SHORT REPORTS

Chocolate bars contaminated with Salmonella napoli: an infectivity study

Before 1982 the isolation of *Salmonella napoli* from human sources was very rare in the United Kingdom. Between April and mid-September 1982, 245 laboratory reports of *S napoli* isolations had been recorded (PHLS Epidemiological Diseases Surveillance Centre, unpublished data). Epidemiological investigation showed a strong association with the consumption of chocolate bars manufactured in northern Italy, and on 23 July 1982 the Department of Health and Social Security issued a press release warning the public not to eat Tommy Junior or Rocky Junior chocolate bars.

As a consequence of this publicity a box of Rocky Junior milk chocolate bars was surrendered voluntarily by a market stallholder. The retailer was also the source of Rocky Junior bars which had been eaten by two local patients suffering from *S napoli* infection. *S napoli* was isolated from some of the bars. Preliminary investigations suggested that the number of salmonellae present in each chocolate bar was very small. As chocolate is a microbiologically stable product we decided to attempt to establish the probable infective dose by examining individual chocolate bars for *S napoli*.

Methods and results

Six chocolate bars from each of eight packets of Rocky Junior chocolate bars were examined. Each bar was weighed and homogenised in sufficient 1% peptone water to form a 1/10 dilution. Volumes of 10 ml, 1 ml, and 0·1 ml of this homogenate were added to three tubes each of selenite broth. After incubation and subculture of the homogenates and selenite broths on to selective agar media the presence of *S napoli* was confirmed serologically by slide agglutination. The most probable number of *S napoli* per 10 g of each chocolate bar was estimated using tables.[1]

S napoli was isolated from 42 of the 48 Rocky Junior chocolate bars examined. The table shows the most probable number of *S napoli* per 10 g of each chocolate bar. The results indicate an average of 1·6 *S napoli* organisms per gram. The mean weight of the individual bars was 16·0 g (range 12·3-18·4 g). Of the 42 bars containing salmonellae, 28 contained fewer than 10 *S napoli* and 12 contained 10-40 *S napoli*.

Most probable number of Salmonella napoli per 10 g of each chocolate bar

Bar	Packet No							
	1	2	3	4	5	6	7	8
A	15	4	ND	4	23	+	9	23
B	240	4	3	4	9	+	+	7
C	15	ND	3	4	4	4	7	9
D	15	ND	4	+	3	4	4	3
E	23	+	4	ND	+	4	3	15
F	43	+	4	ND	3	ND	+	4

ND = Not detected.
+ = Detected in peptone homogenate only.

Comment

Immediately after the press release *S napoli* was isolated from a boy aged 10 who had eaten two Rocky Junior bars on each of two consecutive days. His brother, aged 13, and his mother had also eaten two bars each on one day only but did not have symptoms. Examination of faecal specimens showed that the brother was also excreting *S napoli*.

Theoretically it requires only one viable salmonella bacterium to reach the small intestine and begin its rapid multiplication to initiate symptoms. In practice large numbers of salmonellae may be ingested without untoward effect. Studies using spray dried contaminated egg[2] suggested that in normal healthy people 10^6 salmonellae may be required to produce symptoms. Investigations after an outbreak of *S eastbourne* infection due to contaminated chocolate[3] suggested that an inoculum of as few as 1000 organisms was enough to cause illness.

Gastric acidity plays an important part in killing the organisms before they have had an opportunity to colonise the lower gastrointestinal tract. Virulence also plays a part. Evidence suggests that milk chocolate confers some protection on the salmonellae, allowing long term survival in the product and resistance to gastric acidity.[4 5]

An estimate based on the average value of 1·6 *S napoli* organisms per gram of chocolate bar obtained in this study suggests that the infective dose for our patients was roughly 50 organisms. This is considerably lower than those previously suggested as necessary to cause salmonellosis in otherwise healthy people.

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Infant chlamydial pneumonia

Pneumonia in infancy caused by *Chlamydia trachomatis* has rarely been suspected in Britain,[1 2] though it is fairly common in the United States of America.[3] We report on a patient with typical clinical and laboratory features.

