Gamma-glutamyltransferase in ascitic fluid in patients with various liver diseases

Diagnosis	No of patients	Enzyme activity (U/l)	
		Mean ± SD	Range
Hepatocellular carcinoma	5	24·2 ± 16·3	9–50
Alcoholic cirrhosis	22	58·1 ± 83·4	5·8–375
Miscellaneous	6	40·0 ± 58·5	4–156

the high activity in ascitic fluid from these patients is not surprising. In fact, the activity in seven of the patients with alcoholic liver disease was higher than the highest activity in the hepatoma group, the highest being in one patient with portacaval shunt established one month previously. Nevertheless, our different findings do not seem to be accounted for solely by our alcoholic patients, since both studies comprised patients with inactive cirrhosis, chronic active hepatitis, Budd-Chiari syndrome, and metastatic liver disease.

Comment

In our experience, therefore, measurement of γ-GT activity in ascitic fluid cannot be used to detect hepatocellular carcinoma or to differentiate between different liver diseases.

[1] Peters, T J, *et al*, *British Medical Journal*, 1977, **1**, 1576.
[2] Scandinavian Committee on Enzymes, *Scandinavian Journal of Laboratory Investigation*, 1976, **36**, 119.

(*Accepted 13 July 1979*)

Departments of Medicine and Clinical Chemistry, Sahlgren's Hospital, S-413 45 Gothenburg, Sweden
ROLF OLSSON, MD, lecturer in medicine
JOHAN WALDENSTRÖM, MD, lecturer in clinical chemistry

Trauma and severe proliferative retinopathy in diabetes mellitus

We describe two young men who developed severe proliferative retinopathy within six months of the diagnosis of diabetes mellitus. Both patients had suffered major trauma before this diagnosis, and we speculate that this may have precipitated the development of their retinopathy.

Case histories

Case 1—A 21-year-old restaurant manager was in a car accident in October 1972 and sustained multiple fractures. After admission to hospital he remained drowsy, unwell, and dehydrated. Diabetic ketoacidosis was diagnosed, and he was treated appropriately. He had noted some intermittent blurring of vision during the previous few months, but no retinopathy had been seen on examination. In February 1973 his eyes were re-examined because of severe blurring of vision. Proliferative retinopathy was found and he was referred to the diabetic retinopathy clinic at Hammersmith Hospital. Visual acuity was 6/5 on the right and 6/6 on the left. Examination showed that he had bilateral florid diabetic retinopathy. He was advised to have pituitary ablation, but this resulted in only slight diminution of pituitary gland function and there was no real improvement in the retinopathy; hence further yttrium-90 implantation of the pituitary gland was carried out giving a total dose of 600 000 rads. The retinopathy improved considerably but he retained some new vessels, and photocoagulation was therefore performed. Visual acuity remained normal.

Case 2—A 34-year-old lorry driver sustained a fractured pelvis, tibia, and fibula in a road traffic accident in September 1975. While in hospital he complained of intermittent blurring of vision. After discharge this became more severe and severe proliferative retinopathy was noted in both eyes. An oral 50-gn glucose tolerance test was performed. The blood concentrations (mmol/l) were as follows: fasting 7·6; 30 min 9·0; 60 min 11·1; 90 min 11·0; and 120 min 8·8. Diabetes mellitus was diagnosed. He developed a large vitreous haemorrhage in the left eye and was referred to the diabetic retinopathy clinic at Hammersmith Hospital. His visual acuity was 6/9 in both eyes; ophthalmoscopy showed severe proliferative retinopathy of both discs and retinal periphery, while in the left eye there was also a large preretinal haemorrhage. Because of the severity of the retinopathy the right eye was treated immediately with the xenon arc photocoagulator. Further treatment was given three and seven weeks later and in all 1696 xenon arc burns resulted in regression of the new vessels in this eye and maintenance of visual acuity at 6/9. A massive vitreous haemorrhage developed one day after the right eye was treated and has not cleared since. The visual acuity has remained around 3/60 to counting fingers since.

Comment

The striking feature of these two young men was that they both developed severe diabetic retinopathy shortly after the appearance of diabetes and associated with severe trauma. Severe proliferative and florid retinopathy is rare after so short a duration of diabetes, particularly at this age,[1 2] being usually seen in diabetes of long-standing and poor control.[3] In non-diabetics retinopathy is well described after severe trauma, particularly multiple fractures.[4] The question posed by these two cases is whether the retinopathy resulted from diabetes, trauma, or both. Many factors, both genetic and acquired, are probably concerned in diabetic retinopathy, one suggested factor being a disturbance of blood viscosity, which is also affected by severe trauma.[5] We would speculate, therefore, that trauma, possibly by its effect on blood viscosity, may have been the precipitating factor in producing severe proliferative retinopathy in the susceptible fundi of these two young, newly diagnosed diabetic patients.

We thank Dr T D R Hockaday, Dr T M Hayes, and Dr J O Williams for permission to report these cases and for their helpful comments.

