

Purulent leptomeningitis mimicking subarachnoid haemorrhage on CT – a case report

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Abstract

The report of a 15-year-old female presenting with headache, fever, meningism and impaired consciousness. CT scan of the brain showed areas of increased density in the interhemispheric, sylvian fissure and adjacent to the tentorium and diffuse cerebral swelling. She was initially diagnosed to have subarachnoid haemorrhage. Blood culture later grew *Strep. pneumoniae*. Patient died in spite of antibiotics treatment. Autopsy showed purulent leptomeningitis without subarachnoid bleed. This case demonstrated that purulent leptomeningitis may mimic subarachnoid haemorrhage on CT, causing increased density of the subarachnoid space.

Key words: meningitis, CT scan

INTRODUCTION

The most common cause of increased density noted in the subarachnoid spaces is due to a subarachnoid haemorrhage. These appearances may also occur following intrathecal injection of contrast medium. We would like to report a case of purulent leptomeningitis whose CT scan had similar appearances, and was thus mistaken as subarachnoid haemorrhage.

CASE REPORT

This was a 15-year-old female presented to the casualty department, Bishop Auckland General Hospital, with a headache of several hours duration. She had been well prior to this. The headache was mainly right sided and was throbbing in nature. It was associated with nausea, vomiting of clear fluid and feeling hot. She did not have any history of previous similar illness. There was no history of any drug ingestion, alcohol abuse or trauma. On admission she was found to be drowsy but responsive to commands. She had a temperature of 38.5 degrees Celsius. The pulse rate was 90 beats per minute with a BP of 135/85. The respiratory rate was about 30 per minute. There was evidence of meningism with neck stiffness. The pupils were reactive to light, with the right being more responsive. There was also evidence of a decrease in intra-ocular pressure. A provisional diagnosis of a subarachnoid bleed was made with a

differential of drug abuse, drug reaction or intracranial abscess. The blood biochemistry was normal and the total WBC count was normal.

A CT scan of the brain (Figure 1) showed diffuse cerebral swelling with effacement of the CSF cisterns as well as the lateral and third ventricles. In addition the ambiens cisterns, basal cisterns and interhemispheric cisterns appeared dense. No intracranial haematoma was seen. A diagnosis of subarachnoid haemorrhage was made. No lumbar punctures were done in view of the markedly elevated intracranial pressure. She was then transferred to the regional neurological centre where a repeat CT scan showed similar appearances. Her condition deteriorated and she required ventilation. Her blood cultures taken the day before grew *Strep. pneumoniae* so she was then put on intravenous antibiotics. However on the third day her condition worsened with fixed, dilated pupils, with no corneal, vestibulo-ocular or gag reflexes. There was no spontaneous respiration and was declared brain dead. At autopsy, there were diffuse changes of leptomeningitis with pus noted within the subarachnoid spaces. There was widespread cerebral swelling, no evidence of subarachnoid bleed was found.

DISCUSSION

Non-contrast CT studies of the patients with acute leptomeningitis are frequently normal. The changes that have been reported include areas of

