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CASE SERIES

Intracranial haemorrhage complicating infection in infant masquerading as non-accidental injury – A case series

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Abstract

Introduction: The death of a 'healthy' child in a carer's house is always suspicious until proven otherwise. There is a high burden of responsibilities for a Forensic Pathologist to analyse the case from different perspectives before reaching a reasonable conclusion. *Case series:* We report a case of acute Subdural Haemorrhage (SDH) complicating lung infection and a case of subarachnoid haemorrhage (SAH) complicating multi-organism infection with possible concomitant liver disease. Both the infants were brought-in-dead to the hospital from the carer's house. There were no findings suggesting trauma. *Conclusion:* Acute Intracranial Haemorrhage (ICH) especially SDH has often been associated with inflicted head injury. Though the nontraumatic or "spontaneous" acute ICH is rare, it does exist due to multiple factors. Both the cases illustrated portrayed that the significance of the ICH has to be determined based on the autopsy findings as well as analysing the case holistically before arriving at the conclusion. Misdiagnosis of such uncommon complications could result in a catastrophic outcome.

Keywords: Intracranial Haemorrhage; Infection; Nontraumatic Subdural Haemorrhage; Subarachnoid Haemorrhage, case report

INTRODUCTION

The death of a 'healthy' child in a carer's house is always suspicious until proven otherwise. There is a high burden of responsibilities for a Forensic Pathologist to analyse the case from different perspectives before reaching a reasonable conclusion. Acute Intracranial Haemorrhage (ICH) especially subdural haemorrhage (SDH) has often been associated with inflicted head injury.[1] Though the nontraumatic or "spontaneous" acute ICH is rare, it does exist due to multiple factors.[2,3] Here, we highlight two similar cases presented with intracranial haemorrhage complicating infection in infants, masquerading non-accidental injury.

CASE SERIES

Case 1

A 52-day-old male infant was found unresponsive at his babysitter's house and was brought to the hospital by the carer. There were no signs of life upon arrival to the Emergency Department. Resuscitation commenced for 30 minutes but he did not revive, hence pronounced brought in dead. Blood was taken for biochemistry analysis and lumbar puncture (LP) was done at the Emergency Department (ED). The diagnosis of non-accidental injury was part of the suspicion of the treating doctor. Antenatal and birth history were uneventful, born full-term via spontaneous vaginal delivery. History obtained from the relevant parties revealed nothing alarming except the mother noticed the child had poor feeding and breathing difficulty during feeding for the past few days. A skeletal x-ray done was clear of any fracture.

Autopsy findings

An autopsy examination was performed on the

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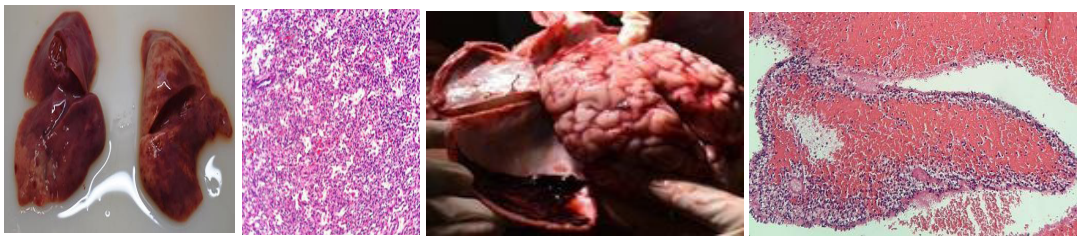

