

CEREBRAL CHALLENGE

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Case 1

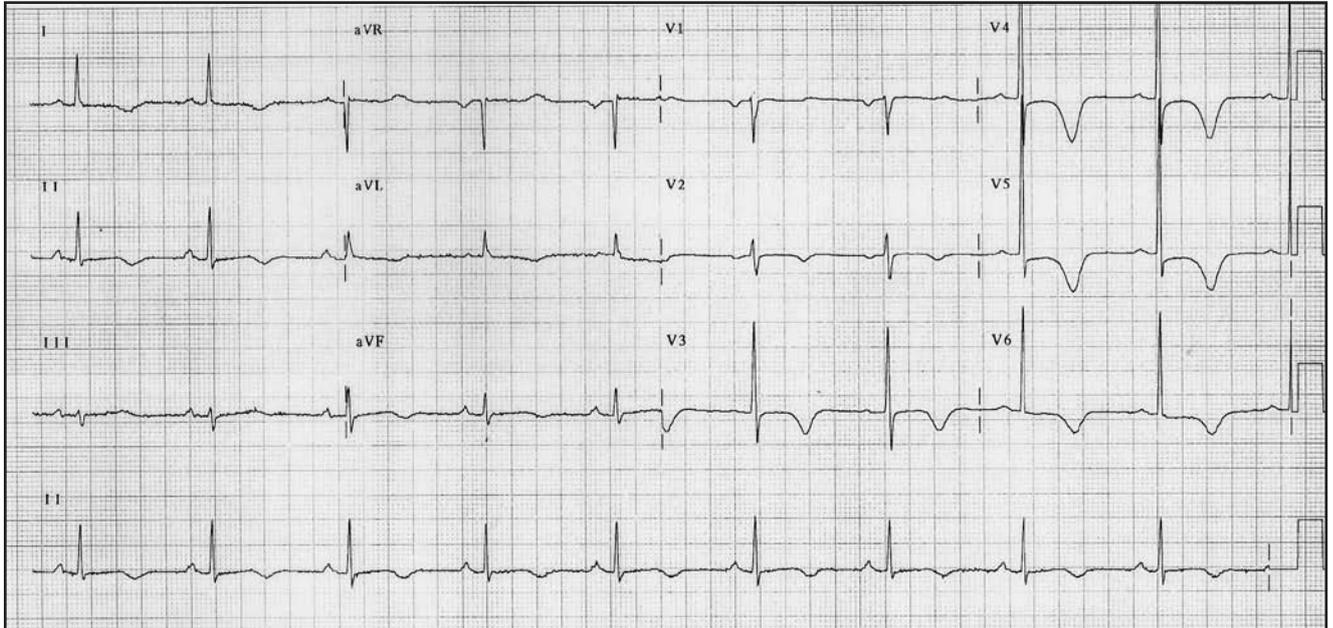


Figure 1: You are asked to anaesthetise a woman of 56 years for abdominal hysterectomy. She says she is fit and well with hypertension treated with a calcium antagonist. She denies any cardiac disease, but does admit to suffering occasional chest pain, but she has not visited her doctor. Her blood pressure is 180/92mmHg and she has had an electrocardiogram (ECG).

What does the ECG show and what are the implications for this woman's anaesthetic management?

Case 2



Figure 2: You take over a case from one of your colleagues - a 69-year-old man who is undergoing a laparotomy to relieve bowel obstruction. Your colleague noted that he had a two month history of worsening shortness of breath on exertion and put this down to bronchitis. He has also lost 8kg in weight over the last 3 months. He has been a lifelong heavy smoker with a 40-pack year history. Preoperatively examination showed him to be cachectic, with a respiratory rate of 26 per minute and oxygen saturations of 93% on air. The surgeons have relieved a band adhesion causing small bowel obstruction and have closed his abdomen. When you extubate him his breathing is laboured and he promptly desaturates. You anaesthetise him again, reintubate and ventilate him. Examining his chest, you find dullness to percussion over the right base with absent breath sounds and request a chest Xray (CXR).

What does the CXR show? Are you happy with the CXR that has been taken?

