prolactin concentrations during treatment, and trials are under way to test this hypothesis.

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# SHORT REPORTS

## Steroid cards: patient compliance

Long-term oral corticosteroid treatment is associated with secondary adrenocortical atrophy. For this reason, each patient on steroids is issued with a card which indicates the nature and dosage of treatment. We have investigated the extent of patient compliance in carrying notification of steroid drug use.

### Patients, methods, and results

One hundred patients, receiving corticosteroid drugs for rheumatoid arthritis, were interviewed. Each patient was asked (1) Do you have a *steroid* card? (2) Were you ever issued with a card? (3) Can you produce your card? (4) Do you know why you should always carry your card? The answers to the first three questions were scored on a "Yes" or "No" basis. Evaluation of a correct or incorrect answer to question (4) was made by the interviewer.

Eighty-seven patients said that they had cards and 88 agreed that a card had been issued. Seventy-six could produce their cards, while 77 knew why they should be carried. The reasons for the discrepancies in the figures are shown in the table. In 43 cases the dose of drug currently being taken was compared with the dose on the card. Agreement was found on 29 occasions (67%) and discordance in 14 instances (33%). The mean discrepancy was 2·46 mg prednisolone equivlaent (standard deviation 1·42 mg).

*Breakdown of patients' replies to questioning*

| Do you know why you should always carry your card? | | | | | | | |
|---|---|---|---|---|---|---|---|
| Yes (n = 77) | | | | No (n = 23) | | | |
| Card produced | Card not produced | | | Card produced | Card not produced | | |
| | Never issued | At home | Lost | | Never issued | At home | Lost |
| 65 | 4 | 8 | 0 | 11 | 8 | 3 | 1 |

### Discussion

In this survey almost a quarter of patients could not produce notification that they were taking steroid treatment. They must therefore be considered to be at risk of receiving inadequate treatment in an emergency. Half of them claimed that a card had never been issued. It was impossible to verify this. With one exception, the remainder claimed that the cards were at home, although most of them were aware of the necessity to carry the card at all times. Some four subjects who had never received a card appeared to know why they should have one. Overall, only 65 patients could both show a card and explain why it should be carried. The discrepancy between recorded and actual doses in one-third of cases is of more academic than practical importance, since emergency "steroid cover" is an all-or-none phenomenon unrelated to daily dosage. Nevertheless, along with the failure of the patients to produce a card, for whatever reason, it points to a lack of close supervision of potentially hazardous treatment.

During the survey cards were issued to all subjects who were unable to produce one; dosage was updated when necessary; and a full explanation given to each patient about the potential hazards of steroid treatment.

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## Simple method for diagnosing protein-losing enteropathies

Excessive loss of plasma proteins into the gastrointestinal tract occurs in several disorders. Methods currently available for detecting this loss are cumbersome, imprecise, and expensive and entail administering radioactive isotopes and collecting faeces over several days.[1]

$\alpha_1$-Antitrypsin, a glycoprotein present in normal serum at a concentration of 1·9-5·0 g/l, has been measured in meconium from healthy infants and from those with cystic fibrosis.[2] This protease inhibitor itself appears to be resistant to degradation by gut proteases.

We present evidence that the measurement of $\alpha_1$-antitrypsin in a random faecal sample may be used to diagnose protein-losing enteropathy.

($\alpha_1$-*Antitrypsin* ($\alpha_1$-*AT*) *concentrations in faecal and serum samples from normal subjects and cases A and B*

