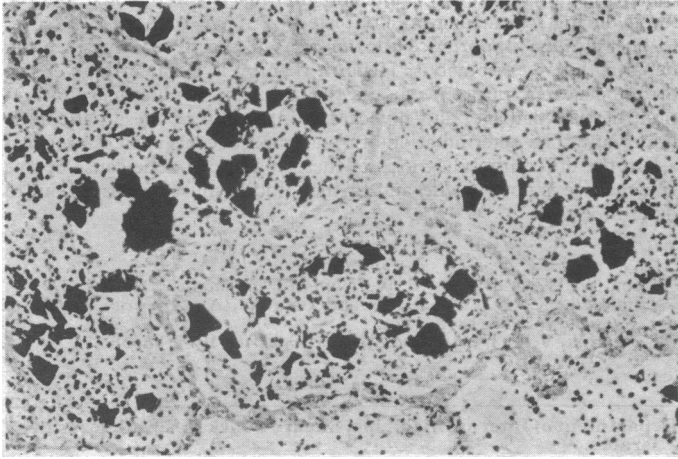


At necropsy the heart showed a recent area of infarction associated with atherosclerotic occlusion and thrombosis of the right coronary artery. The entire pericardial surface was rough and reddened. The lungs, normally lobed with dark shiny anthracotic surfaces and dull grey cut surfaces, contained much 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.

We thank Dr. M. S. Dunnill for his interest, Mr. T. Reed for the photomicrograph, and Mrs. R. Hunt for the typescript.

¹ Liber, A. F., *American Journal of Pathology*, 1974, 74, 106a.

Histology Department, Gibson Laboratories, Radcliffe Infirmary, Oxford OX2 6HE

A. J. CHAPLIN, F.I.M.L.T., Chief Technician

P. R. MILLARD, M.D., M.R.C.PATH., Lecturer in Pathology

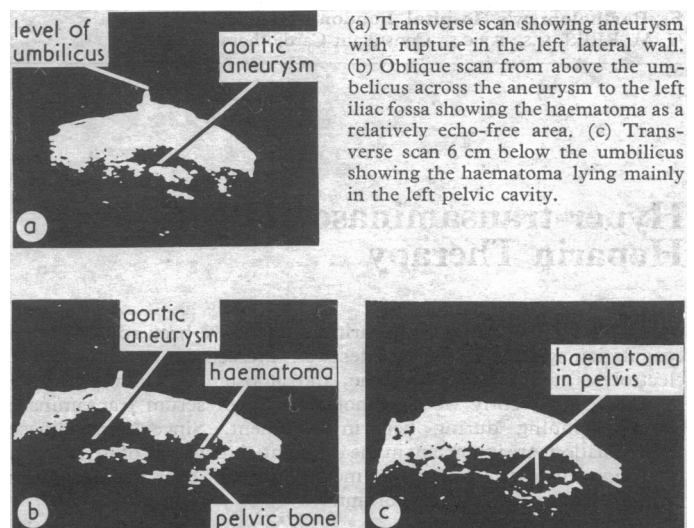
Ultrasonography and Possible Ruptured Abdominal Aortic Aneurysms

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) Transverse scan showing aneurysm with rupture in the left lateral wall. (b) Oblique scan from above the umbilicus across the aneurysm to the left iliac fossa showing the haematoma as a relatively echo-free area. (c) Transverse scan 6 cm below the umbilicus showing the haematoma lying mainly in the left pelvic cavity.

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.

Ultrasonography has obvious advantages in an emergency including safety, quickness, and lack of upset to the ill patient.

We thank the Department of Medical Illustration at Stobhill Hospital for the print. We also thank Dr. Agnes Macgregor for permission to include the first case. The typing was kindly done by Mrs. Nasmyth and her staff.

¹ Pryor, J. P., *British Medical Journal*, 1972, 3, 735.

² Alpert, J., Brief, D. K., and Parsonnet, V., *Journal of the American Medical Association*, 1970, 212, 1355.

³ Jamieson, C. W., *Journal of the Royal College of Surgeons of Edinburgh*, 1974, 19, 386.

⁴ McGregor, J. C., Pollock, J. G., and Anton, H. C., *Scottish Medical Journal*, 1975, 20, 133.

⁵ Janover, M. L., *New England Journal of Medicine*, 1961, 265, 12.

Stobhill General Hospital, Glasgow, G21 3UW

J. C. MCGREGOR, M.B., F.R.C.S., Surgical Registrar
J. G. POLLOCK, M.B., F.R.C.S., Consultant Surgeon
H. C. ANTON, M.B., F.F.R., Consultant Radiologist

Discussion

Delirium tremens and alcoholic cardiomyopathy were rejected in favour of a unitary diagnosis of beriberi (see table).² The dramatic response to two injections of parenterovite accorded with the diagnosis.⁴ Alcoholic cardiomyopathy was considered unlikely in a high output state with normal E.C.G.s. Alcoholism is a growing problem, and food is expensive. The risk of sudden cardiac failure in wet beriberi is so severe that the diagnosis should be considered. Together with a clinical examination a pyruvate test is a quick and safe indicator of this condition, and treatment is immediately effective.⁴

¹ *British Medical Journal*, 1974, 4, 731.

