SHORT REPORTS

Cyst infection in acquired renal cystic disease

Cystic degeneration of the kidney in patients with advanced renal failure is common¹; whether cysts can harbour infection remains to be shown.² We report on three cases of infected cysts in acquired renal cystic disease.

Case reports

Case 1—A 60 year old man was admitted to hospital because of uraemia, hypertension, recurrent fever, and leucocytosis. The plasma concentration of creatinine was 821 $\mu\text{mol/l}$, and a urine culture was positive for *Escherichia coli*. Ultrasonography showed normal sized hyperechogenic kidneys with more than three 1-2 cm cysts in each. There was a 6 cm cyst that was hyperechogenic at the upper pole of the left kidney. Renal computed tomography confirmed the presence of bilateral multiple cysts. A thick wall, enhanced by contrast infusion, was seen around a slightly dense cyst on the upper pole of the left kidney (figure).

Both kidneys are occupied by low density cysts. A thick wall, enhanced by contrast infusion, is seen around a slightly dense cyst (arrow).

Renal biopsy showed chronic glomerulonephritis with mesangial deposits of C3. Percutaneous aspiration of the cyst yielded purulent fluid which was positive for *E. coli*. He was given tobramycin; the fever disappeared, and the urine became sterile and the white cell count normal.

Case 2—A 60 year old woman with renal failure secondary to diabetic nephropathy was admitted to hospital because of fever and dysuria. Plasma creatinine concentration was 482 $\mu\text{mol/l}$, she had leucocytosis and pyuria, and a urine culture was positive for *E. coli*. A radiograph of the abdomen showed air in the left renal profile. Ultrasonography showed multiple renal cysts and a hyperechogenic parenchyma. Computed tomography confirmed the presence of the cysts and suggested an abscess at the lower renal pole and perirenal space of the left kidney. We removed a cyst, the fluid of which was infected by *E. coli*. Tobramycin was administered and the white cell count became normal and the urine sterile.

Case 3—A 78 year old woman presented with uraemia and creatinine values of 1120 $\mu\text{mol/l}$ and was started on haemodialysis. Ultrasonography showed small, hyperechogenic kidneys with multiple cysts. On admission she had a fever with no apparent cause. No urine cultures were available. She died a few days later of haemorrhage of the upper digestive tract. Necropsy showed two end stage kidneys with multiple corticomedullary cysts; an infected cortical cyst was found in the left kidney.

Comment

Although acquired renal cystic disease was initially described in patients undergoing haemodialysis, it can also affect patients with long term advanced renal failure.³ It is generally without symptoms but may present with pain associated with haemorrhage or neoplasia, or both. Infected cysts have not been reported before in acquired renal cystic disease.⁴

Cyst infection is a rare complication of simple renal cysts, and the

incidence of cyst infection in adult polycystic kidney disease has not been established. In either case patients have pyuria and most have a history of urinary tract infection. The infection is usually persistent and often progresses despite adequate antibiotic treatment because the antibiotic reaches the cystic fluid in only small amounts.

Differentiation of an infected cyst from a non-infected one may be difficult by ultrasonography: renal ultrasonography shows the presence of internal echos, and computed tomography detects thick walls or slightly dense cysts. A diagnosis can be confirmed only by puncturing the cyst. Continuous antibiotic treatment with percutaneous drainage of the cyst guided by ultrasonography or computed tomography is an alternative to surgical drainage and antibiotic treatment.⁵ Infected cysts should be suspected in patients with acquired cystic disease and persistent fever.

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Neonatal respiratory distress caused by aspiration of a vaginal tablet containing prostaglandin

The use of prostaglandin E_2 for cervical ripening and induction of labour is becoming more common. It may be given in a viscous gel,¹ vaginal tablets,² or pessaries³ and is considered both safe and effective.¹⁻⁴ We report a case of neonatal aspiration syndrome caused by a vaginal tablet containing prostaglandin.

Case report

A 28 year old woman who had had a caesarean section three years previously was admitted to hospital at 40 weeks of pregnancy because of onset of labour. External cephalic version had been performed without complications at 38 weeks' gestation; otherwise the course of the pregnancy had been uneventful. The cervix was ripe, and the baby's head was 2 cm above the ischial spines. A fetal cardiotocogram was normal. Because the patient was two days post term and the contractions remained irregular after six hours' observation, a vaginal tablet containing 3 mg of prostaglandin E_2 (Prostin E_2) was inserted into the posterior vaginal fornix. Uterine contractions improved, and 13½ hours later the cervix was 6 cm dilated. After amniotomy a normal amount of clear amniotic fluid escaped. Cardiotocography showed normal reactivity, but occasional, early decreases in fetal heart rate were also seen. Fifteen hours after insertion of the vaginal tablet the cervix was fully dilated, and 40 minutes later a girl weighing 3190 g was born in the normal vertex position. The umbilical cord was around the neck and was cut immediately after delivery of the head.