Case report

A 7 week old Negro baby presented in May 1982 with a seven day history of cough that had worsened over the previous 48 hours, when he had become noticeably tachypnoeic. The cough was paroxysmal, suggesting pertussis, but he did not whoop. Until this illness he had been well; he had been born at term in hospital by normal delivery, the birth weight being 3940 g. He had developed sticky eyes shortly after birth, which had been treated with drops of physiological saline. Neither parent had symptoms of sexually transmitted disease. His mother's puerperium had been normal, and there were no previous pregnancies.

Examination showed a quiet baby still trying to feed despite respiratory distress. He was cyanosed with a respiratory rate of 80/min; the chest was hyperexpanded with generalised crackles on auscultation. Apart from a tachycardia of 140 beats/min findings were otherwise normal, and he remained afebrile throughout.

There was an initial leucocytosis of $24{\cdot}6 \times 10^9$/l with mild eosinophilia of 3% (0·738), 43% neutrophils, 47% lymphocytes, and 7% monocytes. Six days later the total white cell count was $14{\cdot}7 \times 10^9$/l with 10% eosinophila (1·47). Blood gas analysis disclosed hypoxia: oxygen tension was 6 kPa (45 mm Hg), carbon dioxide tension 5·5 kPa (41 mm Hg), and pH 7·42; the hypoxia slowly improved over four days. Radiography showed diffuse interstitial shadowing with small nodules throughout both lung fields. Pernasal swabs failed to grow *Bordetella pertussis*. Swabs of pernasal secretions were inoculated, within four hours of collection, on McCoy cells treated with cycloheximide. Cultures were examined by Giemsa staining and at 48 hours showed inclusions typical of chlamydial infection. The table shows results obtained in serum specimens taken a week after the onset of symptoms in the

Serological results (reciprocal titres in microimmunofluorescence test)

Antigen	Infant		Mother
	IgG	IgM	IgG
C trachomatis A-C	64	8	256
C trachomatis D-K	256		6400
Lymphogranuloma venereum 1-3	128	8	256
Herpes simplex virus 1 and 2	0		

patient and several weeks later from the mother. Genital swabs from the mother were negative for *Neisseria gonorrhoeae*, *Trichomonas vaginalis*, and *Candida*.

The baby was initially thought to have severe bronchopneumonia and was treated with parenteral flucloxacillin and amoxycillin, oxygen, and nasogastric feeding. His condition improved slowly. When the results of chlamydia culture became available treatment was changed to oral erythromycin. He recovered and was well at follow up.

Comment

Chlamydial pneumonia of infancy has been well characterised since its first full description,[3] and our patients' illness closely resembled the most commonly described pattern. The disease usually occurs in the second or third month of life. Respiratory symptoms such as rhinitis or mild cough gradually increase, and in the fully developed syndrome the infant, who remains afebrile, develops tachypnoea with a paroxysmal cough. This cough bears some resemblance to pertussis, but whooping is not a feature, though apnoeic attacks may occur. The blood count characteristically shows mild eosinophilia, and the chest radiograph is often abnormal with bilateral interstitial shadows and hyperinflation. Moderate hypoxia with a normal arterial carbon dioxide tension is found.

Isolation of *C trachomatis* from the nasopharynx is usually accepted as evidence of the disease, but this is strengthened by additional serological findings suggestive of recent infection in the form of specific IgM antibody. In this case there was also an exceptionally high maternal antibody concentration.

C trachomatis has now emerged as a common cause of pneumonia in infants in the United States of America, especially in those groups with the highest incidence of sexually transmitted infection.[4] It seems unlikely that this infection is as rare in this country as the lack of previous reports would indicate since the illness is often mild and develops long after the baby has left hospital. A careful prospective survey would be timely.

We thank Professor Darougar of the Institute of Ophthalmology for performing the serological tests.

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Galactorrhoea as side effect of domperidone

Domperidone (Motilium) has recently become available for use in Britain. It is a potent peripheral dopamine antagonist which has gastrointestinal kinetic properties.[1] Unlike the similar agent metoclopramide, domperidone does not cross the blood-brain barrier and is therefore virtually free of central adverse effects.[1] To our knowledge galactorrhoea has been reported on only one previous occasion,[2] and the *British National Formulary* (No 4, 1982) does not mention this adverse effect.

