Letters to the Editor

Iatrogenic prostatic granulomata

In recent years attention has been drawn to the occurrence of iatrogenic prostatic granulomata after transurethral resection of prostate. We report three cases of iatrogenic granulomatous prostatitis caused by intravesical instillation of Bacillus Calmette-Guerin (BCG) for flat carcinoma in situ. Subsequently two of these patients had transurethral resection for chronic prostatitis and one patient had a radical cystectomy with prostatectomy for invasive bladder carcinoma.

Histological examination showed caseating and non-caseating granulomata of varying size. These were composed of aggregates of epithelioid and foreign body giant cells with surrounding lymphocytes and plasma cells. The granulomata were found both in the suburothelial connective tissue and in intimate association with the prostatic ducts and acini (figure). Stains for acid fast bacilli were negative in all three cases.

The exact mechanism by which intravesical BCG causes granulomatous prostatitis is not understood. It may be a systemic effect of BCG, or a result of reflux of urine containing BCG into the prostatic ducts. BCG is being used with increasing frequency in the treatment of superficial bladder cancer. Histopathologists must be aware that this treatment is a cause of granulomatous prostatitis when interpreting granulomata in the prostate.

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References

Diverticular disease of the right colon

Diverticular disease of the left colon is a common condition, but diverticulosis confined to the right colon is rare. I report two cases of the latter that I saw in quick succession.

Both were white men who presented in the same way. The first was a 78 year old man who had profuse haemorrhage from the bowel. Diverticular disease of the right colon was diagnosed preoperatively and right hemicolectomy carried out. The second was a 57 year old man who presented with uncontrollable rectal haemorrhage for which a subtotal colectomy was eventually carried out. In this case the cause of the bleeding was not diagnosed preoperatively, but angiodysplasia was suggested.

In both resected specimens there were diverticula in the right colon. They were present throughout the hemicolecetomy specimen, which measured about 30 cm and were found in the first 30 cm of the subtotal colectomy specimen. Compared with left sided disease, there were comparatively few diverticula in both specimens, and their openings were identified on the mucosal surface as small pits arranged in straight lines in the long axis of the bowel. In the subtotal colectomy specimen the mucosa surrounding the diverticular orifices was heaped up and small sessile polyps were also found. In contrast to left sided disease there was no muscle hypertrophy or exaggeration of the haustral pattern.

Removal of the serosal fat showed that the diverticula were thin walled sacs, some of which were extremely elongated and could be likened to mirror image polyps projecting from the smooth serosal surface (figure). As in left sided disease they were situated between the longitudinal muscle bands. Microscopic examination showed a continuous muscle coat that was attenuated in some of the diverticula. The small polyps found in the subtotal colectomy specimen were metaplastic.

The cause of the haemorrhage in the
Lipaemic interference: effects of lipaemic serum and Intralipid

Lipaemia interferes with a variety of clinical chemistry methods. Most studies evaluating the effect of lipaemia use Intralipid 20% to simulate the turbidity effects of increased triglyceride concentrations. In reality, any interference with testing is likely to be the result of chylomicron accumulation in serum. Whether the interference from Intralipid is comparable with the interference seen in lipaemic serum samples is unknown. We carried out a study to compare the interfering effects of lipaemic serum samples and lipaemia simulated by Intralipid on various common tests on desk top analysers.

The instruments evaluated were the Abbott Vision (Abbott Laboratories, North Chicago, Illinois, USA), the Reflotron analyser (Boehringer Mannheim Canada, Inc, Dorval, Quebec), the Kodak DT60 analyser (Eastman Kodak Co, Rochester, New York, USA) and the Ames Seralyzer (Ames Division, Miles Laboratories Elkhart).

The Vision uses individual test packs into which capillary tubes with whole blood, plasma, or serum are inserted. The blood is spun down inside the analyser and mixed with reagents. The absorbance is read by a photometer and the result calculated and printed. Tests evaluated were glucose, urate, alkaline phosphatase, urea nitrogen, and cholesterol.

The Reflotron analyser is a photometer controlled by a microprocessor that uses test strips. The strip—which accepts blood, serum, or plasma—is inserted into the analyser and the coloured product of the reaction is measured by the photometer. The tests evaluated were glucose, urea nitrogen, cholesterol and y-glutamyltransf erase.

The Kodak DT60 analyser uses special slides for performance of tests on serum or plasma. All reactions take place within the multilayered elements of the slide. The bar code reader in the analyser automatically identifies the test to be performed; all processes are controlled by the self-contained microprocessor. The tests evaluated were glucose, urate, potassium, bilirubin, creatinine, and amylase.

The Seralyzer is a reflectance photometer and uses test strips. For each test a different test module is inserted into the analyser. This module identifies the test measurements, processes the reflectance signals from the test strip, and calculates the result. The test strips use serum or plasma. The tests evaluated were glucose, creatinine, aspartate aminotransferase, theophylline, phenytoin, and phenobarbital.

Simulation of turbidity was done by adding 10% Intralipid (Cutter Laboratories, Berkeley, California, USA) to pooled sera with known concentrations of analyte. A triglyceride concentration of 3-4 mmol/l was obtained. Concentrations of all analytes tested on each of the instruments were measured.

Patient who had a subtotal colectomy was mucosal ulceration in at least one diverticulum; blood had, in fact, been noted in some of the diverticula when blocks were taken. This must also be the explanation in the other case even though no definite ulceration was seen in the diverticula sampled.

Perry and Morson suggested that diverticular disease confined to the right colon should be regarded as a distinct form of diverticulosis, to be differentiated from classic left sided disease, from the more extensive disease that also affects the transverse colon, and from solitary diverticula. The findings in these two cases support this view, and it is noteworthy that one of the two cases described by Perry and Morson presented with profuse rectal haemorrhage. Right sided diverticular disease should not be overlooked as a possible cause of unexplained bowel haemorrhage.

Letters to the Editor

To test the effect of lipaemic sera, blood was drawn from normal volunteers one hour after ingestion of 600 ml of 18% butterfat ice cream. Serum was separated and used for studies testing interference by "lipaemic serum". To obtain the same target concentration of triglycerides (3-4 mmol/l) for the lipaemic serum, it was appropriately diluted with aqueous assayed material that had known concentrations of analytes to be tested. Concentrations of all analytes were then measured on the above mixture. To assess the degree of interference, lipaemic serum samples and Intralipid were then added to plasma based quality control material with known concentrations of analyte. The final concentration of triglycerides was 3-4 mmol/l. Interference was considered to be important when the apparent change in the concentration of analyte exceeded the daily imprecision of the particular method for each analyser.

The results are summarised in the table. Each test was done in duplicate. It is clear from these results that at a given triglyceride concentration (3-4 mmol/l in this case) the interference for some tests can be different depending on whether the triglyceride is present in Intralipid or in lipaemic serum samples. For example, Intralipid showed no interference.

<table>
<thead>
<tr>
<th>Test</th>
<th>Intralipid</th>
<th>Lipaemic serum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>NI (0%)</td>
<td>NI (10%)</td>
</tr>
<tr>
<td>Urate</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Urea nitrogen</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>NI (0%)</td>
<td>NI (10%)</td>
</tr>
<tr>
<td>y-Glutamyltransferase</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Potassium</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Glucose</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Urate</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Creatinine</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>+15%</td>
<td>+15%</td>
</tr>
<tr>
<td>Phenobarbital</td>
<td>+15%</td>
<td>+15%</td>
</tr>
</tbody>
</table>

NI—no interference.
Diverticular disease of the right colon.

N Gubbay

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