FIG. 1: Autopsy findings Case 1: (a) consolidation of lung, (b) infiltration of the lung spaces by polymorphs (10x), (c) thin film of acute SDH, (d) acute haemorrhage surrounded by large numbers of neutrophils (10x)

same day. The deceased body parameters were corresponding to age. Externally, there was no evidence of injury. Internal examination revealed consolidations involving both the lung, confirmed histologically (right: 36 g, left: 38 g) (Fig. 1a and 1b). The heart weighed 34 g with no abnormalities seen. Examination of the scalp showed no subgaleal haematoma. Upon opening the skull, there was a thin film of acute SDH measuring 12 g over the left temporal region, confirmed histologically (Fig. 1c and 1d). The brain weighed 590 g and appeared congested. Sectioning did not reveal any pathology. The circle of Willis was healthy. Further extension of dissection to the back of the neck did not reveal any injury. The laboratory investigation of the cerebrospinal fluid showed no growth after 48 hours of incubation, blood-stained appearance and no organism seen on Gram Stain and India Ink. Lung tissue submitted for culture was negative for respiratory syncytial virus panel and blood culture yield *Staphylococcus Aureus*. Toxicology analysis of the blood specimen was negative for ethyl alcohol and common drugs.

Case 2

A 4-month-old male infant was found unconscious at the carer's house. He had a history of passing out loose motion a few days before his demise. He was the last of three siblings and the offspring of a non-consanguineous marriage. The other siblings were in good health. There was no previous history of admission, and the prenatal and birth histories were unremarkable; the baby was born naturally via vaginal delivery. The vaccinations were up to date. There was no history of fever as well. The infant was brought-in-dead and was resuscitated for more than 30 minutes by the emergency department personnel. Intracardiac culture and cerebrospinal fluid (CSF) culture were taken by the resuscitating team prior to postmortem.

Autopsy findings

Postmortem examination was performed three days later. He was pale but there were no external features of dysmorphism or injuries. His weight and anthropometry measurements were appropriate for his current age. Internally, there was symmetrical subarachnoid haemorrhage (SAH) at the interhemispheric and bilateral temporal regions (Fig. 2a and 2b). However, there was no brain contusion or subgaleal hematoma. His liver was enlarged (286 g) and showed fatty changes on sectioning. The thymus gland also was enlarged. His lungs were oedematous (right: 94 g, left: 64 g). Retinal haemorrhage and cervical spinal cord contusion were not detected. The other organs were unremarkable. The histology section of the liver showed diffuse microsteatosis with prominent extramedullary haematopoiesis (Fig. 2c). Section from the brain showed cerebral oedema. There was no evidence of vascular malformation or aneurysm in the cerebral blood

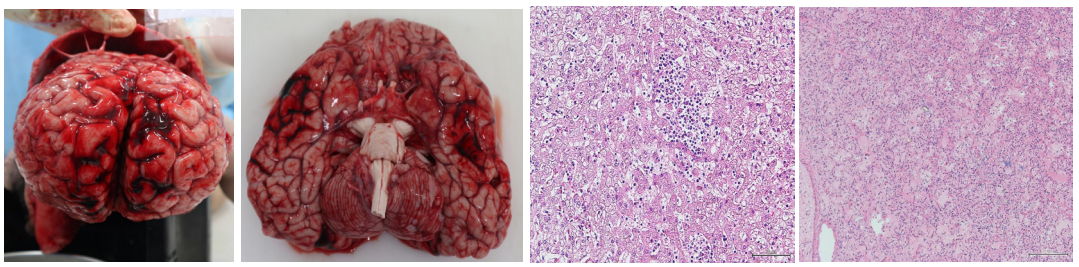

FIG. 2: Autopsy findings Case 2: (a) (b) acute SAH (c) extramedullary haemotopoiesis in the liver (10x) (d) infiltration of the lung spaces by inflammatory cells (10x)

vessels. Section from both eyes revealed no evidence of retinal haemorrhage. The meninges showed recent subarachnoid haemorrhage. Section from his lungs showed inflammatory cell infiltration (Fig. 2d). Postmortem blood culture and CSF culture grew *Enterobacter, Klebsiella* and *Citrobacter*. His blood was also sent for inborn error metabolism (IEM) studies and showed mild elevation of glutamic acid, methionine, leucine and glycine which may suggest liver dysfunction with hyperammonemia and ketosis. The deceased blood and urine were negative for ethyl alcohol and common drugs.