² Slater, E., and Roth, M., *Clinical Psychiatry*, 3rd edn., p. 354. London, Baillière, 1969.

³ Granville-Grossman, K., *Recent Advances in Clinical Psychiatry*, p. 117. London, Churchill, 1971.

⁴ Brain, W. R., *Diseases of the Nervous System*, 7th edn., p. 728. London, O.U.P., 1969.

Department of Psychiatry, University of Liverpool, Liverpool L69 3BX

JOYCE RIDING, M.R.C.PSYCH., Lecturer

Wet Beriberi in an Alcoholic

Alcoholism is now not usually associated with nutritional deficiency,¹ but when it occurs, inadequate vitamin B₁ in the diet may lead to heart failure. In this condition (wet beriberi) there is fluid retention and cardiac dilatation in a high output state. The following report is of one of two cases of wet beriberi seen in six months in a small catchment area.

Case Report

A retired bricklayer aged 65 was referred for psychiatric treatment after his second admission to a surgical ward with ascites and gross oedema of both legs. The third night after admission he became confused and aggressive and signed himself out, only to be readmitted within a week with an identical picture; on the second night he was again confused and aggressive.

He gave a history of severe chronic abuse of alcohol and a prolonged inadequate diet. Drinking had occurred up to and between admissions and periods of confusion had occurred at home over six months. He had had amnesias for years but never delirium tremens or fits. All other aspects of the history were regarded as insignificant. Crepitations were heard at both bases of the lungs, and nothing abnormal was found on extensive and sophisticated investigations except an increase in heart size with congestion of both lungs in the presence of repeatedly normal electrocardiograms (E.C.G.s). Psychiatric opinion was sought before laparotomy. The patient was very flushed with a dry hot skin and the blood pressure was 160/90 mm Hg. There was no tremor or anxiety. He seemed calm but fatuous and he confabulated, showed a total loss of secondary memory, and was disorientated for date but not for person and place. There was no thought disorder. He showed the mental symptoms combined with a high output cardiac failure described in the unitary diagnosis of wet beriberi (see table).²

Distinguishing Features of Delirium Tremens and Beriberi

Delirium Tremens	Beriberi
Pallor	Flushed skin
Cold skin	Hot and dry skin
Tremor	No tremor
Ataxia	No ataxia
Anxiety and terror	Irritability and aggression at night only
Hallucinations	Illusions
Disorientation	Disorientation
Memory defect	Memory defect
Response to treatment, 3-4 days	Response to treatment, immediate

Because of the danger of sudden cardiac failure all investigations were discontinued except for a pyruvate tolerance test,³ which gave grossly abnormal results. Parenterovite 10 ml was given intravenously for seven days and the patient was sedated with chlordiazepoxide 10 mg three times a day. Sleep was undisturbed that night and he showed no further confusion of thought or behavioural difficulties. Appetite improved and the oedema resolved in seven days. Psychological assessment confirmed a Korsakoff syndrome, which remained.

When his condition improved and blood pyruvate levels returned to normal he was discharged. But he resumed drinking and presented again with ascites and gross oedema. He was readmitted and responded to treatment. After pyruvate values returned to normal he was discharged but parenterovite was given twice a week by a community nurse. After one month he was well in spite of drinking.

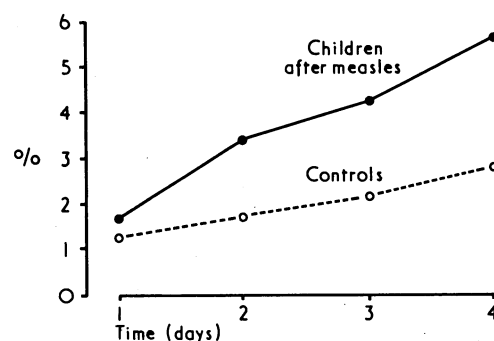
Measles: A Protein-losing Enteropathy

Undernourished children who suffer an attack of measles are often precipitated into overt kwashiorkor.¹ There are several possible explanations for this, perhaps the most important being that protein and calorie requirements are high during any infectious process accompanied by fever. Other contributing factors include the anorexia during the acute phase of measles and the tradition among certain African tribes of withholding animal protein from a child during and after measles.

Measles might also affect the state of nutrition through direct action on the intestinal mucosa. Morley² noted that many West African children had blood in their stools during the first few days after the onset of measles and reasoned that the intestinal mucosa was probably affected similarly to the skin. Other generalized skin diseases have been associated with both maldigestion and protein-losing enteropathy. The following study was undertaken to ascertain whether there is increased protein loss from the intestine during the acute phase of measles in malnourished children.

Patients and Methods

Ten malnourished children with measles were studied two to 14 days after the first appearance of a typical measles rash. Their mean age was 17.1 months, weight 7.06 kg, and serum albumin 31 g/l. Six children with moderate to severe kwashiorkor served as controls. Their mean age was 20.6 months, weight 8.67 kg, and serum albumin 14 g/l. An intravenous injection of 10-15 μ Ci of ⁵¹CrCl₃ solution was given to each child within two hours of



Mean accumulated stool radioactivity expressed as percentage of intravenous dose plotted against time.