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FAT EMBOLISM AND ALCOHOLIC PSYCHOSIS

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INTRODUCTION

The role of fat embolism in the genesis of the acute picture of alcoholic psychosis and in the causation of death in some such cases has been outlined by Lynch *et al.*[6,7] In autopsy studies these workers found fat emboli in the cerebral vessels in 90% of chronic alcoholic patients with mixed pure fatty and cirrhotic livers. Many of these patients had respiratory symptoms and delirium tremens during life. Furthermore, in a series of patients with delirium tremens these authors found fat globules in the sputum, the occurrence of which could be correlated with the clinical course of the psychosis — the number of globules in the sputum increasing as the condition worsened and decreasing as recovery occurred. It was concluded that fat embolism occurred commonly in chronic alcoholics and that it played a major part in the aetiology of alcoholic psychosis.

Chronic alcoholism is common in the White population of Johannesburg and a large percentage of the admissions to the Johannesburg General Hospital include patients with a common background of prolonged and excessive alcoholic intake.

A control study was therefore carried out at the hospital to evaluate clinically the significance of fat globules in the sputa of alcoholic patients.

MATERIAL AND METHODS

Alcoholics and patients with other acute and chronic medical conditions admitted to this hospital were studied.

An arbitrary subdivision into 3 groups was made:

1. Chronic alcoholics with acute alcoholic psychoses.
2. Chronic alcoholics without psychoses. These were patients with cirrhosis, peripheral neuritis, acute intoxication and 'incidental' illness.
3. Miscellaneous 'medical' conditions in which there was no history or stigmata of alcoholism.

Altogether 94 patients were then drawn from these 3 large groups in a non-selective manner; thus, as far as we could judge, they constituted random samples.

Fasting, early-morning sputum, free of oral saliva, was collected in a clean fat-free receptacle. Where possible, the sputum examinations were made daily and an average of 4 specimens from each of the 94 patients was examined.

The specimens were sent to the South African Institute for Medical Research with no clinical details. This was done so that there would be a perfectly objective examination. The sputum was thoroughly mixed, and one drop of sputum was added to an equal volume of Sudan IV stain and allowed to stand for 2 minutes before counting under a coverslip at a magnification of 450×. After several high-power fields had been examined, an average fat-globule count was determined, falling into one of the following ranges: 0, 1 - 2, 3 - 10, and 10 - 20.

RESULTS

A tender, enlarged liver was almost invariably present in the patients with acute alcoholic psychosis. We were unable to establish a correlation between the sputum fat-globule count and the severity of the mental picture in these patients. It was felt that embolization probably occurred phasically and that the sputum count did not therefore necessarily represent what was happening in the body at that moment in time.

It is noteworthy that in over half the 'medical' patients with no alcoholic background, small numbers of fat globules were sometimes present in the sputum. This finding bore no obvious relationship to the associated pathology, to the presence or absence of hepatomegaly, or to chest infection.

Statistical Analysis

Statistical comment on the results was made by Prof. J. E. Kerrich, Professor of Statistics at the University of the Witwatersrand, as follows: 'Certain difficulties arise in the analysis of the data. Firstly, the number of observations vary from patient to patient, depending on whether the patient had a productive cough or not and upon the duration of hospitalization. Secondly, the counts for a given patient vary from time to time, as pointed out previously. Finally, statistical evaluation would be facilitated if the globule counts were given in absolute figures rather than being expressed as falling into a numerical range.'

The data have been condensed in Table I.

TABLE I. FINDINGS IN 290 OBSERVATIONS ON FAT GLOBULES IN SPUTUM SPECIMENS

No. of fat globules	*Alcoholics*						*Non-alcoholics*	
	With psychosis		*Without psychosis*		*Total*			
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
0 ..	41	38·3	59	59·6	100	48·6	39	46·4
1–2 ..	43	40·2	33	33·3	76	36·9	40	47·6
3–10 ..	18	16·8	7	7·1	25	12·1	3	3·6
11–20 ..	5	4·7	0	0·0	5	2·4	2	2·4
Total ..	107	100·0	99	100·0	206	100·0	84	100·0

The chi-square test in the first 2 groups gives a result significant at the 0·5% level. Chi-square values are shown in Table II.

TABLE II. CHI-SQUARE VALUES

Comparison	Value of chi-square	Degrees of freedom	Significance
'With psychosis' *vs* 'without psychosis'	15·3	3	Significant at 0·5% level
'Without psychosis' *vs* 'medical controls'	3·86	2	Not significant
'With psychosis' *vs* 'all others'	15·9	3	Significant at 0·5% level

The estimates of the means and standard deviations of the fat-globule counts appear in Table III.

