result[7]; almost half of our patients acquired their infection in hospital.

Previous reports accept that *B catarrhalis* infection occurs in immune compromised hosts[8,9] or in patients with chronic pulmonary disorders,[2,3] but our finding of infection in otherwise healthy non-smoking adults, supported by others,[3] suggests that there may be an increase in the number of more virulent strains. Historically, the virulence of *B catarrhalis* seems to have fluctuated. In the early part of this century it was associated with epidemics of severe "flu like" illness.[10] Thereafter its appearance in reports, usually in cases of meningitis, endocarditis, or conjunctivitis, was sporadic until the mid-1970s. Since then the number of reports from different countries suggests that more virulent organisms are appearing rather than simply reflecting an increased tendency to report them.

The pathogenic mechanisms of *B catarrhalis* are still poorly understood. Its deoxyribonuclease production enables it to inflame mucous membranes. Unlike *Neisseria gonorrhoeae* and *Neisseria meningitidis*, *B catarrhalis* does not produce IgA1 proteases, which cleave IgA1 into Fab and Fc portions and thus breach the protective immunoglobulin barrier of the mucous membrane.[11]

Other members of the family Neisseriaceae have not been studied as extensively as *N gonorrhoea* and *N meningitidis* as generally they have been regarded as harmless organisms of little clinical import. As *B catarrhalis* has recently been causing disease more often than is normally expected microbiologists and clinicians should not dismiss it too readily as normal flora.

The reason for this resurgence is obscure, though the role of antibiotic pressure warrants careful scrutiny. Ampicillin has been used widely in the Edinburgh area, and this may have played a part in increasing the number of strains of *B catarrhalis* producing β lactamase. Similarly, trimethoprim, though not used as extensively in Edinburgh, has been shown to induce bacterial resistance to itself.[12] The increasing use of such antibiotics may alter the nasopharyngeal flora sufficiently to lead to an increased incidence of infections with *B catarrhalis*. Further studies are required to test this hypothesis.

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SHORT REPORTS

Metabolic acidosis due to D-lactate

Metabolic acidosis caused by D(−)-lactic acid has been well documented in patients after bowel surgery.[1] I describe a patient who had not undergone bowel surgery but developed D(−)-lactic acidosis; this has not been reported before.

Case report

A 60 year old man had a 10 year history of chronic pancreatitis; he had undergone cholecystectomy nine years previously and a Peustow procedure (filleting of the pancreatic duct and jejunal anastomosis) seven years previously. He had also been treated with pancreatic enzymes and cimetidine. He developed chronic renal failure (interstitial nephritis), and haemodialysis was started when he was 59; shortly afterwards he received a cadaveric renal allograft. Immunosuppression with azathioprine and prednisolone was started. The early course after transplantation was complicated by four episodes of rejection, each treated with methylprednisolone, and by the development of diabetes mellitus, which was treated with insulin.

Two months after transplantation he had been well with stable but poor graft function until he was readmitted after a week of lethargy and nausea and vomiting. On examination he did not have a fever but was poorly perfused, cyanosed, tachypnoeic (48 breaths/min) and hypotensive. His haemoglobin concentration was 125 g/l, white cell count $3{\cdot}4\times10^9$/l, sodium concentration 141 mmol(mEq)/l, potassium 4·4 mmol(mEq)/l, chloride 103 mmol(mEq)/l, bicarbonate 13 mmol(mEq)/l, urea 21 mmol/l (126 mg/100 ml), and creatinine 193 μmol/l (2·2 mg/100 ml). He also had acidosis with pH 7·3, oxygen tension 11·4 kPa (86 mm Hg), carbon dioxide tension 2·7 kPa (20 mm Hg), standard bicarbonate 14 mmol(mEq)/l, and base excess −16 mmol(mEq)/l; the anion gap was 29 mmol(mEq)/l, and L(+)-lactate concentration was low at 1·1 mmol/l (9·9 mg/100 ml). There was no evidence of salicylates, β-hydroxybutyrate, or ketones in the blood, and the glucose concentration was 6·8 mmol/l (123 mg/100 ml). The D(−)-lactate concentration was 6·8 mmol/l (61 mg/100 ml) (normal <0·2 mmol/l (<1·8 mg/100 ml)).

