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Alcohol consumption in arthritic patients: clinical and laboratory studies

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SUMMARY In popular belief patients with chronic arthritis take alcohol for its analgesic effect. To test this we studied by validated questionnaire the past and present alcohol consumption of 103 patients with primary osteoarthritis of the hip (OA), 95 patients with rheumatoid arthritis (RA), and 90 orthopaedic non-arthritic controls. OA men were most likely and RA men least likely to have been heavy drinkers at any time of their lives. Mean red corpuscular volume (MCV), γ-glutamyltransferase (GGT), and serum uric acid (SUA) levels did not correlate with reported alcohol consumption. Two of 93 OA femoral heads examined had avascular change; both were from heavy drinkers. The abstemiousness of RA men compared with their OA counterparts was due to a striking increase in joint pain after drinking alcohol ($p=0{\cdot}004$), fear of adverse drug reactions with alcohol, and a widespread belief not expressed by OA men that 'alcohol and arthritis do not mix'.

Key words: rheumatoid arthritis, osteoarthritis, osteonecrosis, alcohol drinking.

Alcohol consumption has almost doubled in the United Kingdom over the past twenty years.[1] A growing national awareness of alcohol-related problems has followed,[2 3] but there are few studies of alcohol consumption in specific hospital inpatient groups.[4–6] 20% to 27% of inpatients,[5 6] and 32% of casualty department attenders[7] in Britain have been found to have medical or social problems related to drinking. Patterns of alcohol consumption in chronic arthritics before and after the development of arthritis are not known. Knowledge of these patterns would add to the psychological profile of arthritic patients[8] and would extend the information on the drinking habits of a major group of patients with chronic disease.[9]

Excessive alcohol consumption has been causally linked with gout,[10 11] pancreatitis-associated arthritis,[12] and 'spontaneous' avascular necrosis of the femoral head (SANFH).[13–16] The latter link remains unproved but strongly suspected because SANFH is reported most frequently from countries with a high alcohol consumption per person, such as the USA[17] and France.[18] However, its true frequency may be greater, since by the time patients seek medical attention there may be advanced femoral head destruction with secondary osteoarthritis radiologically indistinguishable from other causes of femoral head damage.[19–21]

We have compared lifetime alcohol consumption in patients with rheumatoid arthritis (RA), in patients with severe symptomatic hip degeneration (OA), and in controls without symptomatic arthritis. Further, we attempted to confirm reports that mean red corpuscular volume (MCV), serum γ-glutamyltransferase (GGT), and serum uric acid (SUA) can be correlated with admitted alcohol consumption.[22 23]

Patients and methods

We studied unselected patients over the age of 40 admitted under five consultants to the Nuffield Orthopaedic Centre in the 16-month period from September 1981 to February 1983. The OA group comprised 103 patients (52 male) admitted for total hip replacement for 'primary' degenerative hip disease. Those with hip degeneration secondary to well-documented past or present disease such as congenital hip dislocation, femoral neck fractures, inflammatory polyarthritis, and Perthes' disease

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were excluded. The RA group comprised 95 patients (48 male) with definite or classical rheumatoid arthritis[24] admitted to the rheumatology unit for both medical and surgical therapy. The control group comprised 90 patients (46 male) admitted for minor orthopaedic surgery, usually on hands or feet, who did not fit into the OA or RA groups. In an attempt to define the control group more clearly patients with Dupuytren's contracture, which is claimed to be alcohol related,[25] symptomatic arthritis of large weight-bearing joints, inflammatory joint disease, or painful conditions causing chronic disability (such as low backache) were excluded from this group. Any patient unwilling or unable to answer the questionnaire was excluded from the study.

QUESTIONNAIRE

Each patient answered a structured questionnaire which was completed by an interviewer (AB) who was aware of the patient's diagnosis. The questionnaire was based on the QF categorisation of Griffiths Edwards.[26] Details of day by day alcohol consumption during a 'typical' recent week were recorded and used as a basis for comparison with weekly consumption 1, 10, and 20 to 30 years previously with life events such as marriage and military service as 'anchor points' to help recollection ('time-line follow-back method').[27] Drinking before the age of 30 was disregarded because we felt it would be unrepresentative of patterns in future years. Weekly consumption was scored 1–9 (Table 1) and the scores awarded were used for comparisons at the end of the study.

