

The pathogenesis of the pulmonary fibrosis is also unknown. It is tempting to suggest that it commences from the deposition of autoimmune complexes, but this patient had no evidence of the glomerular damage that is usually found if circulating immune complexes are present. Unfortunately, no immunological studies were carried out on the patient's lungs.

Acknowledgments

We are grateful to Dr Roger Williams for permission to publish details of this patient who was under his care, and Mr George Harwood for technical assistance.

References

- DINES, D.E., CLAGETT, O.T. & GOOD, C.A. (1967) Non-tuberculous pulmonary parenchymal conditions predisposing to spontaneous pneumothorax. *Journal of Thoracic and Cardiovascular Surgery*, **53**, 726.
- DUBOIS, E.L. (1966) *Lupus Erythematosus*, p. 172. McGraw-Hill Book Company Inc., New York Blakiston Division.
- HOFFBRAND, B.I. & BECK, E.R. (1965) 'Unexplained' dyspnoea and shrinking lungs in systemic lupus erythematosus. *British Medical Journal*, **1**, 1273.
- HOLDEN, M. (1973) Massive pulmonary fibrosis due to systemic lupus erythematosus. *New York State Journal of Medicine*, **73**, 462.
- HUANG, C.T. & LYONS, H.A. (1966) Comparison of pulmonary function in patients with systemic lupus erythematosus, scleroderma, and rheumatoid arthritis. *American Review of Respiratory Diseases*, **93**, 865.
- HUGHES, G.R.V. (1971) Significance of Anti-DNA antibodies in systemic lupus erythematosus. *Lancet*, **ii**, 861.
- LAITINEN, O., SALORINNE, Y. & POPPIUS, H. (1973) Respiratory function in systemic lupus erythematosus, scleroderma and rheumatoid arthritis. *Annals of the Rheumatic Diseases*, **32**, 531.
- MUNDY, G.R. & MACDONALD, C.B. (1972) Atypical presentations of diffuse fibrosing alveolitis. *British Journal of Diseases of the Chest*, **66**, 261.
- OLSEN, E.G.J. & LEVER, J.V. (1972) Pulmonary changes in systemic lupus erythematosus. *British Journal of Diseases of the Chest*, **66**, 71.
- PURNELL, D.C., BAGGENSTOSS, A.H. & OLSEN, A.M. (1955) Pulmonary lesions in systemic lupus erythematosus. *Annals of Internal Medicine*, **42**, 619.
- SAWKAR, L.A. & EASOM, H.F. (1971) Recurrent spontaneous pneumothoraces in systemic lupus erythematosus. *Chest*, **60**, 604.
- SCADDING, J.G. & HINSON, K.F.W. (1967) Diffuse fibrosing alveolitis (diffuse interstitial fibrosis of the lungs). Correlation of histology at biopsy with prognosis. *Thorax*, **22**, 291.

Addendum

The authors of a recent paper* reviewing diffuse interstitial lung disease in SLE could find no cause for the lung lesions in this condition. The participation of SLE in the disease processes in the lungs is unresolved.

* EISENBERG, H., DUBOIS, E.L., SHERWIN, R.P. & BALCHUN, O.J. (1973) Diffuse interstitial lung disease in systemic lupus erythematosus. *Annals of Internal Medicine*, **79**, 37.

Postgraduate Medical Journal (December 1975) **51**, 855-857.

Pancreatitis and the pill

I. P. F. MUNGALL
M.B., B.Chir., M.R.C.P.

R. V. HAGUE
M.B., Ch.B., M.R.C.P.

The Royal Hospital, West Street, Sheffield S1 3SR

Summary

An association between acute relapsing pancreatitis and the contraceptive pill has recently been suggested (Bank and Marks, 1970; Davidoff, Tishler and Rosoff, 1973). We wish to report a further case of acute pancreatitis in a patient taking the pill. In contrast to previous cases the serum lipids were normal and the pancreatitis was severe and ultimately fatal.

Case report

A thirty-four-year-old housewife and mother of eight had been on a combined contraceptive pill (ethinyloestradiol and mestrol acetate) since 1969.

In June 1973 she developed severe, recurrent attacks of upper abdominal pain and vomiting. She had previously been healthy and there was no history of alcoholism. Her general practitioner suspected gallstones and referred her for a surgical opinion in November, 1973. She was noted to be obese but there were no specific physical signs and she failed to attend for investigation.

In December 1973 she was admitted to the Casualty Department having suddenly collapsed with central and left-sided chest pain. She discharged herself before she could be assessed, only to be readmitted a few hours later with a recurrence of

the pain. On examination, she was severely ill with circulatory collapse, dyspnoea and gross cyanosis. A superficial thrombophlebitis was noted on the medial aspect of the right thigh. No specific abnormality was found in the chest, central nervous system or abdomen.