He was treated for suspected sepsis (subsequent blood cultures grew *Escherichia coli*) with broad spectrum antibiotics (ampicillin, flucloxacillin, and gentamicin) and was given sodium bicarbonate. After a respiratory arrest requiring ventilation he was given haemodialysis with bicarbonate dialysate. He deteriorated, became anuric and hypotensive, and developed progressive lung shadowing; he died two days later.

Method of measuring D(−)-lactate—The D(−)-lactate concentration was measured by substituting D(−)-lactate dehydrogenase (EC 1.1.1.28, Boehringer Mannheim Biochemicals, No 106941) for L(+)-lactate dehydrogenase (EC 1.1.1.27) in the kit for estimating lactate (Boehringer Mannheim Biochemicals test combination No 139084).

Comment

D(−)-lactic acidosis has been well described in association with the short bowel syndrome after jejunoileal bypass surgery, most commonly for obesity.[1-5] The syndrome has not been reported in patients who have not had bowel surgery. High concentrations of D(−)-lactate have been associated with dizziness, ataxia, dysarthria, fatigue, confusion, headache, and other neurological syndromes.[1,3,4] The D(−)-lactate has been assumed to result from the metabolism of carbohydrate by bacteria abnormally found in the small bowel, or after the rapid transit of carbohydrate to the large bowel. If the amount of D(−)-lactate produced exceeds the capacity for further metabolism then it will be absorbed.

The acidosis in this patient was thought to be disproportionate to the degree of renal impairment. Blood was therefore screened for other possible acids, including D(−)-lactic acid, which is a cause of acidosis.[1] D(−)-Lactate is normally undetectable,[4] and the measured concentration of 6·8 mmol/l (61 mg/100 ml) suggests that this contributed greatly to the acidosis. Unfortunately the organism cultured from his blood was not characterised for its ability to produce D(−)-lactate. *E coli* is not known to produce D(−)-lactate, and the source may have been another gut organism. This patient had not undergone surgery resulting in anatomical or functional bowel shortening, and, although the Peustow procedure may sometimes be associated with creation of a blind loop, he did not have a history of diarrhoea or other suggestive features. The source of the D(−)-lactate is therefore speculative.

D(−)-Lactic acidosis may be more common than is thought and perhaps should be looked for in cases of metabolic acidosis in which the identity of the acid is not apparent.

I thank Professor J S Cameron, Dr C S Ogg, and Dr D G Williams for permission to report this case and Dr Pat Toseland for measuring the D(−)-lactate concentrations.

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Diurnal variation in onset of acute closed angle glaucoma

Acute glaucoma results from closure of the angle in the anterior chamber of the eye by apposition of peripheral iris to cornea, which prevents the outflow of aqueous humour from the anterior segment; with continuing production of aqueous humour the intraocular pressure rises precipitously, producing the characteristic symptoms and signs of closed angle glaucoma—namely, decreased visual acuity, haloes around lights, and pain, which is often severe.[1] Diurnal variation of intraocular pressure is well recognised[2]; we attempted to determine whether the onset of acute closed angle glaucoma varies on a similar diurnal cycle.

Patients, methods, and results

One hundred consecutive patients (mean age 66·3 (SD 10·1) years) presenting with acute closed angle glaucoma referred to this glaucoma unit during the eight years to 20 December 1983 were analysed retrospectively. Each patient was questioned individually, by a single observer, to determine the time of day (to the nearest hour) when symptoms of acute glaucoma became apparent; as angle closure produces rapidly progressive, severe effects the onset of symptoms is a reasonable approximation to the start of the acute attack. Details were verified from patients' records. Acute closed angle glaucoma was significantly more prevalent among women than men (71 women *v* 29 men; $\chi^2=17{\cdot}64$, df=1, $p<0{\cdot}001$).