In addition to establishing alcohol consumption the questionnaire sought information about whether alcohol use made arthritic pain better, worse, or left it unchanged. Patients were also asked whether the development of arthritis had affected the frequency with which they went out to consume alcohol at public houses or clubs.

Seventeen patients were retested by the same interviewer at intervals of 2 weeks to 6 months after the original questionnaire. Seven of these patients were also requestioned by other interviewers. In all cases the coefficient of correlation (r) between scores was 0·88 or better ($p<0·001$); however, questions concerning consumption 20 or 30 years previously were less well validated ($r=0·6$, $p<0·05$).

Table 1 *Alcohol consumption scores: QF categorisation**

1. Nil or occasional	Less than 1 drink per month
2. Infrequent light	Less than 4 drinks per month
3. Frequent light	Less than 4 drinks per week Drinking on 2 or fewer days every week
4. Steady	5 to 21 drinks per week Drinking on 3 to 5 days per week
5. Steady daily drinker	As (4) but admits to drinking 7 days per week
6. Threshold	28 to 56 drinks per week Drinking on 4 or fewer days weekly
7. Heavy	28 to 56 drinks per week Daily consumption
8. Very heavy	60 to 100 drinks per week
9. Gross	More than 100 drinks per week

'Drink'=½ pint (0·3 l) beer or lager=standard measure of spirits=glass of wine or sherry (small measure)=8–10 g alcohol.
*Modified from reference 26.

BIOCHEMICAL AND HAEMATOLOGICAL INDICES OF ALCOHOL CONSUMPTION

MCV, SUA, and serum GGT were measured in all patients on admission to hospital. Venesection was performed between 9 and 10 am without preceding restriction of food or fluid intake. SUA was measured by the uricase method and GGT was measured at a later date on a batch of sera stored at −15°C by a modification of the method of Orlowski and Meister.[28] MCV was measured by automated counter (Coulter Model S Plus).

HISTOLOGY

Examination of resected femoral heads for avascular necrosis was performed in 93 of the 103 OA patients. The pathologist was not aware of alcohol scores in any patients but was aware of our interest in the presence or absence of avascular necrosis in the resected femoral heads.

STATISTICS

Correlation between alcohol score, SUA, MCV and GGT was assessed by linear regression analysis. In other comparisons unpaired *t* tests and the Mann–Whitney U test were used for continuous variables. The χ^2 test with Yates's correction and Fisher's exact test were used for discrete variables. Product-moment correlation coefficients were used where relevant.

Results

Two hundred and ninety patients were approached to take part in the study. Only two refused. Age and disease duration in the different patient groups are detailed in Table 2. Among the men the controls were significantly younger than OA patients ($p<0·05$), while RA disease duration was significantly longer than that of OA ($p<0·01$). Female OA patients were significantly older than both RA patients ($p<0·02$) and controls ($p<0·03$). Disease duration was not significantly longer in female patients with RA than in those with OA.

Table 2 *Patient characteristics*

	Age (years)		*Disease duration (years)*	
	Mean	*SD*	*Mean*	*SD*
OA men (n=52)	63·6	9·7	8·1	7·1
RA men (n=48)	62·2	9·4	11·5	7·1§
Control (n=44)	59·8	8·4*		
OA women (n=51)	68·2	7·7†	8·3	10·8
RA women (n=47)	63·4	9·3	12·4	10·5
Control (n=44)	64·3	9·2‡		

*p<0·05 compared with OA men.
†p<0·02 compared with RA women.
‡p<0·03 compared with OA women.
§p<0·01 compared with OA men.
Two controls did not have their age recorded.