A portable chest X-ray was technically unsatisfactory but probably normal. An E.C.G. showed 'T' wave inversion over the left standard and chest leads. Arterial pO₂ was 18 mmHg and pCO₂ 66 mmHg. In spite of the high pCO₂ the overall clinical picture suggested a diagnosis of massive pulmonary embolism. As her condition was deteriorating rapidly, an immediate pulmonary angiogram was performed with a view to urokinase therapy. This, however, proved to be normal.

It was at this stage that we learnt of previous attacks of abdominal pain. A diagnosis of acute pancreatitis was therefore considered and confirmed by an elevated serum amylase at 1350 iu/l (normal 70–300 iu/l). The serum lipase was also elevated at 276 iu/l (normal 0–75 iu/l), whilst serum calcium was reduced at 7.6 mg/100 ml (normal 9–11 mg/100 ml), and blood sugar was reduced at 13 mg/100 ml.

Results of other investigation performed at this time were as follows: Haemoglobin 11.2 g/100 ml; W.C.C. 9000/mm³; plasma insulin, measured by radioimmunoassay, 38 m iu/l (normal 5–40 m iu/l); serum cholesterol 108 mg/100 ml (normal 150–300 mg/100 ml); serum triglyceride 38 mg/100 ml (normal 50–200 mg/100 ml).

The patient was treated with supportive therapy which included intravenous fluids, antibiotics, steroids and intermittent positive pressure respiration. On the following day she was found to have a thrombocytopenia with a platelet count of 45,000/mm³. Fibrinogen degradation products at 320 µg/ml (normal = < 20 µg/ml) and prothrombin time at 40 sec (control time 11 sec) confirmed disseminated intravascular consumption coagulopathy.

Her condition continued to deteriorate and after 7 days it became obvious that she would not recover. Ventilation was discontinued and she died shortly afterwards. At post-mortem there was evidence of extensive hepatic and pancreatic necrosis with associated calcification. There was no evidence of pancreatic arterial or venous thrombosis and the biliary tree was normal.

Discussion

Bank and Marks (1970) described two cases of acute pancreatitis in which there was a clear temporal relationship between relapses and taking the pill. Two similar cases were described by Davidoff *et al.* (1973). One of these, in common with the case

reported here, had an initial symptom-free period of a few years from starting to take the pill.

These four cases had a number of features in common. Gallstones were suspected in every case and three came to laparotomy. The pancreatitis was mild and resolved completely on stopping the pill. Hyperlipidaemia of a Fredrickson type V was a constant feature and this has been implicated as a possible cause of the pancreatitis. Glueck *et al.* (1972) also described acute pancreatitis in patients with a type V hyperlipidaemia on oestrogen therapy. Hyperlipidaemia however is well recognized in association with acute pancreatitis regardless of the cause (Cameron *et al.*, 1971, 1973) and some workers (Stackhouse, Glass and Zimmerman, 1966; Wang, Strauss and Addlesburg, 1958) have suggested that this may be due to the pancreatitis itself.

The present case differs from those previously reported in that the cholesterol and triglyceride levels were normal and the pancreatitis was severe and rapidly fatal. Circulatory collapse (Farrell *et al.*, 1972; Trapnell, 1968), profound hypoxia (Ransom, Roses and Fink, 1973), hypocalcaemia (Trapnell, 1968), E.C.G. changes (Pollock, 1959) and intravascular consumption coagulopathy (Gabryelewicz, 1971; Greipp, Brown and Galnick, 1972) are well recognized complications of acute pancreatitis and they all reflect the severity of the disease process. The hypoglycaemia is interesting and, to the best of our knowledge, has not previously been described in this context. There would appear to be no ready explanation for it. The normal insulin levels make it unlikely that damage to the islet cells was responsible.

Conclusion

A further case of acute pancreatitis in a patient taking the oral contraceptive pill is reported. It is possible that the relationship between the pill and pancreatitis is purely fortuitous, although reports of previous cases would suggest otherwise. As hyperlipidaemia has been emphasized as a possible aetiological factor in these previous reports, the normal lipid levels in this case are of special interest.

It would seem wise to consider acute pancreatitis in any patient on the contraceptive pill who develops abdominal pain, regardless of the serum lipid levels.

Acknowledgment

We wish to thank Dr D. M. Goldberg who performed the biochemical investigations.

References

- BANK, S. & MARKS, I.N. (1970) Hyperlipaemic pancreatitis and the Pill. *Postgraduate Medical Journal*, **46**, 576.
- CAMERON, J.L., CRISLER, C., MARGOLIS, S., DEMEESTER, T.R. & ZUIDEMA, G.D. (1971) Acute pancreatitis with hyperlipemia, *Surgery*, **70**, 53.

