SHORT REPORTS

Meningitis and septicemia due to Haemophilus influenzae type b in pregnancy

Meningitis caused by Haemophilus influenzae is uncommon in adults. We report a case of meningitis and septicaemia caused by H influenzae type b in a pregnant woman.

Case report

A 22-year-old woman with a history of recurrent otitis media in childhood was admitted to hospital at 35 weeks of gestation with an upper respiratory tract infection of four weeks' duration. The day before admission she had had a fever of 38.5°C, headache, and nausea. A few hours before admission her condition had deteriorated and she had lost consciousness.

Cerebrospinal fluid was cloudy and contained leucocytes 16·1 × 10⁹/l (16 100/mm³; 93% polymorphonuclear cells), protein 8·0 g/l, and glucose 0·4 mmol/l (7·2 mg/100 ml). Cerebrospinal fluid and six blood cultures grew H influenzae type b, sensitive to benzyl penicillin, ampicillin and chloramphenicol. Chest x-ray picture was normal but sputus radiography showed left-sided maxillary sinusitis. Treatment consisted of intravenous ampicillin 16 g daily for eight days. The course was uneventful except for premature uterine contractions, which were stopped with terbutaline intravenously. Three weeks later she gave birth to a boy weighing 3280 g. His development corresponded to 39 weeks of gestation. The Apgar score was 10 after one and five minutes. The placenta was normal and yielded no bacterial growth. One month later mother and child were well.

The patient's serum immunoglobulin concentrations on day 2 after admission were normal except for an IgG value of 4·9 g/l (3 standard deviations below normal); on day 9 the value was 8·9 g/l. Serum IgG, IgM, and IgA antibodies specific to H influenzae capsular polysaccharide type b were detected with the enzyme-linked immunosorbent assay and bactericidal antibodies against the strain isolated from the patient were found one day after admission. Much higher antibody titres were noted in serum samples obtained on days 33 and 58 after admission (figure). At delivery the same titres of specific antiscapular IgG and bacterial antibodies were detected in sera from the mother and umbilical cord, while specific IgM antibodies were detected in cord serum. IgA antibodies specific to capsular polysaccharide type b were detected in breast milk with the enzyme-linked immunosorbent assay on days 1 and 5 after parturition. These antibodies were probably of the secretory IgA type and the titres were obtained with anti-IgA and antiserumponent antibodies (day 1: serum IgA 2048 and secretory IgA 1024; day 5: serum IgA 64 and secretory IgA 64). Specific IgM antibodies in a titre of 64 were detected in the first breast milk sample.

Comment

Meningitis due to H influenzae type b is primarily a disease of early childhood and is rarely encountered in adults. This is partly explained by the lack of bactericidal antibodies in young children and the acquisition of such antibodies in older children and adults. The antibody value that is protective against H influenzae type b disease has not been clearly defined, however.

In our patient maxillary sinusitis was the most probable source of the infection. Initially she also showed a low total serum IgG concentration. Neutrophil chemotaxis, the ability to reduce nitroblue tetrazolium, and cell-mediated immune functions are depressed in pregnancy. Thus suppression of the maternal immune system might have contributed to the severe infection. Capsular polysaccharide type b (unpublished observations) and bactericidal antibodies were present in titres corresponding to those of a healthy adult population, which suggests previous exposure to H influenzae type b or other bacteria possessing cross-reacting antigens. Rising antibody titres indicate a normal immune response.

Placental transfer of specific IgG antibodies may give an important contribution to the infant's defence against H influenzae type b infection. It is not known whether the high amount of specific secretory IgA found in colostrum will protect the newborn breast-fed against infection caused by H influenzae type b locally in the respiratory tract.


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Effect of dobutamine on insulin requirement in diabetic ketoacidosis

Certain beta-adrenergic agonists—for example, isoprenaline, dopamine, and salbutamol—interfere with glucose tolerance in diabetes, resulting in hyperglycaemia and in some cases (ketoacidosis). We report a case in which dobutamine, a synthetic inotropic agent structurally related to dopamine, appreciably increased the insulin requirement of a diabetic in ketoacidosis.

