Comment

Although the small numbers make conclusive claims hazardous, these data do not substantiate a large increase in unprotected anal intercourse. One reason may be rises in oro-anal and anal-digital contact which impugn the validity of rectal gonorrhoea as a surrogate marker for HIV risk behaviour.

Almost a fifth of men who tested antibody positive in this study were not clinic attenders. Just under a sixth of positive men became infected after the government campaigns began in 1986-7. Health promotion for gay and bisexual men remains a priority, and initiatives which concentrate on HIV transmission within relationships should be encouraged.

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Relation of serum sialic acid to lipid concentrations

Ichiro Wakabayashi, Kunihiro Sakamoto, Sachiko Yoshimoto, Hidehisa Masui

Department of Hygiene, Hyogo College of Medicine, Hyogo 663, Japan
Ichiro Wakabayashi, *assistant professor*
Kunihiro Sakamoto, *professor*
Sachiko Yoshimoto, *research officer*
Hidehisa Masui, *research officer*

Correspondence to:
Dr Wakabayashi, Department of Hygiene, Hyogo College of Medicine, 1-1 Mukogawa-cho, Nishinomiya, Hyogo 663, Japan.

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Sialic acid, a constituent of plasma membrane, is present in most acute phase reactant proteins. A recent epidemiological study showed that mortality from cardiovascular diseases was higher in a population with high concentrations of serum sialic acid.[1] The increase is suspected to reflect the existence or activity of the atherosclerosis process. We investigated the relation of serum sialic acid to serum lipids, which strongly influence the occurrence and advance of atherosclerosis.

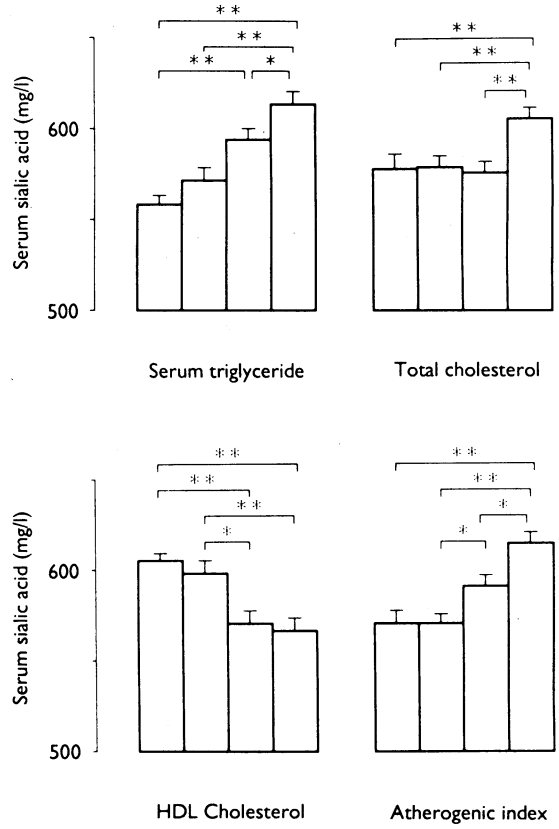
Subjects, methods, and results

Subjects were 382 men (35-54 years old) participating in a periodic medical health examination at their workplace; the response rate was 98%. Blood was sampled between 9 am and noon after subjects had fasted overnight. Serum lipids (total cholesterol, high density lipoprotein (HDL) cholesterol, and triglyceride) were analysed on an automatic analyser. Serum sialic acid was measured by the enzymatic method.[2] An atherogenic index was calculated as:

$$\text{atherogenic index} = \frac{(\text{total cholesterol} - \text{HDL cholesterol})}{\text{HDL cholesterol}}$$

Data are expressed as means±(SE). Student's *t* test was used to compare group means. The values for each serum lipid variable were arranged in ascending order and then the subjects were divided into four groups of approximately equal size. As serum sialic acid concentration is known to increase with age,[1] multiple regression analysis was performed with serum sialic acid as a target coefficient and the four items of age, serum triglyceride, total cholesterol and HDL cholesterol as explanation coefficients. As the atherogenic index is strongly affected by serum total and HDL cholesterol values, it was not included as an explanation coefficient of the multiple regression analysis. p Values $<0{\cdot}05$ were defined as significant.

Mean concentrations of serum sialic acid were compared among the fourths of each serum lipid variable (triglyceride, total cholesterol, HDL cholesterol, and atherogenic index). Mean serum sialic acid concentrations in the highest fourth of serum triglyceride, total cholesterol, and atherogenic index were significantly higher than in the other three divisions, and sialic acid concentrations were higher in each fourth of serum triglyceride and atherogenic index. For HDL cholesterol, however, sialic acid concentrations of the upper two fourths of HDL cholesterol were significantly lower than those of the lower two fourths, and the mean values of serum sialic acid were lower in each higher quartile of HDL cholesterol (figure). Standardised multiple regression coefficients between serum sialic acid and age, HDL cholesterol, triglyceride, and total cholesterol were 0·100 ($p<0{\cdot}05$), −0·146 ($p<0{\cdot}01$), 0·137 ($p<0{\cdot}05$), and 0·030 ($p>0{\cdot}05$), respectively.



