carditis is difficult. Ideally, active infection should be eradicated first; the tissues hold sutures better and the hazard of infecting the valve replacement is decreased. Uncontrollable heart failure may require urgent surgery before completion of a full antibiotic course; however, early mortality rates approach 30%.⁴

We wish to draw attention to the possible risk of I.U.D. insertion causing endocarditis, which in our case resulted in the death of a young, previously asymptomatic patient. Endocarditis still has a mortality of some 30%.⁵ We therefore suggest that I.U.D. insertion in patients at risk from endocarditis should be covered by antibiotics. Because of the Gram-negative organisms in the vagina a broad-spectrum antibiotic such as ampicillin should be used.

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Discussion

Serum transaminases rose in 10 out of 14 patients receiving intravenous heparin for their underlying disease. This rise was unrelated to dose or to the duration of therapy and in no patients were other diseases discovered that could explain this. It is not clear why this happens. Vavgorik³ noted the rise in other serum enzymes, such as aldolase, sorbitol dehydrogenase, and leucine aminopeptidase in patients undergoing chronic hemodialysis with heparinization. No change in the GOT and GPT levels before and after dialysis were noted, but the levels during treatment were not tested.

The clinical importance of the rise in serum enzymes during heparin therapy is obvious. Heparin therapy is frequently given for thromboembolic phenomena, and the diagnosis of pulmonary infarction, hepatic damage, and myocardial infarction in these patients is important. The determination of transaminase levels is an established aid in the differential diagnosis of these conditions. The cause of this enzyme rise is not evident, and further studies must be carried out to determine this.

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Hyper-transaminasemia with Heparin Therapy

Many side effects seen with heparin therapy have been related to the coagulation system. Biological effects on intercellular enzymes,¹ bone electrolytes, antidiuretic hormone, and aldosterone,² however, have been noted. Recently we have noted a rise of serum transaminase levels beginning during heparin treatment. Since transaminase determinations are important in the differential diagnosis of myocardial infarction, liver disease, and pulmonary emboli, rises that might be caused by drugs are very important.

Patients, Methods, and Results

Transaminase levels were recorded before, during, and after heparin therapy in 14 inpatients. All patients received 10 000 units of heparin intravenously every six hours for a period of 10 to 21 days. Blood samples for the enzyme determination were taken from 60 to 90 minutes after the morning injection of intravenous heparin. Serum levels of GOT and GPT were estimated by the SMA 12/60 method, (normal levels with this method are 20 to 40 units for GOT and 20 to 45 units for GPT.) In 10 out of 14 patients the serum transaminase levels rose during heparin treatment (see table). These were definitely abnormal after heparin therapy started with the GPT being higher than the GOT and both falling to normal when treatment ended. The possibility that heparin interfered with the SMA 12/60 determination of serum transaminases was excluded by performing the determination on paired blood samples from normal controls, to one of which heparin was added after blood was withdrawn. No difference was noted between these two samples.

Diagnosis and Maximum Enzyme Level

Age (Years)	Diagnosis	Maximal GOT Level	Maximal GPT Level	Day of Maximum Rise	Duration of Rise (Days)
29	Deep Thrombophlebitis	65	40	5	10
53	Cerebral Emboli	75	110	9	13
24	Deep Thrombophlebitis	70	155	9	13
32	Deep Thrombophlebitis	95	235	7	15
24	Deep Thrombophlebitis	70	120	9	9
33	Deep Thrombophlebitis and Pulmonary Emboli	90	120	12	21
50	Deep Thrombophlebitis	60	60	3	7
46	Pulmonary Emboli	70	135	7	15
33	Deep Thrombophlebitis	85	170	15	17
69	Pulmonary Emboli	70	100	3	13

Calcium Polystyrene Sulphonate: An Unusual Cause of Inhalation Pneumonia

Calcium and sodium polystyrene sulphonate are two potassium absorbing resins administered orally or by retention enema for treatment of hyperkalaemia. Histologically sodium polystyrene sulphonate has been seen on the surface of sections of gastric mucosa.¹ We report here an unusual case of pneumonia associated with the inhalation of the calcium resin.

Case Report

An elderly man was admitted to hospital with a brief history of severe chest pain and shortness of breath. Clinical and electrocardiographical examination showed left ventricular failure, pericarditis, and an acute myocardial infarction. The next day heart block occurred, and the patient underwent cardiac catheterization for maintenance by a pacemaker. Persistent hypotension was treated with intravenous isoprenaline. Oliguria failed to respond to large doses of frusemide and subsequent hyperkalaemia was treated with oral calcium polystyrene sulphonate (Calcium Resonium). Three days after admission the patient had a cardiac arrest and died.

