CIVILIAN GUN SHOT WOUND
FAILURE TO WAKE

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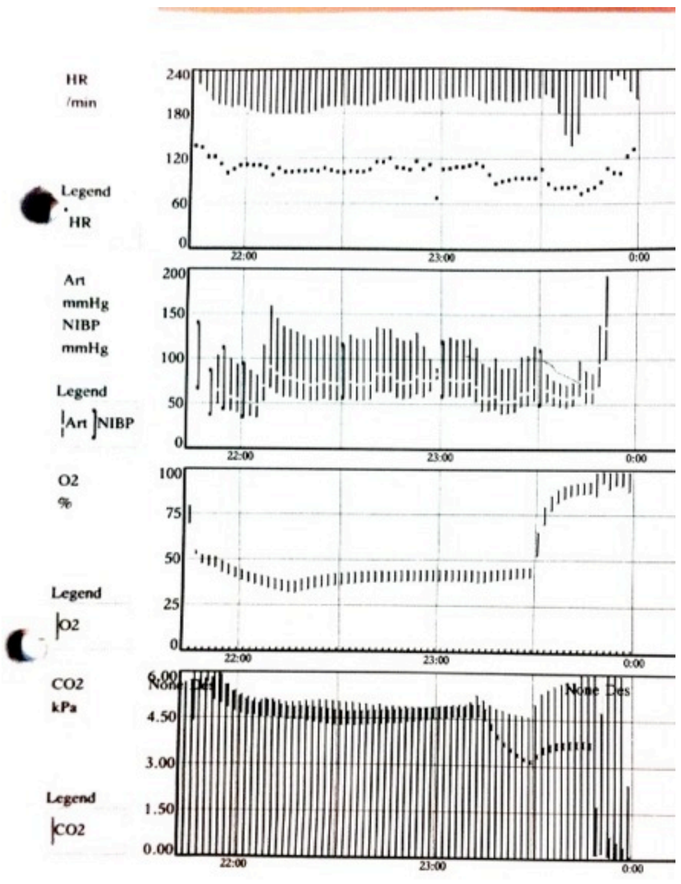
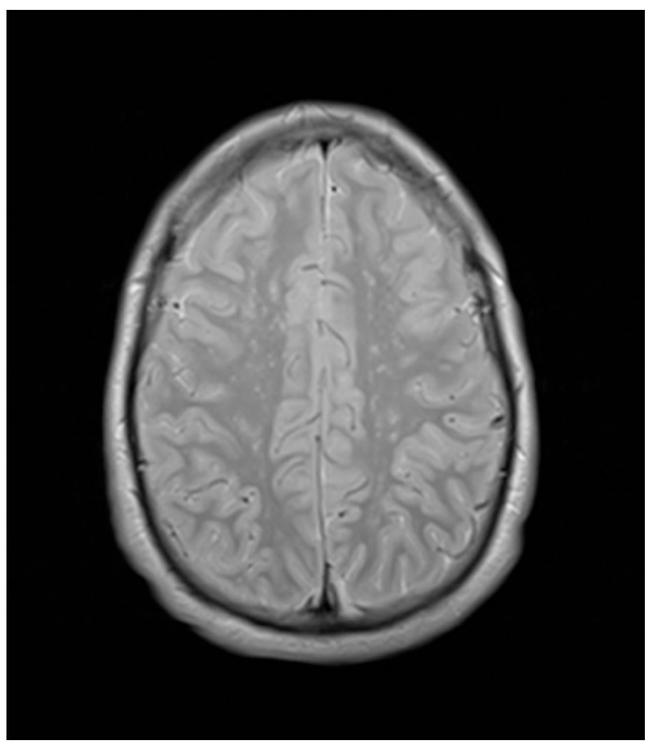
CASE PRESENTATION:

A 24 year old farmer was brought in by air ambulance following a gun shot wound to the ankle. On arrival at 16:00, he was fully conscious with stable observations. He was given 40mg Ketamine at 16:30 and 10mg of Morphine at 18:00 for analgesia.
He was transferred to the operating theatre at 21:30 for examination, debridement and washout.
He underwent an intravenous induction with 150mcg Fentanyl, 150mg Propofol and 50mg Rocuronium. He was a grade 1 intubation and a size 8 endotracheal tube was inserted. Anaesthesia was maintained with Desflurane. The procedure was completed at midnight, the volatile agent was stopped. The patient was opening his eyes to voice and coughing and localising to the endotracheal tube, so he was extubated and transferred to the recovery room. The figure below shows the anaesthetic chart.
At 2am, the patient was in the recovery room and his Glasgow Coma Scale (GCS) was documented as 5 (E - 1, M - 2, V - 1). Arterial blood gases were normal. He was given Naloxone, Flumazenil and Neostigmine - with no improvement in GCS.
At 8am, the patient was re-intubated and transferred to the intensive care unit. His CT head showed no gross abnormality. EEG examination was suggestive of encephalopathy.



Further investigations:

CTPA: showed no intracardiac or pulmonary pathology.
Trans-oesophageal echocardiogram: suggested a small patent foramen ovale, no pulmonary hypertension, no pulmonary embolus and no lung pathology.
Retinal examination: Bilateral retinal ischaemia, cotton wool spots suggesting, fat embolism or retinal hypoxia.
MRI images are shown below and opposite - widespread acute ischaemic damage.
Over the next 16 days, his GCS improved and he was discharged to the ward. 20 days after the initial presentation, the patient went back to theatre for further reconstructive surgery, the anaesthesia were uneventful and the patient woke up without any issues.



