MEIGS' SYNDROME: A CASE REPORT

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Meigs in 1934,[1] and Meigs and Cass[2], drew attention to the occurrence of cases of ovarian fibroma associated with simultaneous fluid effusions in the abdominal and chest cavities, the effusions disappearing spontaneously when the tumour is removed.

In the 25 years since 1937, only a hundred or so cases of this syndrome have been reported, the age of the patients ranging from 9 to 75 years. Most of these cases have occurred in the U.S.A. and the apparent rarity of the syndrome may be explained by the fact that it is seldom recognized outside the gynaecological field.

Lemming[3] suggests that the benign tumour need not always be a fibroma, the important criterion being that the effusions disappear spontaneously on removal of the tumour.

Case Report. The patient, aged 57 (nulliparous), was admitted to hospital with two weeks history of breathlessness and tiredness, thought to be the result of a cold contracted four weeks previously.

On examination there was evidence of fluid in the right pleural cavity but no other respiratory abnormalities were demonstrated. The fluid was confirmed on x-ray; no tracheal deviation was seen. In the abdomen there was a large, irregularly-shaped, firm mass palpable on the left side, extending from the suprapubic to the epigastric region. The liver and spleen were normal. The temperature was normal, B.P. was 130/80, pulse 80 and the blood group was O, Rh +ve. The ECG showed left axis deviation. All other systems appeared normal.

Aspiration of the chest produced 7½ pints of a clear straw-coloured fluid containing small clusters of serosal cells and leucocytes, but no neoplastic cells. Subsequent x-ray revealed no evidence of disease of lung parenchyma and repeated tests for tuberculosis baccilli on the fluid and sputum were negative.

Laparotomy was performed some days later and a large left ovarian tumour with few adhesions was removed. Two pints of clear, straw-coloured, peritoneal fluid were also aspirated. No other abnormalities were discovered. The day following the operation, six pints of pleural fluid were aspirated (nine days after the first chest aspiration).

Apart from a transient attack of phlebitis, the patient made an uneventful

recovery. Several days after operation, x-ray showed the chest to be completely clear of fluid.

Pathologist's report. The tumour which was removed measured 25 cms. in diameter and its outer surface was colluted. The cut surface showed solid and cystic areas with the latter predominating. On microscopic examination of sections from six different parts of the tumour, it was seen that all showed similar histological appearance despite the gross difference of the solid and cystic areas. The septa of the tumour were all delicate and the lining was uniformly of tall columnar cells. The cytoplasmic boundaries were indistinct in many parts where they appear to merge with the pseudomucinous contents of the loculi. There were also occasionally short papillary projections of the epithelium and stroma, and epithelium was always one cell thick. There were no signs of malignancy and the tumour was a pseudomucinous cystadenoma of the ovary.

Examination of the body fluids. Blood examination was carried out before surgery and the results are shown in table I.

TABLE I

PRE-OPERATIVE BLOOD ANALYSIS OF THE PATIENT

Haemoglobin	108 per cent—15.86 gm. per cent	P.C.V.: 42 per cent
E.S.R.	16 (Westergren)	
White cell count	6000	Blood urea: 20 mgm. per cent
Blood film	Neutrophyll 72 Lymphocyte 22 Monocyte 6	No abnormality detected
Na^+: 126 m.eq./l	K^+: 4.5 m.eq./l	Cl^-: 102 m.eq./l

The results of initial investigations carried out on the pleural and peritoneal fluids are shown in table II.

TABLE II

SOME INFORMATION ON THE PLEURAL AND PERITONEAL FLUIDS

Fluid	*Colour of fluid*	*S.G.*	*pH*	*Total protein (17% N.)*	*Other information*
Pleural	Clear straw-coloured	1.016	7.8	3.56	81 leuco-cytes/cm.
Peritoneal	Clear straw-coloured	1.017	8.0	3.62	—

The usual urine tests, e.g. for albumin, sugar, S. G., Bence-Jones' proteins, etc., were carried out before surgery and nothing abnormal was detected.