At necropsy the heart showed a recent area of infarction associated with atherosclerotic occlusion and thrombosis of the right coronary artery. The entire pericardial surface was rough and reddened. The lungs, normally lobed with dark shiny anthracotic surfaces and dull grey cut surfaces, contained much free flowing grey fluid. The main bronchi and intrapulmonary bronchi had reddened congested mucosae. The remaining organs showed only congestive changes.

Photomicrograph of pneumonic alveoli containing basophilic calcium polystyrene sulphonate. (H. and E. $\times 183$.)

Sections of the lungs showed pulmonary oedema and patches of broncho-pneumonia. In the latter there were strikingly large numbers of strongly basophilic angular fragments ranging in size from 5-75 μm (see fig.). These were not present in the more normal areas of lung. Initial investigation showed that the particles were colourless, birefringent, autofluorescent, and positive for periodic-acid Schiff. Subsequently they were shown to be strongly positive with Schiff reagent without prior oxidation, with hexamine silver, and by the Ziehl-Neelsen method. They were orthochromatic, variably positive by Gram's method and with aldehyde fuchsin and Sudan black, and negative with alcian blue and by von Kossa's method.

Discussion

This case caused diagnostic difficulty owing to the extraordinary appearance of the material in the pneumonic alveoli. We reject the possibility that it was an extraneous artefact because of its coincident distribution with the areas of pneumonia.

Sections of normal lung artificially contaminated by sodium or calcium resins showed particles morphologically and tinctorially identical to those found in our patient's lungs. The direct reaction of Schiff's reagent with the ethylene linkages in the polystyrene is regarded as a virtually pathognomonic feature of sodium polystyrene sulphonate.¹ Thus the physical and histochemical characteristics leave little doubt as to the nature of the particles.

Inhalation pneumonia resulting from aspiration of food or gastric contents is not uncommon, and irritating gases and chemical substances are also well recognized causal agents. Though there is no record of this patient vomiting it seems most likely that the resin initiated the pneumonic process. We know of no other similar case of pneumonia due to inhalation of calcium polystyrene sulphonate, and in view of the bizarre histology we considered that this case should be reported.

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Ultrasonography and Possible Ruptured Abdominal Aortic Aneurysms

Four patients presented with possible ruptured abdominal aortic aneurysms. The diagnosis was confirmed in three and excluded in the fourth after ultrasonography. Ultrasonography is a quick and non-invasive technique which may clarify the diagnosis in an emergency. The patients were wheeled on a trolley from the admission room to a Kretz scanning device and examined with minimal disturbance in less than five minutes. All were emergency admissions.

Case Histories

A man of 63 had a six-day history of left hip pain and backache with pallor and a tender mass in the left loin which was obviously pulsatile. A renal lesion or a leaking aneurysm was suspected. Ultrasonography confirmed the latter, which was verified at operation.

A man of 84 presented with a few hours history of abdominal and back pain. He had hypotension and a pulsatile abdominal mass, and ultrasonography showed an aneurysm which had ruptured producing a haematoma in the left iliac fossa (see fig.). The patient died before we could operate and the ultrasonic findings were confirmed at necropsy.

A woman of 66 had had backache for several days and intermittent claudication. She was hypotensive and a tender left hypochondrial mass was palpable. Ultrasonography showed no evidence of an aneurysm. Laparotomy disclosed retroperitoneal bleeding due to the erosion of splenic vessels from a carcinoma of the transverse colon. The aorta was normal.

A woman of 61 presented with a two-day history of severe backache. She had shock, abdominal tenderness, and a small pulsatile mass in the hypogastrium. Ultrasonography showed a ruptured aneurysm, which was confirmed at operation.

Discussion

Rupture of an abdominal aortic aneurysm may be confused with almost any intra-abdominal condition presenting as an acute emergency.¹ The mortality of such a rupture is 100% if untreated and 50% when recognized and treated surgically.² This mortality rate becomes even higher if the diagnosis is delayed and the patient becomes hypotensive or anuric.³ An early diagnosis improves the chances of successful surgery. Though the correct diagnosis may often be made clinically there is still need for a reliable and quick investigation to confirm it. This may help in organizing theatre and staff for a vascular procedure.

Aortography in the unruptured aneurysm has well-known hazards including haemorrhage through the puncture in the aortic wall. We cannot recommend its use in an emergency. A plain x-ray film of the abdomen may help diagnosis when the wall of an aneurysm is calcified. Such calcification, however, is present in only about half the cases.⁴ If there is calcification, extension of a soft tissue mass beyond the calcified rim or displacement of bowel gas anteriorly suggests a rupture.⁵ Loss of the psoas shadow may also be found.