Case report

A 45-year-old insulin-dependent diabetic woman presented with severe ketoacidosis. She was comatose, dehydrated, and ketotic with a heart rate of 100/min and blood pressure of 90/40 mm Hg. There was no clinical evidence of infection, and initial investigations showed a blood glucose concentration of 80 mmol/l (1441 mg/100 ml), bicarbonate 2 mmol/l (meq/l), hydrogen ion 180 mmol/l (18 ng/100 ml), haemoglobin 10·2 g/dl, and white cell count of 18·5 × 10⁶/l, and normal chest x-ray appearances. Treatment consisted of an intravenous infusion of 5 units of neutral insulin (Actrapid MC) hourly and fluid replacement. Seven litres of isotonic saline were given over the first 24 hours, together with potassium replacement as indicated by plasma potassium estimation (approximately 20 mmol/l (meq/l) hour). She responded well, becoming fully orientated within 24 hours. She was clinically...
normal at this stage with blood pressure 130/70 mm Hg. Blood glucose concentration was 13 mmol/l (234 mg/100 ml) and bicarbonate 12 mmol/l.

She again became hypotensive, however, with blood pressure of 90/60 mm Hg and deteriorated over the subsequent 12 hours, despite continuation of the insulin infusion. Blood glucose concentration rose to 34.5 mmol/l (621 mg/100 ml) and bicarbonate fell to 9 mmol/l. Chest x-ray showed extensive bilateral pulmonary shadows. Her progress rapidly worsened despite treatment with gentamicin and fluocoxacin, culminating in cardiorespiratory arrest. After resuscitation she required ventilation and developed low cardiac output failure, which was treated with dobutamine. Introduction of this drug was associated with an appreciable increase in insulin requirement. Despite an infusion of 80 units of Actrapid MC hourly for five hours her blood glucose concentration remained raised at >30 mmol/l (>541 mg/100 ml). After improvement in cardiac function the dobutamine infusion rate was reduced. This was associated with an appreciable fall in blood glucose and insulin requirement. On complete withdrawal of dobutamine the insulin requirement fell to 2 units/hour (figure).

**Comment**

In this patient the temporal relation between the reduction in the dobutamine infusion rate and the precipitous fall in blood glucose concentration suggested a causal relation between the use of dopamine and insulin resistance. Hypotension, acidosis, and infection probably played only a small part in the insulin resistance. Furthermore, these factors remained constant at the time of the reduction in the dobutamine infusion rate and could not account for the sudden fall in blood glucose value.

Dobutamine is commonly used as an inotropic agent; glucose intolerance during its use has not to our knowledge been reported in man. Studies in dogs demonstrate no effect on blood glucose. The effect of dobutamine on the blood glucose in this patient may well have been dose dependent in view of the relatively normal insulin requirement at the lower rate of dobutamine infusion.

Other beta-adrenergic agonists (salbutamol, adrenaline, dopamine) increase insulin requirement in diabetics and may produce hyperglycaemia in normal subjects. In normal subjects this effect may be compensated for, as these agents also stimulate insulin release. In insulin-dependent diabetics, however, this compensatory mechanism is diminished or absent and the hyperglycaemic effect is likely to predominate. Whether or not dobutamine acts by stimulating glucose production or by other mechanisms—for example, as an insulin antagonist—remains to be investigated.

Our observations indicate the need for caution in the use of dobutamine and other beta-agonists in diabetes.


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Iliopsoas bursitis in rheumatoid arthritis: an unusual cause of leg oedema

Ankle swelling is a common diagnostic problem in rheumatoid arthritis. Though popliteal bursitis is a well-recognised cause of unilateral ankle oedema, disease of the iliopsoas bursa is an uncommon cause of hip pain, still less a cause of leg oedema. We describe a case of iliopsoas bursitis which presented clinically as an iliofemoral venous occlusion and which resolved with conservative management.

**Case report**

A 64-year-old woman with seropositive rheumatoid arthritis of 15 years’ duration was referred to the rheumatic diseases unit with increasing swelling of the right leg for three weeks. Fifteen months previously she had had a total condylar geometric arthroplasty of the right knee, after which she had noticed the slow progression of oedema of the leg and continuing pain in the knee.

On admission the right leg was grossly oedematous to the thigh, and a small tender mass was palpable in the right groin 2 cm below the inguinal ligament lateral to the femoral artery. Ascending venography showed extrinsic compression of the lateral aspect of the external iliac and femoral veins (figure). An ultrasound scan disclosed a cystic mass 8 cm long under the right iliopsoas muscle. This was aspirated and 200 ml viscous synovial fluid, sterile on bacteriological culture, was removed. Injection of contrast medium later confirmed the presence of an iliopsoas bursa which did not communicate with the hip (figure). The hip was clinically and radiologically normal. After bed rest and raising the leg the oedema resolved.

**Comment**

The iliopsoas or iliopsoas bursa lies anteriorly on the iliopsoas ligament of the hip capsule beneath the psoas and pectineus muscles.