Serous Effusions

AN ATTEMPT AT A CLINICAL-PATHOLOGICAL CORRELATION

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SUMMARY

One hundred and three serous fluid examinations were analysed to try to discover a simple formula whereby a pathological and/or aetiological diagnosis may be made without recourse to multiple, invasive, sophisticated or expensive procedures. It was hoped also to find features which would identify effusions arising from similar mechanisms. These hopes were not fulfilled. Instead, the study prompts a re-examination of traditional concepts on the question of transudates and exudates, as occurs in our group of patients belonging to the Swazi nation. The literature, with relevant points on the issue in general, is examined.

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Single or multiple serous effusions are commonly encountered in a Black hospital practice. The causes of such fluid collections are many, some clinically obvious, others requiring special diagnostic procedures.

At the Mbabane Government Hospital, handling about 9 000 admissions per annum, serous effusions constitute 5% of all medical cases. This article reports the results of a study conducted over a period of 3 years, in order to derive some benefit from a comparative analysis. The hoped-for gains could include minimising the need for invasive, often expensive, complicated laboratory procedures; obviating the need for multiple punctures and other painful procedures (if an inferential diagnosis can be made from only one puncture); providing support for the traditional characterisation of pathological fluids, especially if such findings could make pathological or aetiological diagnosis easier or possible.

MATERIAL AND METHODS

One hundred and three patients were selected, including 30 with ascites, 24 with pleurisy, 16 with ascites plus pleurisy, 8 with pericardial effusion, 6 with pleurisy plus pericarditis and 6 with polyserositis. A combination of ascites and pericarditis was not seen.

The criteria for selection were that all patients were to be adults; only first hospital admissions before any form of treatment had been given; and the collection of a sizeable quantity of fluid. All ward analyses and biopsy procedures were done by one person.

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Definitions and Terminology (Tables I - V)

Naked-eye appearances of serous fluid are described as yellow or straw-coloured, bloody, or heavily bloody.

An exudate is implied when the specific gravity exceeds 1,016 and the protein level is above 3 g/100 ml.

Transudate describes the specific gravity and the protein level as lower than 1,016 and 3 g/100 ml, respectively.

The fluid is indeterminate where the specific gravity and the protein level are discordant.

A probable diagnosis is based on strong collateral evidence, a definite diagnosis on pathological proof.

RESULTS

Forty-nine examinations of ascites alone or associated with pleurisy and/or pericarditis are shown in Table I.

Regarding ascites only, analysis showed the following: the specific gravity was consistently low, with no recognisable relationship to the protein level, the latter exhibiting a general exudate range, except for case 10 (cirrhosis), in which the protein level was 2,8 g/100 ml in the face of a serum albumin level of 3,2 g/100 ml.

The average or single protein content showed no special correlation with the pathological diagnosis, neither did the presence of large amounts of blood influence this measurement. By far the highest reading was recorded in a heavily bloody aspirate, while a hypoproteinaemic case contained a high exudate amount.

Fluid sugar determination was of no value.

The ascitic effusion was of no predictive use as to the nature of other associated effusions.

There was a total of 4 patients with macroscopical blood in the effusion. Microscopical blood would presumably give a higher figure.

Twenty-four examinations of pleural effusions only are shown in Table II.

The specific gravity of pleural fluids shows a decided and consistently high value, irrespective of site, side, and size of effusion collection.

There is no detectable correlation among such parameters as specific gravity, protein level and macroscopical appearance, and pathological diagnosis.

The average protein content is higher than in ascitic fluid, by 1 g/100 ml. Case 3 demonstrated an odd