The baby had apnoea and bradycardia and an Apgar score of 5 at one minute. After receiving oxygen by mask her condition improved slightly, and at 10 minutes she had an Apgar score of 7. Her breathing remained irregular, however, and she began to cough; a white granular mass was seen in the mouth and pharynx. Despite suction of the upper airways respiration remained difficult, and the child was intubated at 11 minutes. An additional white mass resembling a tablet was suctioned from the lower airways until extubation 40 minutes after

birth. No meconium was found in the airways. A chest x ray picture at 40 minutes showed an infiltrate in the right lower lobe. After extubation her condition remained stable, and she was followed in the neonatal unit for four days. On discharge at 5 days of age the girl was healthy and the chest x ray picture was normal.

The bronchial aspirate was analysed by thin layer chromatography, which showed an unidentified substance that was also present in the vaginal tablet. No prostaglandin E₂ was shown, however, with this technique. The concentration of prostaglandin E₂ in the aspirate, as determined by specific radioimmunoassay (antiserum from Pasteur Institute, Paris),³ was 1.5 ng/ml, which fell within the range of 0.3-4.7 ng/ml in normal neonates (n=4).

Comment

We believe that this is the first reported case of neonatal aspiration syndrome caused by undissolved remnants of a vaginal tablet containing prostaglandin. Results of thin layer chromatography suggested that the granular material suctioned from the airways contained the vehicle substance of the tablet. The clinical signs were the result of mechanical obstruction of the airways and were not caused by prostaglandin E₂. This conclusion is supported by the normal concentration of prostaglandin E₂ in the aspirate. Thus the tablet had probably released its prostaglandin into the vagina, and this apparently promoted the labour. Remnants of the vehicle substance were then transferred to the mouth of the baby while the head was passing through the vagina. The subsequent aspiration, which occurred during the baby's first breaths, was promptly treated by intubation and suction of the airways.

Aspiration syndrome is one of the most common emergencies in the delivery room. This potentially life threatening condition may also be iatrogenic, as shown in this case report. Obstetricians and paediatricians should be aware of the risk of using prostaglandin tablets, a risk that may be avoided by replacing prostaglandin tablets with prostaglandin viscous gel, which disappears from the vagina more quickly than tablets.⁴

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Treatment of sensorineural deafness associated with ulcerative colitis

There is evidence that the pathogenesis of inflammatory bowel disease and its extraintestinal manifestations depend on immunological mechanisms. Recently, sensorineural deafness was added to the list of extraintestinal complications of ulcerative colitis, and as the disease is also associated with rheumatoid arthritis and polyarteritis nodosa it may have an immune basis.

In two of three cases the hearing loss associated with ulcerative colitis improved with steroids either alone¹ or in combination with cyclophosphamide.² The precise relation between improved hearing and drug treatment, however, was not studied in detail. As the hearing loss may result in serious morbidity prompt and appropriate treatment of this complication is essential. We describe the relation between hearing loss in ulcerative colitis and treatment with prednisolone and sulphasalazine either alone or in combination with azathioprine.

Case report

A 42 year old man who presented with a 17 year history of "colitis" and with symptoms of diarrhoea and rectal bleeding was found to have total ulcerative colitis of moderate severity on colonoscopy, which was confirmed by biopsies. His symptoms and macroscopic disease responded promptly to a short course of prednisolone and sulphasalazine. Four months after presentation he developed vertigo, nystagmus, and ataxia of sudden onset followed by complete deafness in the right ear and progressive deafness in the left. Neurological investigations, including a lumbar puncture and computed tomography of the brain, yielded normal results, but audiometry showed sensorineural deafness with complete loss of hearing in the right ear and 15 decibel loss in the left ear for most of the speech frequencies. His hearing remained stable at this level, and he was discharged taking sulphasalazine, the ulcerative colitis being quiescent at that time.

Three months later his hearing suddenly deteriorated. His ulcerative colitis remained in remission, autoantibody levels were normal, and immune complex titres were undetectable. On the assumption that his hearing loss was an extraintestinal manifestation of ulcerative colitis, prednisolone 60 mg/day was started. His hearing rapidly improved with this regimen but deteriorated whenever the dose was reduced below 40 mg/day (figure). The addition of azathioprine (2 mg/kg/day) allowed the steroid treatment to be slowly reduced and then stopped without appreciable deterioration in hearing (figure). Sulphasalazine was continued throughout in a dose of 1 g thrice daily. The hearing loss remained stable for over nine months despite an exacerbation of ulcerative colitis.

Prednisolone dosage and loss of hearing before and during treatment with azathioprine.

Comment

This report provides a further example of sensorineural hearing loss associated with ulcerative colitis. It shows dissociation between the activity of the colitis and impairment of hearing and the responsiveness of the disorder to prednisolone and azathioprine. In a young patient sudden loss of hearing results in severe disability and salvaging of residual auditory function is vital. On the basis of this experience we would recommend immediate treatment of such sensorineural deafness with a combination of azathioprine and prednisolone.

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