Electrophoretic Separations of the Fluids and Sera. The fluids investigated were samples of pleural and peritoneal fluids, a normal serum, and the patient's serum before, and two weeks after operation. The materials were used as fresh as possible and in most instances the paper electrophoretic separations were done overnight on the day that the samples were received.

The separations were done on strips of Whatman No. 1 paper in a Tiselius

type of apparatus using a veronal barbiturate buffer[4] (pH 8.6; ionic strength: 0.1[5]) at 0°C. They were run overnight for 16 hours at 200 volts/strip. The strips were dried and stained with 1 per cent bromophenol blue in 95 per cent ethanol saturated with mercuric chloride[6]. The papers were then washed with water until the background was colourless, washed with dioxan and finally with ether before drying. They were scanned, immersed in liquid paraffin, on an Eel "scanner". A typical scan diagram for each is shown in Fig. 1. The relative protein concentrations were worked out for all by planimetry,[7] using the method of Tiselius and Kabat[8] to separate the fractions. The results obtained from six electrophoretic patterns of each sample were averaged and these are seen in table III.

TABLE III

PROTEIN RATIOS OF THE SAMPLES BY PLANIMETRY, EXPRESSED AS PER CENT OF TOTAL AREA

Fluid	*Albumin*	α1	α2	β	γ	*A/G Ratio*	*Total Protein* (17% *N.*)
Normal serum	53.63	3.99	9.40	13.39	19.61	1.140/1	6.51
Patient's serum*	44.97	5.15	12.97	18.23	18.48	0.820/1	5.94
Patient's serum†	38.00	5.05	11.65	18.80	26.50	0.613/1	5.92
Pleural fluid	49.98	7.48	8.00	13.93	20.61	0.994/1	3.56
Peritoneal fluid	50.66	6.47	7.80	14.61	20.46	1.026/1	3.62
Ave. of 12 normal sera[9]	57.40	4.16	8.52	14.00	16.00	1.350/1	—
Range of normal sera[9]	52.1—60.5	2.4—6.0	6.8—11.2	12.4—17.8	11.0—20.6	—	—

(*Before operation. †After operation.)

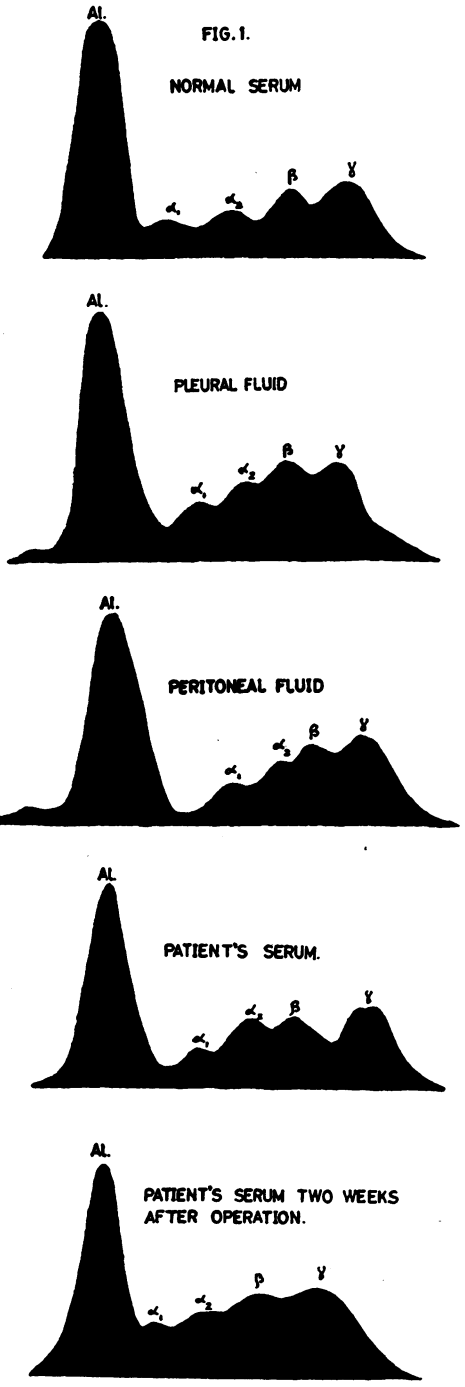
Unfortunately, insufficient serum was available to allow a complete protein analysis by preciptation to be carried out.