Table 3 *Male drinking patterns: top three score groups (7–9) only*

	OA	*RA*	*Control*
n	52	48	46
Max 7–9	21	9*	13
Current 7–9	9	2	1*
Increase since joint disease	4	6	
Alcohol relieves arthritic pain	6	8	
Alcohol worsens arthritic pain	0	8 (+3 abstained)	

*p<0·05 compared with OA men.

Table 4 *Female drinking patterns: score groups 5–9 only*

	OA	*RA*	*Control*
n	51	47	44
Max 5–9	8	11	10
Current 5–9	5	6	6
Increase since joint disease	4	9	
Alcohol relieves arthritic pain	2	0	
Alcohol worsens arthritic pain	1 (+1 abstained)	3 (+1 abstained)	

Drinking patterns are detailed in Tables 3 and 4. Maximal lifetime consumption is defined as the maximum weekly consumption score for more than 5 years of a patient's life over the age of 30, i.e., the highest sustained drinking score in a patient's adult life. OA men are overrepresented in the top-scoring groups 7 to 9, but at current consumption scores the difference is significant only compared with the control group and at maximal lifetime scores with the RA group. Similar numbers of OA and RA patients had increased alcohol consumption after the development of arthritis and claimed relief of arthritic pain from alcohol. A striking difference was that eight RA men curtailed consumption because their joints were worse even after small amounts of alcohol, whereas no OA men had done so (p=0·004). Another three RA men claimed to have cut alcohol consumption completely as a direct result of arthritis or its treatment. There were no significant differences between drinking patterns in the female groups, though three RA women felt worsening of joint pain after alcohol compared with only one OA woman. The low admitted consumption among women made comparisons difficult and caused us to choose a lower threshold score of five when defining the 'top-scoring' group.

Table 5 *Comparison of heaviest and lightest drinkers (significant comparisons only)*

Age—OA men (years)			
Maximal lifetime consumption			
Score (7–9)	n=21	Mean 58·3 (SD 8·7)	
Score (1–3)	n=13	Mean 69·8 (SD 7·4)	p<0·001 *t* test
Current consumption			
Score (7–9)	n= 9	Mean 57·2 (SD 9)	
Score (1–3)	n=28	Mean 66·8 (SD 8·8)	p<0·01 *t* test
GGT—RA women (IU/l)			
Maximal lifetime consumption			
Score (5–9)	n= 9	Mean 18·0 (SD 9)	
Score (1–2)	n=18	Mean 37·3 (SD 25)	p<0·01 *t* test
MCV—RA women (fl)			
Current consumption			
Score (5–9)	n= 6	Mean 88·5 (SD 7·6)	
Score (1–2)	n=26	Mean 81·9 (SD 6·9)	p<0·05 *t* test

Table 6 *Mean values of SUA, MCV and GGT in patients and controls with regression values for correlations of these variables with alcohol consumption scores*

Men	*OA*	*RA*	*Control*
SUA			
(μmol/l mean ±SE)	371 (±12·8)	335 (±21·7)	368·1 (±14·5)
	r=0·107	r=0·274	r=0·219
MCV			
(fl mean ±SE)	88·3 (±0·64)	84·1 (±1·15)*	88·5 (±0·96)
	r=0·153	r=0·137	r=0·082
GGT			
(IU/l mean ±SE)	35·5 (±4·5)	56·9 (±10·9)†	28·3 (±3·3)
	r=0·021	r=0·056	r=0·233
Women			
SUA			
(μmol/l mean ±SE)	303 (±12·5)	313 (±21·4)	305 (±13·7)
	r=0·253	r=0·006	r=0·068
MCV			
(fl mean ±SE)	88·8 (±0·58)	82·7 (±1·04)‡	88·3 (±0·56)
	r=0·243	r=0·256	r=0·05
GGT			
(IU/l mean ±SE)	39·8 (±7·3)	33·8 (±4·8)	30·2 (±5·4)
	r=0·041	r=0·201	r=0·058

None of the r values was statistically significant.
*p<0·01 compared with OA and control.
†p<0·05 compared with control.
‡p<0·001 compared with OA and control.
SI conversion: SUA μmol/l×0·01681=mg/l.