| Subject/case | Age (years) and sex | Diagnosis | Types of samples | Faecal $\alpha_1$-AT (mg/g dry weight) | Serum $\alpha_1$-AT (g/l) | Ratio of faecal:serum $\alpha_1$-AT |
|---|---|---|---|---|---|---|
| | | | *Paediatric controls* | | | |
| 1 | 2½ F | Cerebral atrophy | Three-day collection Day 1 | 2·1 | 3·3 | 0·64 |
| | | | Three-day collection „ 2 | 1·0 | 3·3 | 0·30 |
| | | | Three-day collection „ 3 | 1·35 | 3·3 | 0·41 |
| | | | Random sample | 1·35 | 3·3 | 0·41 |
| 2 | 3 M | Cerebral atrophy | Three-day collection Day 1 | 1·2 | 3·3 | 0·36 |
| | | | Three-day collection „ 2 | 0·7 | 3·5 | 0·20 |
| | | | Three-day collection „ 3 | — | — | — |
| | | | Random sample | 1·2 | 3·3 | 0·36 |
| 3 | 10/12 M | Klippel-Feil syndrome | Three-day collection Day 1 | 1·8 | 3·8 | 0·47 |
| | | | | 2·5 | 3·8 | 0·66 |
| | | | | 1·0 | | |
| | | | Three-day collection „ 2 | 1·6 | | |
| | | | | 1·7 | | |
| | | | Three-day collection „ 3 | 1·0 | | |
| | | | Random sample | 1·8 | 3·8 | 0·47 |
| 4 | 13 M | Rheumatic fever | Random sample | 0 | 5·65 | 0 |
| 5 | 13 M | Rheumatic fever | „ „ | 0·6 | 3·2 | 0·19 |
| 6 | 10/12 M | Respiratory infection | „ „ | 0·3 | 4·45 | 0·07 |
| 7 | 13 M | Healed Stevens-Johnson syndrome | „ „ | 1·4 | 3·5 | 0·40 |
| 8 | 13 M | Diabetes mellitus | „ „ | 0·6 | 2·8 | 0·21 |
| 9 | 10/12 M | Respiratory infection | „ „ | 1·0 | 2·5 | 0·40 |
| 10 | 12 M | Carditis | „ „ | 0·8 | 3·1 | 0·26 |
| | | | Mean values ± SD in random samples | 0·90 ± 0·52 | | 0·28 ± 0·15 |
| | | | *Adult controls* | | | |
| 11 | 26 F | Healthy | Random sample | 0 | 2·0 | 0 |
| 12 | 28 M | „ | „ „ | 0·7 | 2·25 | 0·31 |
| 13 | 27 F | „ | „ „ | 0 | 2·75 | 0 |
| 14 | 33 M | „ | „ „ | 0·3 | 1·65 | 0·18 |
| 15 | 22 M | „ | „ „ | 0·7 | 1·2 | 0·58 |
| 16 | 30 M | „ | „ „ | 1·2 | 1·9 | 0·63 |
| 17 | 22 F | „ | „ „ | 0·7 | 2·5 | 0·28 |
| 18 | 27 F | „ | „ „ | 0·6 | 2·15 | 0·28 |
| 19 | 29 M | „ | „ „ | 0·8 | 2·4 | 0·33 |
| 20 | 33 F | „ | „ „ | 0·6 | 2·8 | 0·21 |
| | | | Mean values ± SD | 0·56 ± 0·35 | | 0·28 ± 0·20 |
| | | | *Patients* | | | |
| A | 3/ F | Protein-losing enteropathy | Three-day collection Day 1 | 19·0 | 3·65 | 5·2 |
| | | | Three-day collection „ 2 | 15·6 | 4·0 | 3·9 |
| | | | Three-day collection „ 2 | 9·3 | 4·0 | 2·3 |
| | | | Three-day collection „ 3 | 7·3 | 4·0 | 1·8 |
| | | | Three-day collection „ 3 | 13·6 | 4·0 | 3·4 |
| | | | 24-hour sample | 8·7 | 2·7 | 3·2 |
| | | | Random sample 2/12 later | 14·9 | 4·15 | 3·6 |
| B | 2½/ M | Protein-losing enteropathy | Average of three-day collection | 8·8 | 3·1 | 2·8 |
| | | | Random sample 3/52 later | 13·6 | 3·3 | 4·1 |
| | | | Random sample 5 days later | 8·6 | 2·3 | 3·7 |

## Subjects, methods, and results

Ten children admitted to hospital for conditions other than gastrointestinal disease and 10 adults (laboratory staff) served as controls. Two children with protein-losing enteropathy were studied.

*Case A*—A 3-year-old girl had presented at 7 months with hypoproteinaemic oedema and facial lymphoedema. Jejunal biopsy showed lymphangiectasia, which on contrast studies appeared to be widespread. Despite frequent protein infusions and a low-fat diet severe oedema persisted.

*Case B*—A 2½-year-old boy had presented at 14 months with hypoproteinaemic oedema and severe iron deficiency. His diet was normal. Isotopic studies showed excess red-cell and protein loss in his faeces. A total of 14·3% of an intravenous injection of $^{51}Cr$ chromic chloride was recovered from his faeces (normal for an adult < 0·7%). Further investigation failed to disclose the cause of his protein-losing enteropathy and he continued to have bouts of mild oedema associated with hypoproteinaemia.

Faecal samples were frozen immediately after collection, lyophilised, then ground with a mortar and pestle. A 250-mg aliquot was extracted at room temperature with 5 ml 0·9% saline by intermittent mixing with a vortex apparatus over 30 minutes, then centrifuged at 12 000 g for 15 minutes (4°C). Samples (5 µl) of the supernatant were loaded into the wells of commercially available immunodiffusion plates. Serum of known $\alpha_1$-antitrypsin concentration was used as standard.

Blood samples were collected by finger-prick. When three-day faecal collections were required blood was sampled at the beginning of the collection and at 24-hour intervals (three samples). When random faecal samples were collected blood was sampled at the same time.

Separate collection and processing of samples over three days indicated that random sampling was satisfactory. For each child the faecal concentration of $\alpha_1$-antitrypsin varied within about a twofold range, and all values obtained for the children with protein-losing enteropathy were about tenfold higher than normal (see table).

We measured serum concentrations of $\alpha_1$-antitrypsin, as high concentrations might be responsible for excess of the protein in stools. This does not, however, appear to be a factor of much importance, and for our two patients the ratio of stool to serum $\alpha_1$-antitrypsin provided no discriminatory advantage over the faecal concentration alone (see table).

## Comment

The measurement of $\alpha_1$-antitrypsin in a random faecal sample provides a simple and reliable index of excessive loss of plasma proteins into the gastrointestinal tract. The test may be readily performed in a routine clinical laboratory with minimal participation of nursing and laboratory staff and no inconvenience to the patient.

If fresh samples are used, without lyophilisation, the faecal concentration of $\alpha_1$-antitrypsin may be classified as normal or raised within six hours. To eliminate the possibility that excessive water content in some faecal samples could give falsely low $\alpha_1$-antitrypsin values, however, we preferred to use freeze-dried samples.

The measurement of faecal $\alpha_1$-antitrypsin is useful for screening cases of suspected protein-losing enteropathy. Monitoring the response of such patient to therapeutic manipulations is a possible additional use of the test.

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