DIAGNOSTIC VALUE OF THE GLUCOSE CONTENT OF SEROUS PLEURAL EFFUSIONS

BY

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Bacteriological confirmation of the tuberculous origin of non-purulent pleural effusions may be difficult to obtain by direct culture, while the result of a guinea-pig inoculation will not be available for several weeks. Where negative results are obtained, radiography and sputum examination may not be helpful, and a search was therefore made for other diagnostic criteria. Experience gained from glucose estimations of the cerebrospinal fluid in tuberculous meningitis directed attention to the possible diagnostic value of the glucose content of pleural fluid.

TABLE I.—*Summary of Recent Investigations*

Author	No. of Cases	Diagnosis	Fluid Glucose Level (mg./100 ml.)		Method
			Max. Value	Min. Value	
Foord <i>et al.</i> (1929)	34	Prim. tuberculous (9) Sec. " (6) Non- " (6)	116 122 99	64 91 70	Not stated
Pinner and Moerke (1930)	32	Tuberculous	82	0	Folin-Wu
Nassau (1941)	57	Tuberculous	136	4	Hagedorn-Jensen
Gelenger and Wiggers (1949)	33	Tuberculous (10) Non-tuberculous (21) " " (1)	39.5 140	A trace 62 Less than 40	Folin-Wu
Present series	61	Tuberculous (25) Non-tuberculous (30) " Undiagnosed " (6)	118 200 96	10 67 62	Hagedorn-Jensen

Table I summarizes the results of four recent investigations into the glucose content of pleural effusions and includes the results of the present investigations. Foord *et al.* (1929) found that there was little variation in the glucose level of tuberculous and non-tuberculous fluids, and also that the level of the sugar in the pleural fluid paralleled the level in the blood. The method of estimating the glucose is not given, and the lack of agreement between their results and those of others may be due to the use of a method less specific for glucose than the Hagedorn-Jensen and Folin-Wu methods. Pinner and Moerke (1930) investigated only tuberculous fluids. Although they concluded that no significance could be attached to the glucose content, their results agree with those of Nassau (1941) and Gelenger and Wiggers (1949), who found the glucose content in tuberculous effusions depressed to a variable extent when compared with blood values.

Nassau used the glucose level in tuberculous pleural effusions as a prognostic aid, and stated that values over 70 mg./100 ml. were favourable and values under 50 mg./100 ml. were unfavourable prognostically. Gelenger and Wiggers in their series found that the glucose level was below 39.5 mg./100 ml. in 10 tuberculous effusions and above 60 mg./100 ml. in 21 non-tuberculous cases, and conclude that if the sugar level is above 60 mg./100 ml. it is unlikely that the effusion is tuberculous. They quote one non-tuberculous case (bronchogenic carcinoma) in which the level is below 40.

In the present series, 61 cases were examined, 25 of which were tuberculous, 30 non-tuberculous, and 6 "undiagnosed." The term "undiagnosed" is used because bacteriological confirmation of the tuberculous origin of the effusion was not forthcoming, and no other cause presented. In all the tuberculous cases the diagnosis was confirmed by bacteriological examination of either sputum or pleural fluid, or at necropsy. Of the 30 non-tuberculous cases, 13 were due to congestive heart failure, 7 to carcinoma, 1 to leukaemia, 2 followed pulmonary infarct, 1 was in association with ascites and pancreatitis, and 6 were post-pneumonic. The pleural fluid was collected two hours after the patient's last meal. In many of the cases concomitant blood sugars were taken, and these values lay within a normal range. Fluoride was used to prevent glycolysis, and the glucose was estimated by the Hagedorn-Jensen method. The results of the investigation are set out in Table II. No correlation could be found between the blood and pleural fluid glucose levels (Table III).

TABLE II.—*Results in Present Series*

Glucose Levels (mg./100 ml.)	Tuberculous	Non-tuberculous	" Undiagnosed "
Over 100	4 cases	24 cases	1 case
90-100	3 "	3 "	1 "
80-90	1 case	2 "	2 cases
70-80	2 cases		2 "
60-70	2 "	1 case	
50-60	1 case		
40-50	3 cases		
30-40	2 "		
20-30	3 "		
10-20	4 "		
Total No. of cases	25	30	6

TABLE III.—*Blood and Pleural Fluid Glucose Levels in Tuberculous and Non-tuberculous Patients*

Tuberculous			Non-tuberculous		
No.	Pleural Fluid mg./100 ml.	Blood mg./100 ml.	No.	Pleural Fluid mg./100 ml.	Blood mg./100 ml.
1	21	121	2	126	116
8	67	83	3	117	96
9	76	79	4	151	133
10	76	138	5	105	103
11	80	70	6	67	85
12	53	80	7	90	73
17	10	102	13	100	93
18	96	110	14	100	110
20	89	87	15	104	134
23	55	91	16	84	124
24	95	106	21	81	67
			22	132	120
			25	81	85

Discussion

All 30 non-tuberculous pleural effusions had a glucose level of over 60 mg./100 ml. and in 24 of these the level was over 100 mg./100 ml. In the 25 tuberculous cases, 13 showed glucose values below 60 mg./

100 ml. Of the 12 cases above this figure, one was complicated by amyloid disease, while another was a diabetic with a glucose level in the pleural effusion of 220 mg./100 ml.

A glucose level of under 60 mg./100 ml. would therefore appear to be strongly suggestive of the tuberculous origin of a serous pleural effusion. Where the glucose level is over 100 mg./100 ml. the effusion is probably of non-tuberculous origin.