[1] Soler, N G, *et al*, *British Medical Journal*, 1969, **3**, 567.
[2] Steel, J M, Skenfield, G M, and Duncan, L F P, *British Medical Journal*, 1976, **2**, 852.
[3] Colwell, J A, *Diabetes*, 1966, **15**, 497.
[4] Purtsher, O, *Albrech v. Gracfes Archiv für ophthalomolgie*, 1912, **82**, 347.
[5] Dintenfass, L, *Rheology of Blood in Diagnostic and Preventive Medicine*. London, Butterworth, 1976.

(*Accepted 25 July 1979*)

Queen Mary's Hospital, Sidcup, Kent DA14 6LT
WILLIAM D ALEXANDER, MB, MRCP, consultant physician

Hammersmith Hospital, London
MARGARET KEARNS, FRACS, research fellow, hon senior registrar
EVA M KOHNER, MD, FRCP, consultant physician, hon senior lecturer

Radcliffe Infirmary, Oxford
C M ASPLIN, MB, MRCP, senior registrar

Normal ileostomy output: close relation to body size

What determines the normal ileostomy output for an individual is unknown, but we have suspected for some time that it is related to the size of the patient.[1] Such a finding would be of importance to clinicians who manage patients with ileostomies but would also have important implications in gastrointestinal physiology. For this reason we studied the relation between various anthropomorphic measurements and the output of ileostomy fluid from a group of healthy patients with well-functioning ileostomies.

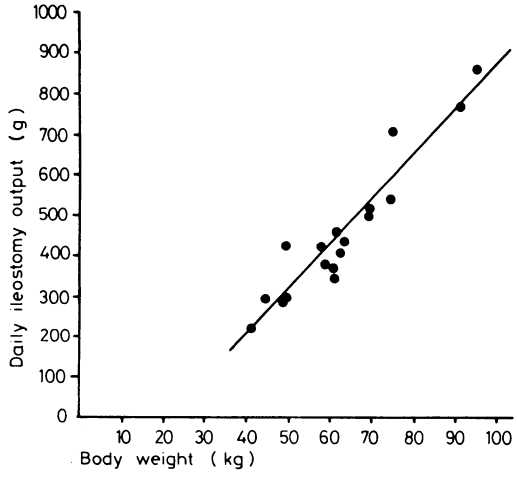
Patients, methods, and results

We studied 18 healthy patients (10 women, 8 men) aged from 27 to 71 years (mean 50·4 years). Each had had total proctocolectomy for histologically proved ulcerative colitis at least one year previously. None had had an ileal resection greater than 10 cm. The patients were studied out of hospital during their usual daily routine and none were taking any medication. They were asked to collect five consecutive full 24-hour samples of ileostomy fluid, each in a separate polyethylene container supplied to them; to eat their normal diet with the exception of cooked cabbage and prune juice[2]; and not to disturb their usual routine. Containers were collected each day from their homes. The weight of each daily sample was measured on arrival in the laboratory and the daily ileostomy volume was expressed as the mean measurement for the five consecutive daily collections. The physical characteristics and chemistry of the ileostomy fluid were also measured in each sample. In all collections they were within the published normal range.[1] At the end of the study the height and weight of the patients were measured

and the thickness of the mid-biceps, mid-triceps, and subscapular skinfolds were measured three times each with a Holtain skinfold calliper. Body density was calculated from the mean of the three measurements at each site by the method described by Durnin and Womersley.[3] Body fat was subsequently derived by Siri's equation,[4] and the fat-free mass calculated by subtracting the weight of body fat from body weight. Total body nitrogen was measured by in-vivo neutron activation using a sealed tube neutron generator producing 14 MeV neutrons.[5]

The mean weight of ileostomy discharge for the whole group was 459 ± 172 g/day (range 222 ± 60 g/day to 861 ± 198 g/day). Each patient had small daily variations in the weight of discharge (mean coefficient of variation 18·9%). The relation between the mean of the five daily collections of ileostomy fluid and body weight is shown in the figure. The regression equations, together with the standard error of the estimate, for the prediction of mean daily ileostomy output (IO) and the various anthropomorphic measurements (that is, height (Ht), body weight (BW), fat-free mass (FFM), and total body nitrogen (TBN)) are:

IO (g) = 10·8 Ht (cm) − 1280 ± 116 r = 0·75
IO (g) = 11·1 BW (kg) − 240 ± 57 r = 0·95
IO (g) = 12·9 FFM (kg) − 135 ± 97 r = 0·84
IO (g) = 0·42 TBN (g) − 171 ± 78 r = 0·90



Relation between daily ileostomy output and body weight.

Comment

The results show a close relation between the size of a patient and the ileostomy output. The relation is closest for body weight and the lean body mass (fat-free mass and total body nitrogen), but there is also a relation between height and the daily output of ileostomy fluid. According to these equations a so-called normal patient of 70 kg should have an average daily output from his ileostomy over five days of around 540 g (95% confidence limits are ±114 g).