- CAMERON, J.L., CAPUZZI, D.M., ZUIDEMA, G.D. & MARGOLIS, S. (1973) Acute pancreatitis with hyperlipemia: The incidence of lipid abnormalities in acute pancreatitis. *Annals of Surgery*, **177**, 483.
- DAVIDOFF, F., TISHLER, S. & ROSOFF, C. (1973) Marked hyperlipidemia and pancreatitis associated with oral contraceptive therapy. *New England Journal of Medicine*, **289**, 552.
- FARRELL, P., FITZGERALD, P., FITZGERALD, O., MCGEENEY, K., GEOGHEGAN, C. & HEFFERNAN, A. (1972) Shock in acute pancreatitis and hypovolaemia. *Gut*, **13**, 844.
- GABRYELEWICZ, A. (1971) Disturbances of blood clotting and fibrinolysis in acute pancreatitis. *Acta haematologica polonica*, **2**, 319.
- GLUECK, C.J., SCHEEL, D., FISHBACK, J. & STEINER, P. (1972) Estrogen-induced pancreatitis in patients with previously covert familial type V hyperlipoproteinemia. *Metabolism*, **21**, 657.
- GREIPP, P.R., BROWN, J.A. & GRALNICK, H.R. (1972) Defibrination in acute pancreatitis. *Annals of Internal Medicine*, **76**, 73.
- POLLOCK, A.V. (1959) Acute pancreatitis. Analysis of 100 patients. *British Medical Journal*, **1**, 6.
- RANSOM, J.H.C., ROSES, D.F. & FINK, S.D. (1973) Early respiratory insufficiency in acute pancreatitis. *Annals of Surgery*, **178**, 75.
- STACKHOUSE, K.L., GLASS, D.D. & ZIMMERMAN, B. (1966) Relationships of lipoprotein lipase and hyperlipemia in pancreatitis. *Surgical Forum*, **17**, 343.
- TRAPNELL, J.E. (1968) Pancreatitis—acute and chronic. *British Journal of Hospital Medicine*, **1**, 181.
- WANG, C., STRAUSS, L. & ADDLEBURG, D. (1958) Experimental pancreatitis and plasma lipids. *Gastroenterology*, **35**, 465.

Postgraduate Medical Journal (December 1975) **51**, 857–859.

Testicular tumour presenting as haematemesis

M. R. LOCK
M.B.B.S., F.R.C.S.

Professorial Surgical Unit, Westminster Hospital, London S.W.1

Summary

The case is presented of a malignant testicular tumour which presented with a haematemesis due to multiple tumour deposits in both stomach and jejunum. No similar case has been previously recorded. The pathology and aetiology of testicular tumours are discussed.

Case history

A well developed, hirsute lorry driver aged 29 years was admitted to Westminster Hospital on 8th February 1972 with a history of dizziness and palpitations for one week. Immediately before admission he had had a small haematemesis and noted darkening of his stools. There was a history of bilateral maldescent of the testes which had been treated at the age of eleven by injections.

On examination he was clinically anaemic, there was a fist-sized mass in the right scrotum which was non-tender and indistinguishable from the right testis itself. The left scrotum was empty and the testis was not palpable.

Investigations

Haemoglobin 3.9 g%; E.S.R. 51 mm/hr (Westergren); a chest X-ray showed multiple tumour deposits in both lungs.

* Present address: St. Mark's Hospital, City Road, London, E.C.1.

Progress

He was transfused with 6 units of blood. A barium meal and follow-through, and a gastroscopy were both normal, no focus of gastro-intestinal bleeding being identified. Two bone marrow biopsies were performed and they showed normoblastic erythropoiesis with a myeloid erythroid ratio of 2:1 which was suggestive of haemorrhage as the cause of the anaemia. Malignant cells were not present. He continued to bleed, including a massive melaena stool. Two further barium meal and follow-through examinations failed to show any lesion. Further transfusion raised his haemoglobin to 10.6 g%, when laparotomy was performed.

First operation 21.2.72

Blood was found throughout the lumen of the small and large intestines from the duodeno-jejunal flexure distally. The absence of gastric and duodenal lesions was confirmed by pylorotomy. A nodule was palpated along the anti-mesenteric border of the lumen of the proximal jejunum; on enterotomy this was found to be a smooth, soft, haemorrhagic lump 1 cm in diameter, bleeding copiously from its mucosal surface. Approximately 10 cm of the proximal jejunum was resected to include the lesion. The liver and para-aortic nodes were free of metastatic deposits. The right spermatic cord was divided