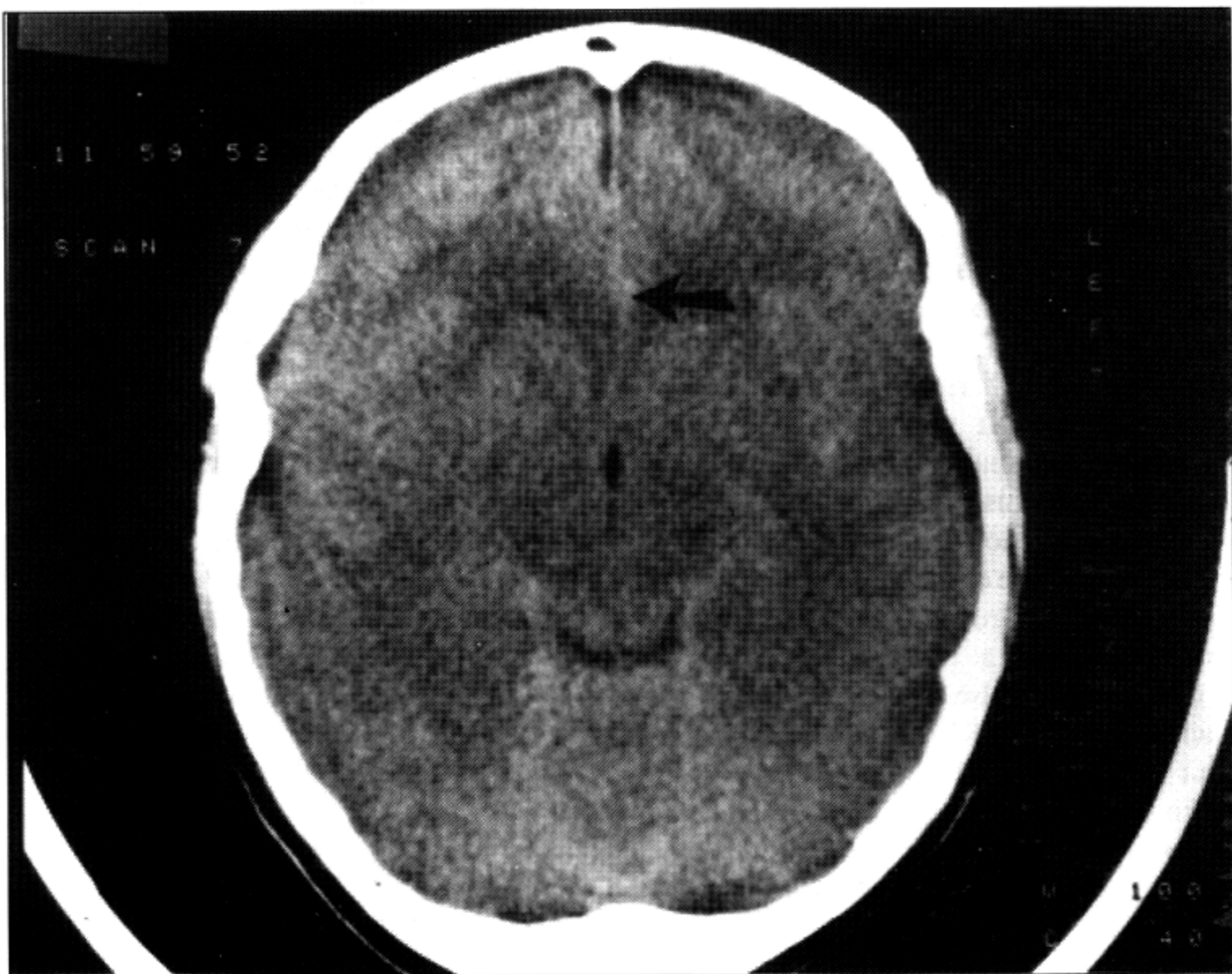
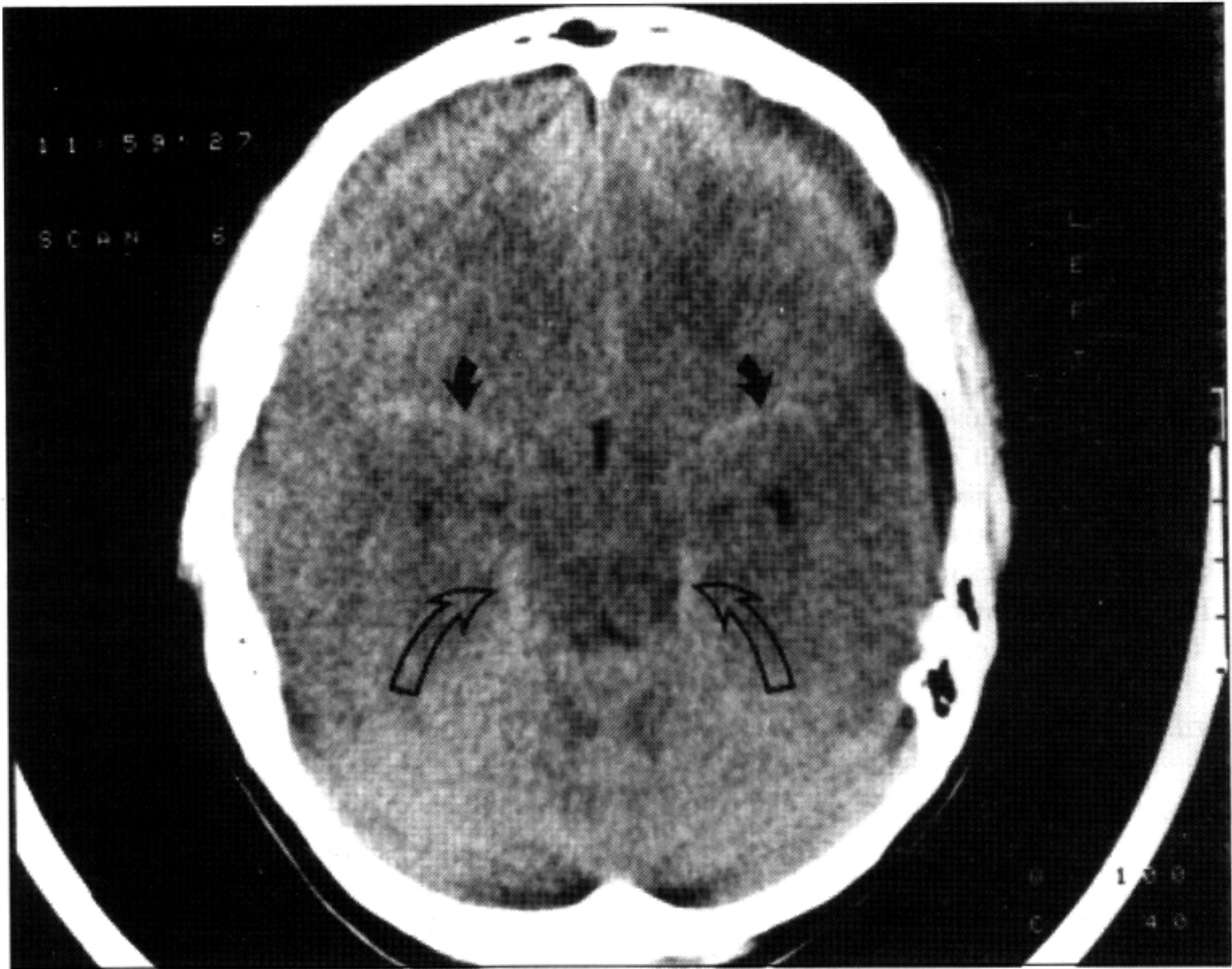