What are the possible underlying causes of his CXR diagnosis?

How would you manage this man from here?

Case 3

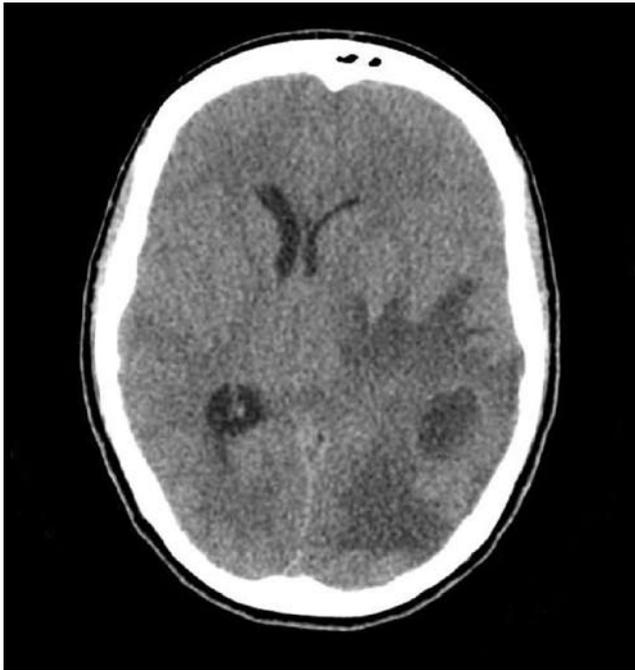


Figure 3a: CT head without contrast



Figure 3b: CT head after IV contrast

A 38-year-old man is brought into the emergency department after having two grand mal seizures at home. He had been feeling generally unwell for a few months and over the last few days had been experiencing headaches. His wife says he has been confused for 3 days and had rigors and fevers this morning.

On arrival in the emergency department he had a Glasgow Coma Score of 8 (Eyes 2, Vocal 1, Motor 5). His pupils are equal but dilated and slow to react. He is haemodynamically stable, with a blood pressure of 138/74 and a heart rate of 106/min. He is pyrexial at 39°C. He has had a CT scan of his head.

What does the CT show?

What are the likely causes of this abnormality?

Discussion

Case 1

The ECG shows sinus rhythm of 60 beats per minute. There is T wave inversion through out the chest leads, V2 to V6. It is likely she has suffered a subendocardial myocardial infarction at some stage in the past, meaning that the innermost layer of heart muscle has

been damaged by ischaemia, but the damage is not affecting the full thickness of the myocardium. Full thickness infarction of the heart muscle results in T wave inversion with formation of Q waves in the ECG leads lying over the area of damaged heart muscle.

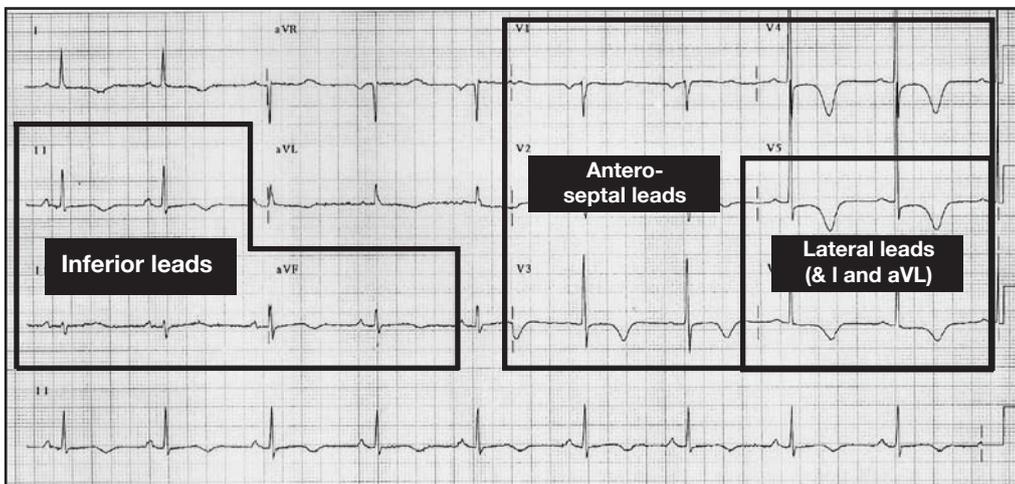


Figure 4: Assuming the leads are properly positioned on the patient, the outlined areas on the ECG correspond to the stated areas of the heart.