TABLE I. ASCITES AND/OR OTHER SEROSITIS

No.	Fluid colour	Specific gravity	Protein (g)	Sugar (mg/100 ml)	Diagnosis
1	Yellow	1,010	4,0	100	CCF
2	,,	1,009	3,4	30	Cirrhosis—hypoglycaemia
3	,,	1,060	3,7	98	Cirrhosis
4	,,	1,010	4,0	76	Undiagnosed—high serum globulins
5	,,	1,010	3,5	80	Undiagnosed—additional tests no value
6	,,	1,008	3,2	70	Possible cirrhosis
7	,,	1,020	3,0	104	Carcinoma pancreas—severe obstructive jaundice
8	,,	1,018	3,6	90	CCF
9	,,	1,008	4,5	115	Malignant hepatoma
10	,,	1,016	2,8	80	Cirrhosis—serum albumin 3,2 g/100 ml
11	,,	1,010	4,7	70	Yellow pleural fluid, exudate
12	,,	1,010	3,3	50	CCF—yellow pleural fluid, exudate
13	,,	1,013	3,7	70	nephrotic—yellow pleural fluid, transudate
14	,,	1,012	3,7	68	Cirrhosis—heavily bloody pleural fluid, exudate
15	Deeply bloody	1,012	4,2	80	Hepatoma
16	Yellow	1,010	5,0	106	Disseminated TB—yellow pleural fluid, exudate
17	,,	1,016	3,0	80	Cirrhosis
18	Faintly bloody	1,016	3,6	130	Bloody pleural fluid, exudate
19	Yellow	1,012	3,3	206	Cirrhosis
20	,,	1,010	4,0	55	Probable TB—associated pleurisy, exudate
21	,,	1,012	4,7	102	Cirrhosis
22	Blood-stained	1,010	5,8	100	Malignant hepatoma
23	Yellow	1,005	3,6	86	Hepatoma, blood-stained pleurisy, exudate
24	,,	1,008	3,3	104	Diagnosis unknown, associated pleurisy and pericarditis
25	,,	1,012	3,8	111	Probable cirrhosis, yellow pleural fluid
26	,,	1,008	3,0	100	CCF, yellow pleural fluid
27	,,	1,014	4,4	80	Hepatoma, jaundice
28	,,	1,014	4,0	135	CCF, yellow pleural fluid
29	,,	1,010	3,2	70	Probable TB, yellow pleural and pericardial fluids
30	,,	1,012	4,7	85	CCF, yellow pleural fluid, possible TB
31	Blood-stained	1,012	3,3	55	Possible TB
32	Yellow	1,010	3,6	100	Undiagnosed, yellow pleural and bloody pericardial fluids
33	,,	1,016	3,0	94	TB, pleural fluid, yellow and exudate features
34	,,	1,002	3,3	70	Probable TB
35	,,	1,008	5,0	90	TB, pleural fluid, exudate
36	Blood-stained	1,016	3,6	74	Undiagnosed
37	Yellow	1,009	3,0	100	Nephrotic syndrome generalised oedema
38	,,	1,012	4,2	86	CCF
39	,,	1,010	3,7	100	Cirrhosis
40	,,	1,010	3,4	69	Cirrhosis, yellow pleural fluid
41	,,	1,014	3,6	108	Probable TB, yellow pleural fluid
42	,,	1,016	4,0	80	Cirrhosis
43	,,	1,012	3,4	102	Possible TB
44	,,	1,016	4,7	79	Possible TB
45	,,	1,014	3,4	95	CCF
46	,,	1,010	3,0	100	Cirrhosis
47	,,	1,012	3,2	84	Malnutrition, hypoproteinaemia
48	,,	1,010	3,5	76	Cirrhosis
49	,,	1,012	4,1	95	Cirrhosis

combination of the highest protein level of the series with the lowest specific gravity of 1,016, due to an amoebic abscess. The sixth patient of this series presented the classical twin features of an exudate with a specific gravity of 1,024 and a protein level of 6,2 g/100 ml, probably due to tuberculous pleurisy.

Sixteen of the 24 examinations qualify as exudates, while the remaining 8 were indeterminate effusions.

TABLE II. PLEURISY CASES

No.	Colour of fluid	Specific gravity	Protein (g)	Sugar (mg/100 ml)	Diagnosis
1	Yellow	1,018	3,5	53	CCF
2	Heavily bloody	1,018	4,0	100	Probable TB
3	Yellow	1,016	5,8	76	Amoebic liver
4	Faintly bloody	1,020	4,1	80	Probable TB
5	Indeterminate	1,017	5,7	79	Possible TB
6	Yellow	1,024	6,2	79	Probable TB
7	,,	1,020	4,2	104	,,
8	,,	1,020	4,0	93	Pneumonia
9	,,	1,014	3,6	80	Cirrhosis—left-sided effusion
10	Heavily bloody	1,016	3,3	87	TB—biopsy proven
11	Yellow	1,020	5,1	35	TB— ,,
12	,,	1,016	3,3	110	Non-specific pleurisy
13	,,	1,018	4,0	76	Possible TB—biopsy unsuccessful
14	,,	1,018	5,5	48	Probable TB, biopsy failed
15	Heavily bloody	1,016	4,0	70	Hepatoma—right-sided effusion
16	Yellow	1,018	3,6	85	Bronchial carcinoma
17	,,	1,016	3,6	70	Non-specific pleurisy
18	Heavily bloody	1,020	4,4	82	TB—biopsy positive
19	Yellow	1,016	4,7	85	CCF
20	,,	1,018	5,1	92	TB—biopsy positive
21	,,	1,020	5,0	95	TB + haemosiderosis, biopsy positive
22	Faintly bloody	1,016	3,3	84	Probable TB, biopsy failed
23	Yellow	1,018	4,4	100	Non-specific pleurisy + haemosiderosis
24	Yellow	1,018	4,0	94	TB—biopsy positive