These results put a new light on past and present studies of ileostomy function in health and disease. Probably they will also have important consequences for our understanding of gastrointestinal physiology.

We thank Professor R E Ellis and Drs L Burkinshaw and C B Oxby for the total body nitrogen measurements.

[1] Hill, G L, *Ileostomy—Surgery, Physiology and Management*. New York, Grune and Stratton, 1976.
[2] Kramer, P, Kearney, M M, and Inglefinger, F G, *Gastroenterology*, 1962, **42**, 535.
[3] Durnin, J V G A, and Womersley, J, *British Journal of Nutrition*, 1974, **32**, 77.
[4] Siri, W E, *University of California Radiation Laboratory Publication No 3349*, 1956.
[5] Oxby, C B, *et al*, *International Journal of Applied Radiation and Isotopes*, 1978, **29**, 205.

(*Accepted 31 July 1979*)

University Department of Surgery, The General Infirmary, Leeds LS1 3EX
G L HILL, FRCS, reader in surgery
S F MILLWARD, MB, house surgeon
R F G J KING, PHD, biochemist
R C SMITH, FRACS, research fellow

Neurological manifestations and mycoplasma pneumoniae infection

We report three patients presenting with acute neurological symptoms associated with mycoplasma pneumoniae infection.

Case reports

Case 1—A pharmacist aged 31 developed an influenza-like illness with a cough productive of yellow sputum seven days before hospital admission. He did not respond to treatment with trimethoprim and sulphamethoxazole, and three days later noted paraesthesiae and progressive weakness of both legs. On admission his temperature was 37·5°C and he had an obvious spastic paraparesis, extensor plantar responses, sensory level at D4, and retention of urine. Haemoglobin was 14·3 g/dl, white cell count 19·5 × 10^9/l (polymorphs 84%), erythrocyte sedimentation rate (ESR) 12 mm in 1 hour. A chest radiograph showed consolidation of the right upper lobe. A myelogram was normal. Cerebrospinal (CSF) contained 25 polymorphs and 12 lymphocytes/μl and protein 4 g/l; no organisms were seen or cultured. Serum mycoplasma antibody titres were raised (see table). He was treated with tetracycline and physiotherapy. His paraparesis improved gradually and he recovered completely within eight months.

Case 2—A 41-year-old woman was admitted with a 24-hour history of increasing restlessness and confusion after a short influenza-like illness with sweating and dry cough. She was afebrile and could move all four limbs but was drowsy with bilateral extensor plantar responses. Blood pressure was 140/80 mm Hg and there were no other systemic signs. Haemoglobin was 14·5 g/dl, white cell count 16·5 × 10^9/l, ESR 25 mm in 1 hour. A chest radiograph was normal. A computerised tomography (CT) scan showed areas of reduced attenuation in the white matter of both cerebral hemispheres. A brain biopsy specimen showed normal appearances. CSF contained 2 lymphocytes/μl and no organisms were seen or cultured. Serum mycoplasma antibody titres were raised (see table). She improved after treatment with tetracycline, and after five months her only deficit was slight expressive dysphasia and right hemiparesis.

Mycoplasma antibody titres in the three patients (measured by complement fixation test)

Case No	Neurological complications	On admission	One month later	Four months later
1	Transverse myelitis	4096	2048	256
2	Encephalitis	1080	640	80
3	Cerebellar syndrome	2048	512	256

Case 3—A 46-year-old man developed an influenza-like illness with headaches and myalgia. Two weeks later he became dysarthric and unsteady on walking with a tendency to fall to his right. On admission he was afebrile and fully conscious but dysarthric and had inco-ordination of both arms and legs with definite ataxia of gait. There was no meningism and examination was otherwise normal. Haemoglobin was 16·9 g/dl, white cell count 7·7 × 10^9/l, ESR 15 mm in 1 hour. Chest radiograph and CT scan were both normal. CSF protein concentration was 0·85 g/l and there was no pleocytosis. Serum mycoplasma antibody titres were raised (see table). No specific treatment was given and his cerebellar signs improved gradually. Six months after the onset he had only slight residual ataxia.

Comment

These three patients presented with acute neurological syndromes, the first affecting the spinal cord, the second the cerebral hemispheres, and the third the cerebellum. Although each had an initial influenza-like illness, there were no other specific clinical features to indicate a primary mycoplasma infection.

The neurological complications of mycoplasma pneumoniae include meningoencephalitis, cerebellar syndromes, cranial and spinal polyradiculoneuritis, and transverse myelitis.[1 2] Few such cases have been reported in Britain[3 4] and only one with transverse myelitis.[5] Antecedent or concurrent chest symptoms are not invariable. Nevertheless, a causal relationship between mycoplasma pneumoniae and the neurological complications seems likely either with direct infection of the nervous system or possibly with a secondary immunological reaction. Although death and persistent neurological deficits have been reported, the prognosis is often favourable.

Our experience supports the view that tests for mycoplasma pneumoniae should be included in the investigation of patients