The table shows the diurnal variation in the time of onset of acute closed angle glaucoma, with a peak incidence during the evening and a trough in the morning.

Hourly distribution of onset of acute closed angle glaucoma over eight years

Time	No of patients	Time	No of patients
0100	2	1300	
0200	6	1400	
0300	1	1500	2
0400	2	1600	6
0500		1700	6
0600	2	1800	11
0700	8	1900	13
0800	4	2000	13
0900		2100	6
1000	2	2200	6
1100	1	2300	3
1200	1	2400	5

Comparison of the 12 hour periods 0400 to 1500 and 1600 to 0300 yielded a significant difference ($\chi^2=31{\cdot}36$, df=1, $p<0{\cdot}001$). The hypothesis of a constant incidence throughout the day was not reasonable ($\chi^2=86{\cdot}24$, df=23, $p<0{\cdot}001$), but when the logarithms of the observed counts were submitted to regression on a sine (hour) and cosine (hour) scale jointly on a 24 hour cycle assuming Poisson type errors, the regression was highly significant ($\chi^2=35{\cdot}83$, df=2, $p<0{\cdot}001$).

Comment

The depth of the anterior chamber of the eye is a dynamic variable that may change rapidly and transiently.[3] Diurnal shallowing of the anterior segment has been shown; the depth and volume of the anterior chamber are significantly lower in the evening than the morning, and the anterior chamber angle decreases by 21%.[4] This obviously facilitates the onset of angle closure; individual differences in diurnal shallowing of the anterior chamber may explain why certain eyes progress to overt angle closure though other, apparently similar, eyes do not.

Closed angle glaucoma is a medical emergency; during a typical episode intraocular pressure may be above 50 mm Hg (normal 10-20 mm Hg), producing irreversible ischaemic ocular damage within a few hours. This is the first significant evidence to confirm the hypothesis that acute glaucoma occurs mainly during the evening, with the following implications. Delay in management overnight may result in considerable impairment of sight; effectively, closed angle glaucoma is a form of blindness that may be prevented but only if management is started at an early stage. The onset of closed angle glaucoma is not easily recognised, but suspicious features include pain or decreased visual acuity with a fairly acute onset during the evening. A history of similar self limited episodes occurring at the same time of day, suggesting intermittent partial angle closure, would reinforce the diagnosis. Immediate ophthalmic treatment serves only to arrest the rapidly progressing ocular damage; of greater importance is early recognition by the general practitioner and immediate referral.

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Source of infection in candida endophthalmitis in drug addicts

Heroin addiction is currently a major medical and social problem in Great Britain. An epidemic of presumed candidal endophthalmitis, a recognised complication of heroin abuse, was recently reported from Glasgow.[1] In this series an appreciable temporospatial clustering of patients was observed, suggesting a common source of infection. We report the results of our attempts to ascertain the possible origins of the infection in addicts.

Patients, methods, and results

Twenty three heroin addicts were diagnosed as having candidal endophthalmitis between November 1982 and August 1985. The diagnostic criteria have been described.[1]

Analysis of drug diluent—The diluent used by 22 addicts was lemon juice from plastic lemons. Five patients submitted their current lemon for analysis; the caps and juices were cultured for fungal contamination. Two lemons yielded pure, heavy growths of *Candida albicans*. Two lemons were nearly full, indicating that they had been bought recently, and one was completely empty; these three lemons yielded negative results on culture. As a control seven plastic lemons bought six to 12 months previously were obtained from friends and colleagues; only one produced a growth of a non-pathogenic yeast. Of 15 lemons bought at random from corner shops in the area where the addicts lived, 11 were within their "best by" dates and yielded negative results on culture; *Aspergillus niger* was cultured from the juice of one of the four lemons past their "best by" dates.

Fungicidal effect of lemon juice—Sulphur dioxide is the preservative used in the plastic lemons. Juice from new lemons was inoculated with *C albicans* and incubated for 48 hours; no growth was seen. The juice from new lemons that had been steamed for 10 minutes to drive off the preservative did not inhibit growth of candida.