FIG. 1: Non-contrast axial image showing linear areas of increased density in the interhemispheric (large arrow), sylvian fissure (small arrow) and adjacent to the tentorium (open curved arrow). There is also evidence of diffuse cerebral oedema.

decreased attenuation within the brain parenchyma (related to ischaemia secondary to arterial or venous thrombosis) or evidence of hydrocephalus. In addition there may be evidence of enlarged subarachnoid spaces, generalized contrast enhancement of meninges and ependyma, sterile and pyogenic subdural collections, cortical infarction and cerebral necrosis.¹⁻³ There may also be evidence of obliteration of the basal cisterns which is postulated to occur secondary to meningeal hyperaemia, collections of subarachnoid exudate or a combination of these processes. The late changes that occur are those of hydrocephalus, encephalomalacia and atrophy, subdural collections and cerebritis and intraparenchymal abscesses.³

Our case confirmed that purulent leptomeningitis may also cause increased density in the subarachnoid space simulating subarachnoid haemorrhage. This observation has been reported previously.⁴⁻⁶ This presumably is due to the purulent process in the CSF associated with the presence of CSF pleocytosis and a high CSF protein level. The exact level of the CSF pleocytosis and raised protein that is required to produce an increased density in the CT scan is however not known.

When the CT scan shows increased density of the subarachnoid space, lumbar puncture would be able to differentiate between subarachnoid haemorrhage and purulent leptomeningitis. Unfortunately, as demonstrated in our patient, the diffuse cerebral oedema with raised intracranial pressure prevented a lumbar puncture from being carried out. Rennick et al⁷ suggested that lumbar puncture may cause herniation even when the CT brain is normal. In such a situation, the use of antibiotics should be based on blood culture as in this patient, or empirically on clinical grounds. This case demonstrated that purulent leptomeningitis may mimic subarachnoid haemorrhage on CT, causing increased density of the subarachnoid space.

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