There are no Q waves visible on this ECG. It is possible to identify which area of heart muscle is involved by a pathological process by determining which leads show electrical abnormalities (figure 4).

The other common cause of T wave inversion on an ECG is acute ischaemia. Diffuse T wave inversion is occasionally seen in patients with intracranial pathology, such as subarachnoid haemorrhage.

This woman suffers occasional chest pain and has not sought medical advice. Her ECG suggests that she may have had a myocardial infarction in the past that she was unaware of – this may have been recently or months or years ago. Her blood pressure is poorly controlled and the voltages in the chest leads of her ECG are slightly high, suggesting that she is developing left ventricular hypertrophy (LVH) as a consequence of this. This is another possible cause of the T wave inversion.

Her surgery is elective and since she is still complaining

Criteria for diagnosis of left ventricular hypertrophy

There are several ways of diagnosing LVH using the voltages on the ECG. Sokolow's criteria are to add the height (number of little squares, mm) of the S wave in V1 to the height of the R wave in V5 or V6 (whichever is larger). If this is greater than 35mm, LVH is present. In this woman it is about 38mm and so she has LVH.

of chest pain, it is sensible to postpone her operation until she has been reviewed by a cardiologist. It is highly likely that she will be investigated further, with an exercise test or angiography, if available. She should be started on aspirin 75mg daily straight away, as secondary prevention of further myocardial infarction. A β -blocker is an appropriate agent to introduce to help control her hypertension, since it also has a role as an anti-anginal and also as a secondary preventative measure post myocardial infarction.

Case 2

The CXR is an AP portable film. 'AP' is short for antero-posterior, meaning that the X-rays have passed from in front of the patient to the X-ray plate behind them – if the film is not labelled it is an AP film.

The obvious abnormality is diffuse shadowing of the right lung field – a 'white-out' of the right lung. The principal differential diagnoses are either complete collapse of the right lung, a right pleural effusion or, in trauma, a haemothorax. Your examination of the patient (decreased air entry with dullness to percussion on the left) would fit with either of these. Note that the film has been taken with the patient lying supine (stated at the top right of the film) and also that you can see the border of the partially inflated lung behind the opacity (shown by arrows in figure 5). This is a

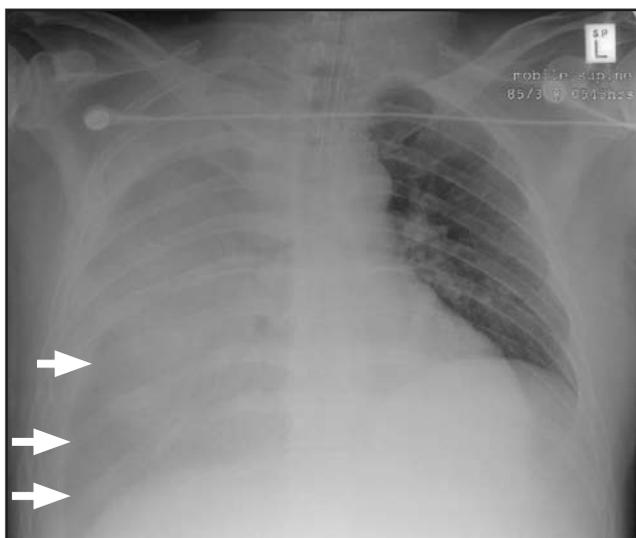


Figure 5: The border of the partially inflated right lung is just visible (arrows).

typical CXR appearance of a pleural effusion when the patient is supine – the effusion lies at the back of the pleural cavity and gives a 'veiling' opacity over the full area of that lung. Note also that the endotracheal tube is correctly placed with its tip at the aortic knuckle and that the left hemidiaphragm is elevated.

