnostic data, in the first 2 months of reporting (as of November 24). VAERS data indicated **82 adverse event reports per 1 million H1N1 vaccine doses** distributed, **compared with 47 reports per 1 million seasonal influenza vaccine** doses distributed. However, **no substantial differences** between H1N1 and seasonal influenza vaccines were noted in the proportion or types of serious adverse events reported. No increase in any adverse events under surveillance has been seen in VSD data. Many agencies are using multiple systems to monitor H1N1 vaccine safety. Health-care providers and the public are encouraged to report adverse health events that occur after vaccination.

ADEM

- The Expert Group on Serious Adverse Events with History of Human Swine Influenza (HSI) Vaccination on 10/02/2010 considered that the clinical features of the patient were compatible with ADEM. However, the expert group considered it unlikely that her illness had been caused by HSI vaccine.

中国疾病预防控制中心免疫规划中心
预防接种异常反应监测室
AEFI Surveillance, National Immunization Program
Chinese Center for Disease Control and Prevention

香港专家证实老妇患脑炎与接种“甲流”疫苗无关

- 香港卫生防护中心 2 月4 日接到怀疑接种甲流疫苗后出现脑炎的报告。专家组2 月10 日针对该报告召开会议，小组主席陈文仲在会后表示，该名老妇确诊为急性播散性脑脊髓炎，与疫苗无关。

- 患者是一名 67 岁老妇，于2009 年12 月23 日接受甲流疫苗注射，1 月12日开始不适，15 日出现发烧、头痛等症状。入院时为疑似格林巴利综合征，住院期间陆续出现败血症、低血压、肾衰竭、神志不清等情况。经检查后，该患者确诊为急性播散性脑脊髓炎，目前情况严重，正接受监护治疗。

- 专家小组成员李颂基说，大部分急性播散性脑脊髓炎病例不能确定真正原因，部分与细菌或病毒感染有关，只有5%与疫苗有关。香港每年只有10~20 例。

- 专家小组成员袁国勇表示，根据相关资料，急性播散性脑脊髓炎与感染有关，其中最严重的是流感病毒。他还强调，目前已有逾十万人接种甲流疫苗，但出现不良反应的个案非常少，公众应对疫苗接种计划有信心。

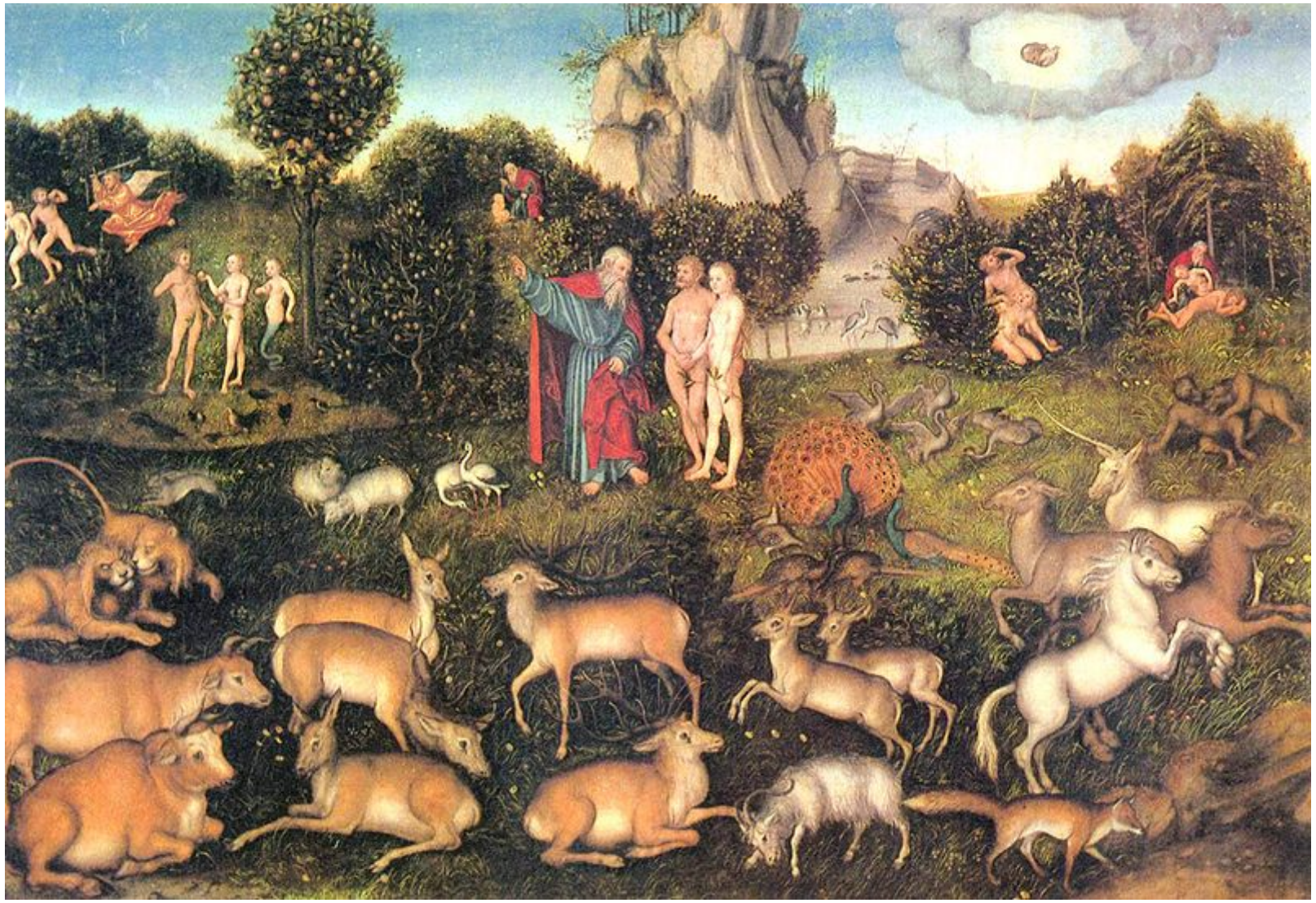
2010 年2 月11 日来源：中国新闻网
http://www.chinanews.com.cn/ga/ga-sszqf/news/2010/02-11/2120271.shtml

Progress of our patient

- ESBL(+) E. coli UTI. Rx: Meronem
- CVVH >> intermittent HD >> full recovery of RFT
- Surgical tracheostomy
- H’ stix ~8,

Neurological: E4VTM6

- Improving awakening
- Reduced sleepiness
- Limb power slowly improving
- Shoulders 1/5; elbows flexion 3/5,
- Hip flexion 1/5, knees extension 3/5



- Thank You