*Serum sialic acid concentrations related to serum lipid variables, in fourths. Bars indicate SE; *=p<0·05, **=p<0·01*

Comment

A slight but significant positive correlation between serum total cholesterol and sialic acid in a large population and a strong positive correlation between serum triglyceride and sialic acid in patients with diabetes mellitus have been reported.[1 3] In our study, serum sialic acid concentration was significantly higher

in groups with high concentrations of triglyceride or total cholesterol and significantly lower in a group with high concentrations of HDL cholesterol. Serum triglyceride showed a positive significant correlation with serum sialic acid and HDL cholesterol showed a negative significant correlation. These findings suggest that the serum concentration of sialic acid is affected by serum lipids. Increases in serum sialic acid concentration in patients with diabetic angiopathies and in smokers have been shown.[4,5] Thus, the risk factors in atherosclerotic cardiovascular diseases, such as smoking, diabetes mellitus, and hyperlipidaemia, are related to serum sialic acid concentration. This may explain why serum sialic acid concentration was raised in a population with a high mortality from cardiovascular diseases.

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Treatment with activated charcoal complicated by gastrointestinal obstruction requiring surgery

S W Atkinson, Y Young, G A Trotter

Lewisham Hospital, London SE13
S W Atkinson, *senior house officer in general surgery*
Y Young, *registrar in anaesthetics*
G A Trotter, *senior registrar in general surgery*

Correspondence to:
G A Trotter, Greenwich District Hospital, Greenwich, London SE10 9HE.

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Activated charcoal has long been a mainstay in preventing the further absorption of many ingested drugs.[1] It also enhances elimination of some drugs after they have been absorbed, particularly aspirin, carbamazepine, digoxin, barbiturates, and phenytoin.[2] At the current recommended adult dose (50 mg every four hours or 25 mg every two hours)[3] adverse effects of activated charcoal are rare and mostly concern the risk of pulmonary aspiration in patients unable to protect their airway, particularly if the charcoal is given after ipecacuanha.[4] We report a case of bowel obstruction with charcoal given for barbiturate overdose.

Case report

An unconscious 24 year old man was admitted to the accident and emergency department having taken a large quantity of barbiturates and benzodiazepines, including Tuinal (amylobarbitone sodium 50 mg, quinalbarbitone sodium 50 mg), temazepam, and diazepam. He had fallen asleep outdoors overnight and when discovered was unrousable. His core body temperature was 26·4°C, pulse 48/min and in sinus rhythm, and blood pressure 90/50 mm Hg. Laryngoscopy showed appreciable amounts of gastric contents in his larynx and trachea. He was intubated, ventilated, and transferred to intensive care. Arterial blood gas analysis disclosed hypoxaemia, hypercapnia, and acidosis. A chest *x* ray film showed bilateral diffuse pulmonary shadowing. Inotropes were begun and he was rewarmed. On advice from the regional poisons unit 25 g activated charcoal (Medicoal) was instilled every four hours down the nasogastric tube with 200 ml water. This was to be continued until the charcoal appeared in the stools. After 18 hours no charcoal was passed per rectum and administration was stopped.

Toxicology studies showed serum concentrations of 6·5 mg/l of both amylobarbitone and quinalbarbitone. After 48 hours he still required cardiorespiratory support. He remained hypoxaemic and repeat chest *x* ray examination confirmed the adult respiratory distress syndrome.

The patient's clinical condition remained unchanged for a further 48 hours until the volume of nasogastric aspirate increased, the abdomen distended, and he became feverish (38·5°C). The abdomen was soft (no muscle relaxants were being given) but borborygmi were increased and obstructive in character. Radiography confirmed small bowel obstruction without evidence of perforation. Abdominal ultrasonography showed free intraperitoneal fluid, which on paracentesis was bile stained. The patient proceeded to laparotomy and was found to have small bowel obstruction due to a large bolus of inspissated charcoal in the caecum with associated areas of full thickness ischaemia but no overt perforation. Culture of the intraperitoneal fluid grew Gram negative organisms.

The charcoal bolus could not be fragmented, so limited right hemicolectomy was undertaken and intestinal continuity restored. Bowel sounds returned after three days. He made a gradual recovery from his pulmonary insult and after withdrawal of cardiovascular support he was extubated. He was discharged from hospital seven weeks after admission.

Comment

Watson *et al* described gastrointestinal obstruction associated with activated charcoal treated conservatively with nasogastric magnesium citrate and saline enemas.[5] Our patient was in danger of caecal stercoral perforation caused by the charcoal "briquette" and would have been unlikely to respond to further conservative measures. Any severely ill patient, from whatever cause, is likely to have decreased intestinal motility. Both hypothermia and aspiration pneumonitis may be implicated in the development of paralytic ileus. Additionally, many drugs whose elimination is enhanced by activated charcoal also cause reduced gut motility.

In our patient the combination of these factors and treatment with activated charcoal resulted in the formation of a solid charcoal bolus in the caecum with life threatening consequences. We recommend that administration of activated charcoal in such patients should be given with an osmotically active aperient and that administration should be discontinued if significant nasogastric aspirates of charcoal are returned or if the charcoal does not appear in the stools within 12 hours.

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