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

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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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