TAELE III. ASCITES-RELATED PLEURAL EFFUSIONS

No.	Fluid colour	Specific gravity	Protein (g)	Sugar (g/100 ml)	Diagnosis
11	Yellow	1,018	3,8	79	CCF
12	,,	1,020	4,0	68	CCF
13	,,	1,002	1,8	78	Nephrotic
14	Heavily bloody	1,002	5,8	50	Cirrhosis
16	Yellow	1,020	3,5	65	TB
18	Heavily bloody	1,020	5,2	80	Probable TB
20	Blood-stained	1,018	4,6	50	Probable TB
22	Blood-stained	1,020	4,0	85	Hepatoma
24	Yellow	1,016	3,8	95	Probable cirrhosis
25	,,	1,020	3,6	90	CCF
27	,,	1,018	4,0	60	CCF
30	,,	1,016	4,1	100	CCF
33	,,	1,022	5,0	72	TB
35	,,	1,020	4,8	70	TB
40	,,	1,020	3,6	85	Probable TB
41	,,	1,018	4,7	64	Cirrhosis

Macroscopical blood was observed in 6 instances, 4 being heavily bloody. Two of the patients with thick muddy haemorrhagic fluids died shortly after admission.

Sixteen patients with ascites plus pleural effusion are shown in Table III, for the purpose of comparing them with the pure pleurisy cases. The general characteristics of the 2 groups are identical. The ascites and pleurisy secretions under the same roof are dissimilar.

TABLE IV. POLYSEROSITIS CASES

Case No.	Source of fluid	Colour	Specific gravity	Protein (g)	Sugar (mg/100 ml)	Diagnosis
23	Pericardium	Yellow	1,020	4,6	90	Unknown
23	Pleura	,,	1,022	4,0	86	,,
28	Pericardium	Blood-stained	1,022	5,0	70	Probable TB
28	Pleura	Yellow	1,022	5,5	87	,,
32	Pericardium	,,	1,018	4,4	80	Undiagnosed
32	Pleura	,,	1,020	4,0	90	,,

Six patients with polyserositis are shown in Table IV. The 3 pericardial effusions are all exudates, one of them blood-stained. In addition, these fluids are dissimilar to the associated ascitides, and we recognised no special pattern from the 3 serous effusions, presumably due to a single mechanism.

Pericarditis plus pleurisy: It is noteworthy that a blood-stained pericardial effusion is often encountered. However, little may be inferred from this small number of patients.

Isolated pericarditis: There were 8 examinations, 6 of them blood-stained. The value of this finding is minimised by lack of pathological proof and the small number of patients. Tuberculosis was probable in 2, and assumed in 4, with good therapeutic response.

DISCUSSION AND CONCLUSIONS

This study has many short-comings, hence the conclusions are tentative and the discussion largely speculative.

It does seem that the macroscopical and biochemical analysis of serous effusions has too many limitations to be of any use in any classification or to suggest a diagnosis. Some procedure that will supply definitive pathological proof is therefore mandatory.

It is not surprising that this is so, if we appreciate that many factors contribute to the final nature and composition of plasma filtrates. The accumulation of a fluid inside or across a body membrane, whether normal or abnormal, is a dynamic process, The cellular and chemical constituents of the filtrate will, irrespective of the initiating mechanism, depend on the site of collection and the mobility of pericardial and pleural cavities having an inherent shearing force; the rapidity of collection and volume of the fluid—this would affect the readiness with which and the degree to which an enclosed fluid would change; the duration of the process of filtration; and the capacity of the membrane limiting the body cavity.

It will be for these reasons, too, that serous membranes seem to react differently to the same pathological agent.

The results of other workers do not help us to understand the problem completely. Thus, Sochocky[1] dismissed the possibility of finding a single diagnostic test after a detailed analysis of pleural effusions. On the contrary, Leuallen *et al.* endowed the protein level of pleural fluids with a predictive diagnostic value. Kirkeby *et al.* claimed a yet unconfirmed usefulness for enzyme tests. These latter workers are quoted by Sochocky.[1]

Short of an exact pathological finding, some suggestive though general indices may characterise certain types of effusions. For instance, heavy cellularity and sediment formation is strongly in favour of an actively formed exudate.[2] Lymphocytic exudates are usually a feature of TB, lymphoma and carcinoma, while a neutrophilia is compatible with bacterial pyogenic infections. Abnormal lymphocytes are common in lymphomata, and recognisable malignant cells are diagnostic.

Pillay[3-5] was of the opinion that heart failure results in effusions which were characterised by the fact that ascites forms more readily than pleural transudate; the ascitic protein tends to be higher, the pleural protein rises after a diuresis; and he postulated a linear relationship between the serum protein level and the transudate protein. These were interesting results, which deserve repeat evaluation. The present study did not yield further information on these points.

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