A complete blood analysis was again undertaken 12 days postoperatively and nothing abnormal was detected.

Discussion

Changes in serum proteins are frequently significant, but as there are many possible reasons for each variation, the changes are not specific.

Porter[10] states that at pH 8.6 it is not always possible to get a good separation between the β- and γ- globulins. Using refined systems of paper electrophoresis, it has been possible to separate as many as nine[11] or even seventeen[12] components from serum. In this study, electrophoretic separations were first tried on thick


Al.
FIG. 1.
NORMAL SERUM
β
γ
Al.
PLEURAL FLUID
β
γ
Al.
PERITONEAL FLUID
β
γ
Al.
PATIENT'S SERUM.
β
γ
Al.
PATIENT'S SERUM TWO WEEKS
AFTER OPERATION.
β
γ

papers, but a much better separation was obtained using paper number 1, and the usual five bands were separated.

The planimetric results are within the normal range for the normal serum, but it is seen that the patient's serum before operation was deficient in albumin, and had excess of α_2– and β–globulins. Deficiency of albumin may be due to malnutrition, impaired synthesis and several diseases[13]. The patient in question appeared to be slightly cachectic, and this was possibly due to the big loss of all protein components of the serum into the ascitic and pleural fluids. If this loss continued for any period of time, it must affect both the serum proteins and the body nitrogen balance. An increase of the α_2–globulin fraction has been seen in some cases of tumour, but Stewart and Dunlop[14] suggest that high δ_2–values during the " catabolic phase " after injury may be a reflection of changes in cell membrane permeability, and this change in membrane permeability may account for the high value recorded here. An increase in β–globulin has been shown to be due to a raised lipoglobulin fraction. In cases with a high protein loss, such as the present one, any dietary protein will probably be used for protein synthesis, and so there may be undue mobilization of fat reserves which would cause an increase in the β–globulin fraction.

The patient's serum after operation showed a further decrease in albumin, a substantial rise in δ–globulin, and although the α_2– and β–globulins were still high, they did appear to be returning to normal. Wilkinson[15] has pointed out that there is a depression of the albumin fraction after surgery which may persist for more than two weeks, and this may, to some extent, account for the further reduction found here. The high δ–globulin may be due either to a general increase following major surgery, or to the appearance of a patch of superficial phlebitis.

The two effusions were found to be of almost identical composition and specific gravity, although slightly different in pH. This confirms the findings of other workers and suggests that they have the same origin. High pH values for the effusions have previously been reported.[16] As the paper electrophoretic patterns of the two effusions show the presence of all the serum components, and possibly some pre-albumin, it would seem that there must be a greatly increased permeability at the site of origin to allow the globulins to diffuse.

Meigs[17] demonstrated in some of his cases, that India ink injected into the ascitic fluid, quickly passed into the pleural fluid. As no dye appeared in the blood or corpuscles, it was assumed that passage was solely through the lymphatics. Yoffey and Courtice[18] have discussed fully the lymphatic drainage of the peritoneum, and

Efskind[19] reported that Chinese ink injected intra-peritoneally was eliminated quickly by the retro-sternal lymphatics and was to be found, within half-an-hour, in the lymphatics on the pleural side of the diaphragm. Florey[20] pointed out that the very porous nature of the lymphatics on the diaphragmatic surface appears to be unique, having the ability to pass small particulate matter.

It has also been observed[21] that the lymphatic drainage does not take place evenly over the whole diaphragmatic surface, but is more extensive on the right side. These anatomical findings may account for the observation[3] that two-thirds of all cases reported have hydrothorax on the right side, and in the remaining one-third, the effusion was either in the left pleural space or bilateral.