It is worth clarifying the diagnosis by performing an erect CXR which will show the pleural effusion more clearly (see figure 6).



Figure 6: An erect CXR of the same patient.

Clinically and on CXR a pleural effusion and complete collapse of one lung can be differentiated by the position of the trachea. In complete collapse the trachea will be pulled towards the side of the collapse (figure 7). In an effusion the trachea will usually be central.

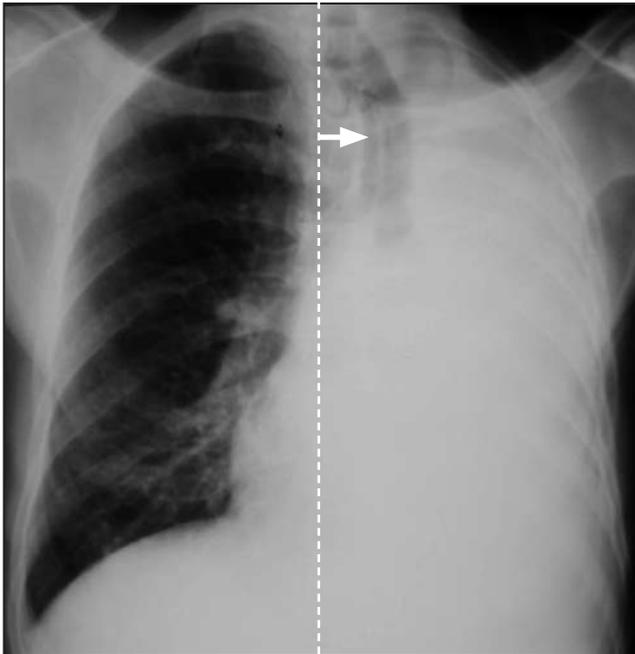


Figure 7: CXR showing left lung collapse/atelectasis with marked deviation of the trachea towards the side of collapse (arrow).

A pleural effusion is an accumulation of fluid within the pleural space. It can be detected clinically when there is more than 500ml present and radiologically when there are 300ml or above.

The causes of a pleural effusion can be divided into two broad categories:

Transudates	Exudates
Heart failure	Parapneumonic effusion
Hypothyroidism	Carcinoma of the bronchus
Constrictive pericarditis	Pulmonary infarction
Hypoproteinaemia (e.g. nephrotic syndrome, hypoalbuminaemia)	Tuberculosis
	Connective tissue disease
	Mesothelioma
	Sarcoidosis (rarely)
	Acute pancreatitis
	Empyema
	Meigs syndrome

Case 3

CT scans are viewed as if looking up through the patient's body from their feet. So as the patient lies, looking upwards, their right side is on the left as we look at the scan.

Bone is bright white, fresh blood appears white, cerebrospinal fluid is black and brain parenchyma is varying shades of grey.

Figures 3a and 3b show a round lesion in the left cerebral hemisphere.

This patient's further management should involve further investigation of the effusion. His underlying history is suspicious of bronchial malignancy, but other diagnoses such as TB are possible. Pleural fluid aspiration can be performed by simple needle aspiration using aseptic technique.

Lights criteria are used to differentiate between transudates and exudates - an exudate has one or more of the following:

- Pleural fluid protein divided by serum protein ratio > 0.5
- Pleural fluid lactate dehydrogenase (LDH) divided by serum LDH ratio > 0.6
- Pleural fluid LDH above the upper limit of normal serum LDH.

In addition samples should be sent for:

- Biochemistry
 - pH < 7.2 suggestive of empyema or malignancy,
 - protein > 30g/l - exudate is more likely
 - glucose - low in infection
 - amylase
- Microbiology (gram stain, culture and sensitivity, acid alcohol fast stain)
- Cytology (malignant cells)

Further investigation of his effusion is warranted, initially with a pleural tap and cytology, followed where available by computer tomography (CT) of the thorax. This will identify any underlying masses, enlarged lymph nodes or pleural thickening that could be suitable for CT guided needle biopsy for a tissue diagnosis.