DISCUSSION

Acute ICH, especially acute SDH occurs frequently in inflicted head injury. The series of Geddes *et al*. records their presence in 84% of infants and 81% of older children.[1] Nontraumatic or "spontaneous" acute SDH is rare, accounting for 0.7–6.7% out of all acute SDHs.[2] According to the review of 193 cases collected by Coombs *et al*.[3], its aetiology included cortical artery bleeding, vascular lesions, coagulopathy, neoplasms, spontaneous intracranial hypotension, cocaine, and arachnoid cyst. Besides, the presence of SDH is a rare complication of diagnostic LP and LP performed for spinal anesthesia.[4,5] In the first case presentation, the significance of the SDH has to be determined based on the autopsy findings as well as analysing the whole case holistically. An acute SDH of greater than 120 ml is invariably fatal, that an acute SDH of 50 to 120 ml is more likely than not to cause death (particularly if there is significant and marked subfalcine herniation and uncal herniation), and that an acute SDH of less than 50 ml is unlikely to be fatal.[6] R. Joseph *et al*. on the other hand describe significant SDH in autopsy as 25 ml or more with involvement in two or more region of the brain (frontal, parietal, temporal, occipital, and cerebellum).[7] The findings of the SDH in the first case, was determined as not significant based on the volume is low, involvement of just a single region with no herniation seen. The acute SDH can be formed due to hypoxia, disseminated intravascular coagulation as a result of the lung infection or even as a complication from the LP done. These causes have also been mentioned by Pollanen. In his article, he emphasised that not all SDH is traumatic and can be due to coagulopathy or rare diseases such as glutaric aciduria and mitochondrial disorders.[8] These alternate explanations are non-controversial and are widely accepted. Therefore, based on the circumstances, the absence of indicator of trauma and post-mortem pathology correlation, the cause of death was given as lung infection in the first case.

In the second case, the intracranial finding is predominantly subarachnoid haemorrhage. Spontaneous subarachnoid haemorrhage, although rare, may be devastating in children. The aetiology of spontaneous SAH may be divided into three categories: arteriovenous malformation (AVM), cerebral aneurysm, and miscellaneous causes.[9] Miscellaneous causes of spontaneous SAH include neoplasms, infection, leukaemia, haemophilia, sickle cell disease, and ingestions of drugs such as cocaine and amphetamines. In this case, the blood and CSF culture isolated enteric organisms potentially associated with loose motion in the infant. It is well known that blood-borne infection could cause coagulopathy. As trauma is ruled out in this case, we believe that patchy subarachnoid haemorrhage is most probably associated with coagulopathy. On the other hand, the histology section of the liver showed evidence of extra-medullary haematopoeisis with enlargement of the thymus indicating that the infant is under stress a few days prior to death. It has been reported that extramedullary may be a consequence of anaemia associated with intrauterine hypoxia, or infections.[10] The findings of steatosis, in this case, could result from either sepsis or IEM. In addition to that, the results of IEM in conjunction with hyperammonia, hepatomegaly and steatosis make liver disease and inborn error of metabolism cannot be ruled out. Considering all the findings stated above, we believe that the blood-borne infection associated with loose motions is stressing out this infant. It is also capable to cause coagulopathy and subsequently patchy SAH. With probable underlying metabolic liver disease, it tilts the balance in this infant and leads to his death at that particular moment. The cause of death was given as SAH complicating multi-organism infection or sepsis with underlying liver disease.

CONCLUSION

Both cases highlight the importance of a careful approach in dealing with intracranial haemorrhage especially in paediatric age group. Though it is not unusual to blame trauma as the cause of intracranial haemorrhage in children, natural causes also need to be explored to prevent false litigation especially when there is a lack of other indicators of trauma. In conclusion, it

is very important to analyse a case holistically. Misdiagnosis of such an uncommon complication could result in a catastrophic outcome.

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