Many aspects of the syndrome have never been satisfactorily explained. It is difficult to understand why it has such a low incidence and why hydrothorax does not appear in all cases of ovarian tumour with ascites. How the fluid is produced and how hydrothorax develops are other questions that remain unanswered. All aspects of the syndrome however must be caused in some way by the tumour since there is an immediate recovery after tumour removal.

Genesis of the ascitic fluid may be explained in four possible ways. Firstly, it may be produced by transudation from the tumour surface. Secondly, there may be special secretory cells within the tumour which produce the fluid. If this is so, much closer examinations, and differential histological staining of sections of the tumour, may reveal differences in the cellular structure between tumours producing the syndrome and those not producing the syndrome. Thirdly, it may be produced as a result of disturbances in hormone metabolism. Some ovarian tumours are oestrogenic. Hypersecretion of oestrogens has an adverse effect on electrolyte balance[22, 23] which is usually shown as increased water percentage in the intracellular spaces of the uterus. Even so, Engel[24] states that oestrogens may have some influence on cell membrane permeability, although their main action is on certain enzyme systems. Lastly, the fluid may be produced by mechanical stretching of the peritoneal lining above the ovary as the tumour grows in size. This would bring about an increase in the size of the inter-cellular spaces which could ultimately be such that lymph would escape through them.

No matter how the ascitic fluid is produced in the peritoneal cavity, it may be assumed that it passes through the lymphatics of the diaphragm, but it is difficult to see how hydrothorax arises if the lymphatic channels remain intact. In the early stages of the syndrome when the tumour is small, it is the build up of the hydrothorax which is the dominant symptom,[3] and for this reason, many

cases have been diagnosed initially as pleurisy or tubercular polyserositis. There are three possible explanations for the genesis of hydrothorax. Clementsen,[25] and later Lemming,[3] suggested that its formation involves a blockade of the intrathoracic lymph flow due to scarring by previous tuberculous infection. The reduced lymph drainage would cause fluid to accumulate gradually in the pleural cavity. It could also be explained by a hypothesis suggesting that the tumour in some way alters the permeability of the wall of the lymphatic vessels throughout the body. As the pleural and peritoneal cavities are the only places in the body where lymph drainage is through a " hollow " cavity, then these would be the only two sites where a large volume of fluid would accumulate. Finally, it could be postulated that the occurrence of hydrothorax is a purely mechanical procedure. If the volume of fluid produced in the peritoneal cavity was much greater than that which could be drained away by the normal lymphatics in the pleural cavity, then hydrostatic pressure building up within these lymphatics would cause them to swell and ultimately allow fluid to diffuse into the cavity. Once the tumour is removed and fluid production ceases, the excess fluid would be drained away by the normal drainage pathways.

Summary

A case of Meigs' syndrome has been studied and all case reports are summarized. Paper electrophoretic separations of sera and of the effusions were carried out and an explanation of the findings has been attempted. A short discussion on the genesis of the syndrome is given.

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PHYSIOTHERAPY IN STROKE ILLNESS

The main problem a " stroke " patient has to face is frustration. The physiotherapist must aim to restore the patient's independence, at all stages of recovery, within the limits of his disability.

All activities demand specific patterns of movement, and their proper performance depends upon free joint movement, the necessary muscle power and finally, help to relearn the activity.

Two main factors lead to deformity: the inability of the patient to move himself, with the consequent development of joint stiffness, and the development of muscle spasm which not only prevents normal movements but also causes painful joint stiffness.

The physiotherapist has to re-educate the patient's sense of balance, maintain the strength of the unaffected side of the body, and train the patient in the best use of his available power so that he may learn to help himself.

Maintenance of range of movement during the early stages must be carried out passively, or stiffness will make functional activities more difficult. The patient can do some of these movements for himself, but the responsibility rests with the physiotherapist to show him how they may most easily be done, and to ensure that full range is maintained.

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