Incidence of cysteine dependent Escherichia coli in a general practice population

Cysteine dependent isolates of Escherichia coli associated with urinary tract infection are now well recognised.1 Media supplemented with cysteine have been specially formulated to detect these strains.2 3 We undertook a study to determine the incidence of cysteine dependent isolates from patients in a number of general practices in Cardiff. This study was also designed to evaluate the need to use a cysteine supplemented medium in preference to a bile salt medium (such as MacConkey agar) for the processing of specimens from such a population.

One hundred and twenty specimens of mid-stream urine from patients with symptoms suggestive of a urinary tract infection and an appreciable degree of pyuria (white cell count of > 10 per mm3) were plated on to aerobic blood agar and MacConkey agar and incubated overnight. The urine samples were then refrigerated. The next day, if there was no growth on blood or MacConkey agar and no evidence of previous antibiotic treatment on the request form, the urine sample was plated on to cysteine and lactose electrolyte deficient (CLED) agar and incubated for a further 24 hours. The sample was not cultured for Mycobacterium tuberculosis since in this laboratory M tuberculosis is rarely isolated in cases of sterile pyuria.4 If any colonies of E. coli appeared on the CLED agar they were subcultured to MacConkey agar to determine whether they were genuine cysteine dependent isolates. Such strains do not grow on this medium and none was obtained from any of the 120 urine samples in this study.

It appears that the incidence of auxotrophic strains of E. coli is probably low in the population served by our laboratory. This accords well with the observation that these organisms are rarely seen in acute urinary infections, being more commonly implicated in chronic conditions, especially after use of indwelling urinary catheters.5

The results of this study also suggest that the use of cysteine supplemented agar media has no particular advantage over MacConkey agar for patients from a general practice population presenting with acute urinary tract infections.

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References

Factors II, VII, IX, and X concentrations in patients receiving long term treatment with warfarin

We read with interest the paper by Paul et al about concentrations of factors II, VII, IX, and X in patients receiving long term treatment with warfarin.1 We would like to report the results of our study in which we compared activated partial thromboplastin times (APTTs) and British ratios in 50 patients receiving long term warfarin treatment.

The patients selected were in the therapeutic range2 and had been so on at least three occasions in the previous six months. British ratios were measured using the Manchester comparative reagent and APTTs using the Manchester APTT reagent. Results showed a good correlation between the APTT and the British ratio (r = 0.74).

We then selected the 15 patients with the highest APTTs and measured factor IX concentrations in their stored plasma samples. Two half factor IX concentrations of 1-6 IU/l and 25-0 IU/l; their British ratios were 4-4 and 2-3, respectively.

Thus patients with a well controlled prothrombin time (which is not sensitive to factor IX) may have considerably reduced factor IX concentrations. We agree with Paul and her colleagues that we should not assume that patients receiving long term warfarin treatment have an equal reduction of their vitamin K dependent factors; some may be more depressed than others, despite “good control.”

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Microvascular thrombosis of the bowel in paroxysmal nocturnal haemoglobinuria

Paroxysmal nocturnal haemoglobinuria is an acquired disorder in which erythrocytes, granulocytes, and platelets show increased sensitivity to complement. The clinical features of this condition are haemolysis and marrow hypoplasia. A major complication is venous thrombosis, which may affect unusual sites such as hepatic and mesenteric veins.1 Many patients also suffer from recurrent abdominal pain, and, while intestinal thrombosis has been suspected as the cause,2 there is little direct evidence for this. We report the histological findings in a patient with paroxysmal nocturnal haemoglobinuria who successfully underwent resection of thrombosed ischaemic small bowel.

The patient, a 37 year old man, had had hypoplastic anaemia since the age of 11. In 1980 ferrokinetic and chromium studies showed shortened red cell survival as well as poor erythropoiesis, but the result of a Ham test (for paroxysmal nocturnal haemoglobinuria) was negative. He first complained of abdominal pain in 1984. Ultrasound examination showed patent hepatic and portal veins, but endoscopy disclosed pronounced duodenitis. The result of a Ham test was then positive, thus confirming the diagnosis of paroxysmal nocturnal haemoglobinuria. In 1985 he developed severe acute abdominal pain, with guarding and rebound tenderness. Despite the low white cell and platelet counts, emergency laparotomy was successfully performed. The entire small bowel was inflamed, and two lengths of ileum, 30 cm and 40 cm long, were resected.

In the resected ileum there were several lengths (up to 8 cm long) in which the gut wall was thickened due to submucosal oedema and haemorrhage but with no muscular hypertrophy. There was irregular ulceration of the mucosa, varying from...