In a recent clinical trial (paper in preparation) we compared the effect of domperidone (20 mg four times a day) with identical placebo in 30 patients (28 women, two men; median age 35, range 19-61) with the irritable bowel syndrome. Five of these patients complained of galactorrhoea and mastalgia and two of mastalgia alone while taking the domperidone. These effects were reported spontaneously and patients had no reason to anticipate any problems with their breasts. We give a summary of the case histories of these patients together with the results of prolactin assays carried out in all those patients with adverse effects and 11 of the other women patients.

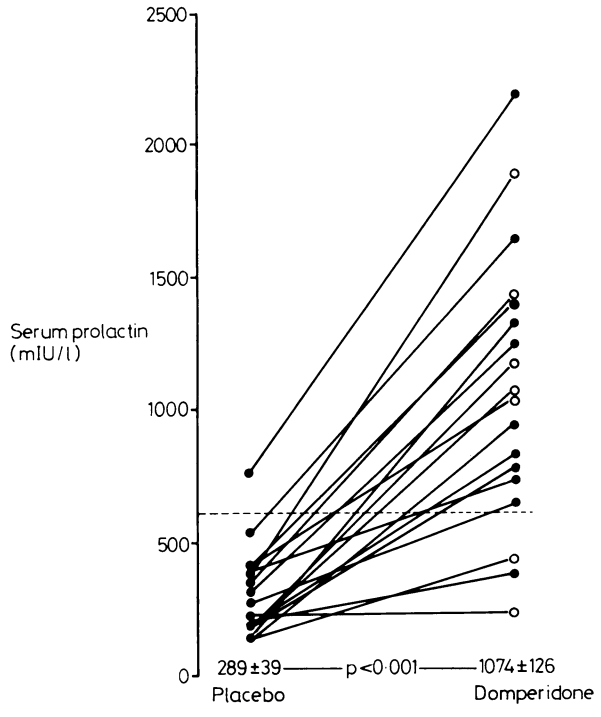
Blood samples were taken during the fourth week of each treatment, within one to two hours after an oral dose, the domperidone and placebo being given in double blind cross over fashion.

Case reports

Two women (aged 22 and 48) developed swollen, tender breasts after three and seven days of domperidone treatment. In both cases the condition had disappeared one week after stopping the drug. Five other women (aged 19-42) developed mastalgia and galactorrhoea, varying from mild and intermittent to a profuse production of milk requiring several changes of clothing a day. In four of these patients the adverse effects developed between three days and two weeks after starting treatment. The other patient had no adverse reactions during the four week trial period but developed galactorrhoea after three months of continuous treatment, having elected to continue with the drug after the clinical trial. In all five cases the galactorrhoea had settled within one week to two months after stopping treatment.

Three of these five patients reported a disturbance in menstruation while taking domperidone. One noted a delayed period, one an early and heavy period, and the other an unduly protracted and heavy period at the expected time. All patients subsequently returned to a regular menstrual pattern once domperidone was stopped.

During domperidone treatment the serum prolactin concentration was abnormally high in 15 of the 18 subjects in whom it was measured (figure). Only one patient had a raised concentration during the placebo period; in her case, however, placebo was given after domperidone, so that the concentration might not have returned to normal. Two patients with breast symptoms had normal prolactin concentrations. There was no direct relation between the actual value and the occurrence of adverse effects.



Prolactin concentrations after four weeks of treatment with placebo and four weeks with domperidone (20 mg four times daily). Dashed line marks normal reference range (< 600 mIU/l). ● ——— ● Subjects without adverse reactions. ● ——— ○ Subjects reporting galactorrhoea/mastalgia.

Comment

We were not surprised that domperidone, like metoclopramide, increased the serum prolactin concentration.[3] Both drugs are thought to have this effect by antagonising dopamine in the median eminence,[4] which lies outside the blood-brain barrier.

Six of the women with adverse reactions complained predominantly of constipation. Constipated patients may in some way be sensitised to