Differential diagnoses:

Incomplete reversal

Opiate excess

Ilicit drug use

Hypoxia

Hypoglycaemia

<u>Fat embolism syndrome</u>

Cerebro-vascular event

Venous thromboembolism

Demyelination

Infection

FAT EMBOLISM SYNDROME (FES):

Fat Embolism: blockage of an artery by a plug of fat that enters the circulatory system.
1862: first seen in autopsy
1873: diagnosis of the clinical syndrome

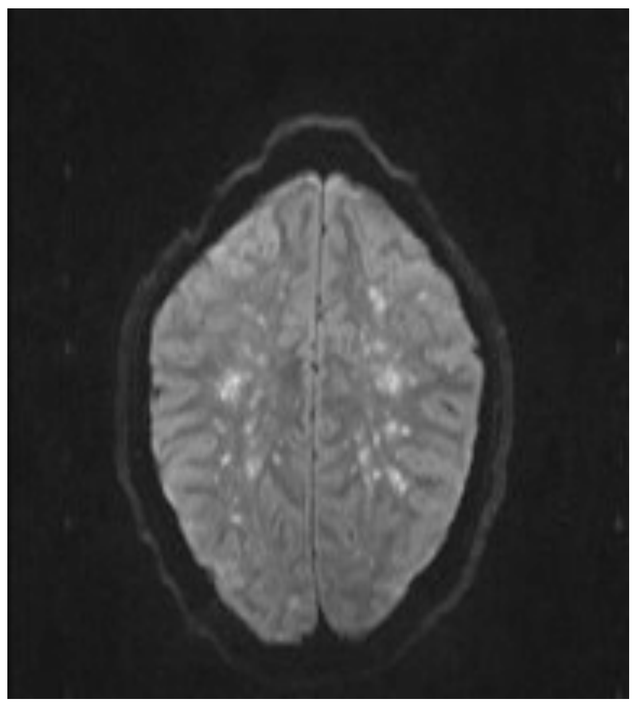
The finding of fate emboli is of doubtful significance in cases where the clinical features are absent.

Fat embolism is most commonly associated with long bone and pelvic fractures, although, fat embolism in some form occurs in nearly all these types of fractures.The syndrome occurs in only 0.9-2.2%.

Diagnosis:

Features of fat embolism syndrome are:

Latent, asymptomatic period: 12-48 hours followed by a Triad of:
Respiratory changes
Neurological abnormality
Petechial rash



Gurd published a set of major and minor criteria, which is the most commonly used diagnostic tool.

Gurd's criteria: At least one major and four minor criteria: (see below)

The pathogenesis of fat embolism syndrome is not clearly understood, there are two theories:

The mechanical theory: large fat droplets released into the venous system. These deposit in the pulmonary capillary beds. Travel through AV shunts to the brain.

MAJOR	MINOR	LAB FEATURES
Respiratory insufficiency	Pyrexia	Anaemia
Cerebral involvement	Tachycardia	Thrombocytopenia
Petechial rash	Retinal changes	High ESR
	Jaundice	Fat macroglobulinaemia
	Renal changes	

The biochemical theory: hormonal
changes caused by trauma and/or sepsis induce systemic release of free fatty acids as chylomicrons. Acute phase reactants cause these to coalesce.

Treatment:

There is no specific treatment. A high index of suspicion and early diagnosis is important. The mainstay is supportive and symptomatic treatment
Mortality is estimated at 5-15%. Most patients fully recover.

Discussion:

This is a case of a young, fit patient who has undergone a trauma. There was a latent period - 17 hours, prior to onset of symptoms. He had cerebral symptoms (1 major criteria), confirmed retinal changes - cotton wool spots and tachycardia, along with anaemia and thrombocytopenia.
MRI images suggestive of widespread acute ischaemic damage and confirmed patent foramen ovale on echocardiography.
The patient recovered conscious level with conservative, supportive management and has undergone further general anaesthesia without any adverse event.

However arguments against fat embolism syndrome as a diagnosis are: the absence of long bone involvement, the absence of lung pathology and he had only 2 minor criteria (not 4). In addition it was felt the PFO present was too small to account for the extent of AV shunt that appeared to have occurred.

BUT... In the 100 case series reported by Gurd: 10 patients presented without lung signs and 4 patients had only minor trauma. In addition Gurd's criteria does not take into account MRI findings

Pell et al 1993 NEJM published a case of fulminating FES caused by paradoxical emboli through a PFO in a patient who on initial imaging had NO evidence of an atrial septal defect and a fossa ovalis that appeared intact.

So while this case may not be a textbook presentation of Fat Embolism Syndrome, Taking into consideration the evidence presented:

- Gurd's Case Series
- MRI imaging
- Pell et al. NEJM

<u>Fat Embolism Syndrome seems the most likely diagnosis.</u>

References:

1) The Fat Embolism Syndrome. Gurd AR & Wilson RI. British Journal of Bone and Joint Surgery 1974; 56B: 408–16.

2) Fat embolism Oxford Journals Medicine BJA: CEACCP Volume 7, Issue 5 Pp. 148-151. Contin Educ Anaesth Crit Care Pain Amandeep Gupta and Charles S. Reilly

3) Fulminating Fat Embolism Syndrome Caused by Paradoxical Embolism through a Patent Foramen Ovale. Alastair C. H. Pell et al N Engl J Med 1993; 329:926-929 September 23, 1993