TABLE III. MEANS AND STANDARD DEVIATIONS OF THE FAT-GLOBULE COUNTS

Group	No. of observations	Observed mean	Observed variance	95% confidence limited for mean globule count
1. Alcoholics with psychosis	107	2·316	12·16±·337	2·32±·67
2. Alcoholics without psychosis	99	·926	2·45±·157	·93±·31
3. 'Medical' controls ..	84	1·290	6·10±·270	1·29±·54
4. Groups (2) and (3) combined	183	1·094	4·16±·151	1·09±·30

The results suggest that alcoholics with psychosis differ significantly from the other 2 groups, which do not differ significantly between themselves.

DISCUSSION

Although experimental work on intravascular fat was carried out nearly 300 years ago by Lower,[8] and later by Magendie in 1821,[9] it was Zenker in 1862[17] who observed fat emboli in a case of crush injury. The value of sputum examination in the diagnosis of fat embolization was first pointed out by Warthin in 1913.[13]

Since then, fat embolism has become accepted as a distinct clinico-pathological entity, occurring most often after trauma involving fatty tissues. In a series of 100 autopsies performed at the Birmingham Accident Hospital, pulmonary fat embolism was found in 89% of cases where it had not been suspected clinically.[12] However, fatty emboli have been reported in a wide variety of non-traumatic conditions,[2,5,11,14-16] and most recently in patients receiving high doses of corticosteroids. Such high doses favour fatty accumulation in the liver, and it has been suggested that some of the steroid encephalopathies are due to cerebral embolization from rupture of a fatty hepatic cyst.[4] The incidence of pulmonary fat emboli in unselected autopsies has been given as about 50% by Lehman and McNattin[5] and Wright,[16] and as 20% by Whiteley.[14] Our finding of fat globules in the sputa of unselected medical patients is in keeping with these pathological observations. The common occurrence of emboli in routine necropsies caused Whitson[15] to cast doubt on the importance of this finding as a cause of death. Other workers subsequently supported this view.[1,12] Scuderi[11] noted that alcoholics were apparently more predisposed than non-alcoholics to fat embolism. Hartroft and Ridout[3] commented on the presence of renal and pulmonary fat emboli in chronic alcoholics.

The clinical picture has been neatly outlined by Sevitt[12] as 'characterized by major cerebral and neurological effects, respiratory symptoms, pyrexia, tachycardia and a characteristic petechial eruption of much diagnostic importance'. A division into pulmonary and systemic forms has been made. The pulmonary form does not, however, appear to be symptomatically significant.[12]

One of the prominent features of cerebral fat embolism is the occurrence of psychotic behaviour, often resembling to a remarkable degree that of classically described alcoholic delirium tremens. The association of cough, dyspnoea, pyrexia, tachycardia and chest signs, in keeping with an acute respiratory infection, has for many years been noted to occur with the syndrome of 'delirium tremens'. This has usually been interpreted as an acute respiratory infection precipitating the mental symptoms in an alcoholic patient. Is this in fact so? Both entities have been thought to be consequent upon fat embolization to lung and brain.[6,7] More recently it has been suggested that the respiratory effects are not due to local fat emboli in the lungs and are explicable on a neurogenic basis, secondary to cerebral emboli.[12] Pulmonary symptoms were not a prominent feature in our patients with delirium tremens.

We have advisedly employed the all-embracing term 'acute alcoholic psychosis' throughout this article, since it has unhappily become a practice to label all forms of psychotic behaviour in alcoholics as 'delirium tremens'. The use of this term appears to us to imply the presence of a single, clearly-defined entity, specific to alcoholics. However, not only is the psychotic picture in alcoholics protean in its manifestations, but also the possible aetiological factors are variable and numerous, ranging from portal systemic encephalopathy and hypoglycaemia to hypovitaminosis and magnesium deficiency.

Clinical and pathological evidence is available to implicate fatty emboli in the genesis of at least some acute alcoholic psychoses.[3,6,7] Our findings tend to be in keeping with the above. It has been postulated that these emboli arise from areas of fatty degeneration in the liver.[6,7,14] The emboli may lodge in the pulmonary vascular bed and be expectorated, or may spill over into the systemic circulation, perhaps *via* the complex bronchopulmonary anastomosis of Marchand, Gilroy and Wilson.[10]

SUMMARY

A quantitative study of the fat globules in the sputum of 94 patients is presented. Random cases were taken from 3 groups — chronic alcoholics with acute psychoses, chronic alcoholics without psychoses, and 'medical' patients with no alcoholic background. The results suggest that the fat-globule count in alcoholics with psychoses differs significantly from that in the other 2 groups — which do not differ significantly between themselves.

Hepatomegaly was almost an invariable feature in those alcoholics with psychotic behaviour. Our findings in the patients with alcoholic psychosis support the concept of earlier workers that cerebral fat emboli, probably arising in the damaged liver, play a role in the genesis of the syndrome of 'delirium tremens'. The pathogenesis of this phenomenon of fat embolization is as yet poorly understood and further work in this field would seem to be indicated.

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