Hearing loss due to mumps

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SUMMARY A 21 month old girl who presented with what seemed to be hypertensive encephalopathy is described. Although her encephalopathy resolved with antihypertensive treatment, subsequent investigations revealed haemophilus meningitis.

We report a case of a child with otherwise uncomplicated meningitis who presented with fever, systemic hypertension, and encephalopathy. The possible pathogenic mechanisms are discussed and the need to consider meningitis in the differential diagnosis of acute hypertension is emphasised.

Meningitis presenting as hypertension

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previously measured 39°C) and blood pressure 200/100 mm Hg. Peripheral pulses and perfusion were normal. There was no evidence of dehydration. Mild neck stiffness was noted. The fundi were normal with no evidence of papilloedema. There were no focal neurological signs, although there was generalised hypotonicity. There was no peripheral oedema and urinalysis revealed a trace of protein, was a mild ketones, and two epithelial cells. There was a single cafe au lait spot. Abdominal examination yielded normal results. Laboratory investigations revealed no abnormalities and included a haemoglobin concentration of 11.8 g/dl, white blood cell count 10 × 10⁹/l, and normal creatinine and electrolyte concentrations. Chest x-ray yielded normal results and blood and urine cultures were taken. On the basis of these features a provisional diagnosis of hypertensive encephalopathy was entertained and the hypertension was treated with frusemide 1 mg/kg and diazoxide 5 mg/kg (in divided boluses over 60 minutes). Over the subsequent 30 minutes her blood pressure fell to 150/80 mm Hg, and this fall in blood pressure was associated with an improved conscious state. She was then transferred to our hospital.

On assessment after transfer her blood pressure was stable at 110/70 mm Hg. Blood pressure was equal in both upper and lower limbs. Her temperature remained raised at 38.8°C. Her conscious state had improved to appropriate verbal responses, although she remained irritable. Fundi were normal but neck stiffness was noted and there was mild peripheral weakness. There was no evidence of dysautonomia. Repeat full blood count showed a haemoglobin concentration of 13.9 g/dl and a white blood cell count of 20 × 10⁹/l with toxic changes. In view of the neck stiffness and now toxic blood film a lumbar puncture was performed, which showed a turbid cerebrospinal fluid under normal pressure with 7600 white cells/ml, 2400 red cells/ml, and Gram negative bacilli. She was begun on treatment with ampicillin and chloramphenicol and slightly restricted intravenous fluids.

Throughout her hospital stay blood pressure remained within the normal range. Temperature and neurological examination became normal within 48 hours and remained so. Cerebrospinal fluid cultures grew Haemophilus influenzae type b. Serum creatinine, urea, and electrolyte concentrations were normal and renal ultrasound showed normal renal anatomy.

**Discussion**

This child is presented to illustrate an unusual presentation of acute meningitis. Although acute transient hypertension is known to occur in diseases associated with raised intracranial pressure,¹ the association with acute meningitis is not documented. There was no clear evidence in our patient for raised intracranial pressure. Her fundi were normal, there were no suggestive neurological signs, and cerebrospinal fluid was tapped at normal pressure on lumbar puncture. At the time of presentation her encephalopathy was considered to be secondary to systemic hypertension, and this seemed to be supported when her conscious state improved after treatment of the hypertension. The dose of diuretic that she received, however, may have resolved a raised intracranial pressure that was not clinically evident.

Whatever the specific pathophysiology, the hypertension in our patient does seem to have been associated with the haemophilus meningitis. There was neither evidence for nor treatment of extracellular fluid volume depletion. There was no suggestion of Riley-Day syndrome. Our investigations cannot, however, totally exclude renal disease or primary catecholamine excess, although neither seem probable.

The pathophysiology of meningitis involves diverse mechanisms that could produce a picture of hypertension and encephalopathy.

(1) The involvement of blood pressure regulatory centres from the hypothalamus and medulla.² ³ For example, (a) ischaemia or hypoxia of medullary centres through blood flow alterations associated with inflammation or ischaemia produced by mechanical compression;³ ⁵ (b) increased blood volume through hypothalamic antidiuretic hormone mechanisms, although usually this would involve a longer time course;³ ⁸ (c) sympathetic overactivity through stimulation of brain stem or spinal centres in which there may be other evidence of autonomic malfunction, such as poor peripheral perfusion, tachycardia, or poor urine output.⁵ ⁶

(2) Raised intracranial pressure, although this would usually be associated with vagal activity.⁴

(3) Some children may have an underlying idiopathic tendency to hypertension in stressful situations.¹

It is not possible to delineate the exact mechanism of our patient’s hypertension. Although mild raised intracranial pressure was possibly the predominant cause, this was certainly not detectable clinically apart from encephalopathy originally attributed to systemic hypertension. The assumption that systemic hypertension in cases of meningitis is secondary to raised intracranial pressure is therefore questionable. It is also evident that meningitis should always be considered in the differential diagnosis of acute systemic hypertension, especially if this is associated...
with fever. This case also illustrates the potential benefit in treating hypertension even in acute neurological disorders, although this should always be performed cautiously.6

References


Phosphatidylglycerol in tracheal aspirates for diagnosis of hyaline membrane disease

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SUMMARY Lecithin:sphingomyelin ratio and phosphatidylglycerol were determined by a rapid, simple method in tracheal aspirates obtained from 132 newborn infants with respiratory diseases, sixty five of whom developed hyaline membrane disease. Phosphatidylglycerol determination was more sensitive (97%) than lecithin:sphingomyelin ratio, but their specificities were similar (76%).

A deficiency in the pulmonary surfactant is believed to cause hyaline membrane disease in newborn infants. Pulmonary maturity has been assessed by the analysis of the phospholipids in the hypopharyngeal and tracheal aspirates of newborn infants. The most widely used test is the lecithin: sphingomyelin ratio described by Gluck et al,3 but this method is time consuming and affected by contamination of the specimen with blood or meconium.

As a consequence, several investigators have suggested that other phospholipids from surfactant and particularly phosphatidylglycerol (PG) should be studied.4 PG is synthesised almost exclusively by lung alveolar cells so that there is no interference in assays by blood or meconium. Both one and two dimensional thin layer chromatography are commonly used to analyse PG. These methods are time consuming, however, and require extensive laboratory technician training.

In 1983 Garite et al described a new, rapid, simple, and reliable method to determine PG in amniotic fluid by a semiquantitative immunological slide agglutination test.5 A complete assay can be performed in about 30 minutes.

We have applied and compared this test to the lecithin:sphingomyelin in the tracheal aspirate.

Patients and methods

Tracheal aspirates were obtained from 132 infants who were admitted to the intensive care unit for respiratory disease. Of these, 65 were diagnosed as having hyaline membrane disease after satisfying clinical and x-ray criteria. The other 67 infants had other respiratory diseases—namely, transient tachypnoea of the newborn (30 cases), congenital pneumonia (six), amniotic fluid aspiration syndrome (11), and other respiratory diseases (20). The gestational ages of the infants ranged from 26 to 42 weeks (mean 33 weeks) and their birth weights from 645 to 4700 g (mean 2160 g). Samples were obtained during the first 24 hours after birth.

The lecithin:sphingomyelin ratio was determined using the method of Gluck et al5 and PG testing by Amniostat-FLM4 were performed on all samples. A lecithin:sphingomyelin ratio ≥2.0 and a PG≥++ or 2 µg/ml were interpreted as being ‘positive’ and as indicating fetal lung maturity and a lecithin:sphingomyelin ratio <2 and PG<++ were interpreted as being ‘negative’.2,5

Results

The Table shows the results of the lecithin:sphin-
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