It is the likely that the effusion contributed to this man's respiratory collapse after extubation. The effusion should be drained, after which he should be weaned and extubated.

This man should be resuscitated with attention to his airway, breathing and ventilation. With a Glasgow Coma Score of 8, where the facilities are available, he should be sedated, intubated and ventilated, whilst appropriate antibiotics are started. A preventative anticonvulsant such as phenytoin should be started (15mg/kg IV slowly).

Further history from the man's wife indicates that he is HIV positive. The most likely causative organism in an immunocompromised patient is Toxoplasma

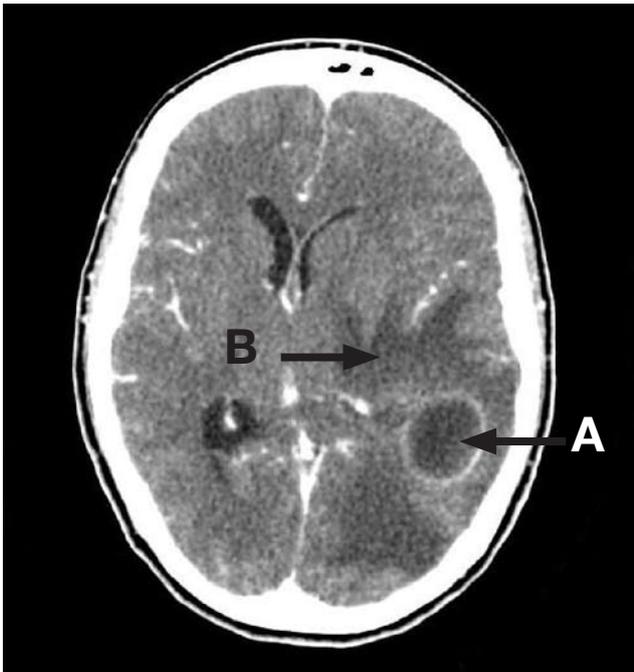


Figure 8: The border of the lesion shows up brightly after contrast – it is a ‘ring-enhancing’ lesion (arrow A). This appearance is characteristic of a brain abscess, although less commonly it can be caused by certain types of brain tumour or cerebral lymphoma. There is oedema of the brain around the lesion, which shows up darker since it contains more water than other areas of the brain (arrow B). The midline is shifted to the right and the left lateral ventricle is effaced (‘squashed’).

gondii, which can cause either single or multiple abscesses. Other organisms to consider include: TB, staphylococcus, streptococcus, salmonella, nocardia, listeria, cryptococcus, histoplasma and candida.

Toxoplasmosis is an obligate intracellular parasite. Cats are the definitive hosts and domestic cats play a

major role in its transmission. Ingesting undercooked or raw meat, blood transfusions, organ transplants and congenital infections are other methods of infestation. The most severe forms of toxoplasmosis are seen in the immunocompromised (AIDS, organ transplant patients, malignancies), usually resulting from reactivation of latent *Toxoplasmosis gondii* infection.

Clinical features may include confusion, headaches, fever, speech disturbances, motor weakness, visual field defects, cerebellar dysfunction, cranial nerve abnormalities, meningism, seizures and shortness of breath.

Laboratory investigations are:

- Serology – immunoglobulin M immunofluorescent antibody test (IgM-IFA), titre 1:160 or greater or IgM enzyme linked immunosorbent assay (IgM-ELISA) titre, 1:256 or greater is diagnostic.
- Cerebrospinal fluid examination – mononuclear pleocytosis, elevated protein, normal glucose.

Imaging:

- CT brain – single or multiple ring enhancing lesions.
- MRI brain – may detect multiple lesions mainly involving the basal ganglia and corticomedullary region, not seen on CT. This is the most reliable imaging for diagnosing *T. gondii* encephalitis.

Histology

- Where facilities exist, brain biopsy is indicated in patients who have a single lesion on MRI, multiple lesions whilst on *T. gondii* prophylaxis, negative serological findings and failure to respond to empirical treatment after 14 days or deterioration after day 3 of treatment.