The six "undiagnosed" cases had glucose levels ranging from 62 to 96 mg./100 ml. Four of these six cases were considered clinically and radiologically to be of tuberculous post-primary type, but bacteriological confirmation was lacking. The four patients were adolescents or young adults, with straw-coloured lymphocytic effusions of recent onset, and gave positive Mantoux reactions.

There were eight tuberculous patients with glucose levels between 60 and 100 mg./100 ml. and seven non-tuberculous. The glucose values in the six "undiagnosed" cases would therefore be of no value in deciding the aetiology of the effusion.

Summary

The pleural glucose level has been studied in 61 cases of serous effusion.

In these cases: (a) a glucose level of less than 60 mg./100 ml. was found only in tuberculous effusions; (b) the majority of tuberculous effusions had glucose levels of less than 100 mg./100 ml., while most non-tuberculous effusions had levels above 100 mg./100 ml.; and (c) no significance could be attached to glucose levels of between 60 and 100 mg./100 ml.

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ACUTE PORPHYRIA WITH UNUSUAL FEATURES

BY

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In view of the recent interest in this disease and its comparative rarity in this country it is thought worth while to record the following case, which presents some unusual features.

Case Report

The patient, a woman aged 23, was admitted in December, 1949, to a surgical ward of the Southern General Hospital with a history of attacks of abdominal pain and vomiting of one week's duration. The pain usually started in the right hypochondrium and travelled forward across the epigastrium and through to the back in the upper lumbar region. It was spasmodic in nature, and severe, and an attack lasted for 15 to 30 minutes. Shortly after the onset of the pain she vomited. These attacks occurred three or four times a day. Her bowels had been regular until two days before admission, since when they had been constipated. Three days before admission her urine was red in colour on one occasion. Her menstrual history was normal, and she had just finished a period when the pain began.

There was nothing relevant in her past history except that she had had a similar attack of pain and vomiting with the passage of red-coloured urine one year previously, when

she was treated for "kidney trouble" with rest in bed for six weeks at home. No further information is available about this illness.

On examination the patient was seen to be a well-grown and well-nourished woman who looked ill. She had definite guarding and tenderness in the right loin and hypochondrium but none elsewhere. Her temperature was normal, pulse 72, and respirations 16. No abnormality was detected clinically in any of the other systems. The urine was dark in colour but gave a negative reaction for both blood and bile. The test with Ehrlich's aldehyde reagent, however, was strongly positive. The importance of this was not appreciated at the time. On the fourth day a trace of bile was detected in the urine (iodine test) and a faint icteric tinge was noticed at the corners of both sclerae. The blood bilirubin then was 0.6 mg. per 100 ml., the thymol turbidity test for liver function was ++, prothrombin time 22 seconds, icteric index 4. The blood picture was normal.

In the meantime the patient's condition had not improved. The attacks of pain had persisted with almost unabated severity and were still accompanied by guarding and tenderness in the right hypochondrium. In view of this fact and the laboratory findings it was decided to perform a laparotomy with the tentative diagnosis of gall-stones. At operation, however, the biliary tract appeared perfectly normal and no other intra-abdominal abnormality was found. It was then that the diagnosis of acute porphyria was proposed. Chemical tests and spectroscopy of the urine speedily established it.

The patient had a post-operative cough and later developed pneumonia. It was noticed at that time that she had some difficulty in coughing and that her sclerae assumed a plum-coloured tinge. Temporarily she made a partial clinical recovery, but her cough persisted. On the seventh post-operative day she felt so well that she asked to be allowed to get up, and enjoyed it. She then complained of attacks of numbness in both thighs, occurring only at night and lasting for about one hour; but there was no objective evidence of peripheral neuritis, and the cerebrospinal fluid was normal. On the 14th day she first complained of weakness and could hardly raise herself in bed. The trunk muscles, thighs, and upper arms showed great loss of power, while the reflexes appeared normal. There was some improvement in the right arm and the thighs after two days, but the other affected muscles remained weak. At this time she developed tachycardia, 120 a minute, and her blood pressure rose from 108/75 to 135/95 mm. Hg. The attacks of abdominal pain had returned on the ninth post-operative day and were severe. On the 18th post-operative day she lost her voice and did not regain it. On the 24th day she had great difficulty in swallowing, and next day the diaphragm became involved and she died with a generalized flaccid paralysis.

During the whole course of her illness the urine was pigmented, the intensity varying from a faint pink tinge to a deep port-wine colour.

Further Investigations.—Once the diagnosis of acute porphyria was established a number of other investigations were carried out in an attempt to find out to what extent the different systems were involved in the disease. The liver function tests carried out on the 19th post-operative day were as follows. Plasma proteins: total, 8.12 g.% (albumin 3.88 g., globulin 4.24 g.). Laevulose-tolerance test: fasting 2.4 mg., half-hour 8 mg., one hour 13.1 mg. per 100 ml. Thymol turbidity test ++; cephalin-flocculation test negative; van den Bergh test negative, direct; blood bilirubin, 1.35 mg. per 100 ml.; icteric index, 6.

Bile could not be detected in the urine post-operatively. On the 12th post-operative day the serum chloride and sodium levels were 325 mg.—336 mg. per 100 ml. respectively. Renal-function tests were carried out from the 12th to the 18th post-operative day and showed no abnormality: urea-clearance test 82%; urea-concentration test, concentrated to 4.6 g.% in the second hour; blood urea, 26 mg. per 100 ml. The blood picture on the ninth post-operative