HISTOLOGY

Avascular change was found in two of the 93 femoral heads examined. Both patients were men with current alcohol consumption scores of 7. One had a maximal lifetime score of 9.

CHARACTERISTICS OF HEAVIEST AND LIGHTEST DRINKERS (TABLE 5)

SUA, MCV, GGT, and age were compared in the heaviest (score men 7–9, women 5–9) and lightest (score men 1–3, women 1 and 2) drinkers within diagnostic groups. Age differences in the male OA group were most significant. At *current* consumption levels the heaviest drinkers tended to be much younger than the lightest ($p<0·01$, *t* test). For *maximal* lifetime consumption scores the differences were even more significant ($p<0·001$). These differences were not found in OA women or in RA or control men or women. Only in RA women were there significant differences in MCV (at current alcohol score) and GGT (at maximal lifetime score) between heaviest and lightest drinkers. In contradiction with expected results, GGT levels were higher in the lightest-drinking RA women than in the heaviest.

EFFECT OF ARTHRITIS ON PLACE OF ALCOHOL CONSUMPTION

Twelve RA and four OA patients admitted that their joint disease had influenced the frequency with which they went out to consume alcohol at pubs, clubs, or other peoples homes; the difference was not significant.

CORRELATION BETWEEN ALCOHOL CONSUMPTION SCORE, MCV, GGT, AND SUA (TABLE 6)

There was no correlation between any of these variables and alcohol consumption score. Mean MCV was significantly lower in both male and female RA groups than in OA and control groups (probably due to a combination of iron deficiency and 'the anaemia of chronic disorders').[29] Mean GGT was higher in RA males than in other male groups.

Discussion

An important feature of the study is the low admitted consumption in all female diagnostic groups. This makes attempts to relate consumption

to disease difficult, and our main conclusions therefore apply to male patients only.

It was not possible for us to study a totally unselected population because all patients submitted for surgery were, by definition, selected by virtue of fitness for operation. Arguably our results underestimate drinking among orthopaedic patients because some of the heaviest alcohol consumers may have been rendered unfit for operation or even have died as a result of alcohol abuse. We used total hip replacement as the criterion for inclusion of patients with degenerative hip disease because this operation represents a defined end-point of disease severity.

Our questionnaire attempted simply to establish alcohol consumption in the patients studied. For ethical and pragmatic reasons we deliberately avoided the use of CAGE[30] or MAST[31] questionnaires which are designed to detect 'problem drinking'. We felt that in our population of mostly elderly, often disabled patients these questions might well give offence, leading to an unacceptably high drop-out rate.[32 33] In any event 'problem drinking' and 'heavy drinking' are often not synonymous.[34] This caution was justified by the extremely low questionnaire refusal rate.

Previous studies[4 6] have confirmed that patients admitted for elective orthopaedic surgery tend to be among the heaviest-drinking hospital inpatients, but these studies have not distinguished between patients with different conditions requiring orthopaedic surgery. From our results no consistent pattern emerges, but it appears that male patients undergoing hip replacement surgery for primary hip degeneration admit to being heavier drinkers than do men with rheumatoid arthritis or minor orthopaedic conditions. Apart from the two cases of histologically proved avascular necrosis of the femoral head, both in self-confessed heavy drinkers, we found no evidence that heavy alcohol consumption led to hip destruction. However, histological sections of the femoral head may have missed avascular segments which could anyway have become unrecognisable in the presence of advanced degenerative change. Additionally, the relative youth of the heaviest-drinking OA men compared with the lightest drinkers suggests that heavy alcohol consumption is at least a part of a life style predisposing to hip degeneration.[35]

Men with rheumatoid arthritis seem from our study to regard themselves as lifelong light drinkers compared with men with severe OA hip and other orthopaedic conditions. The reasons for this are complex but may include a relative amnesia for habits in the prearthritic period due to profound life style alterations following the development of arthritis. However, a number of RA patients felt that drinking alcohol made their joint pain worse. This phenomenon was not associated in our survey with consumption of any particular alcoholic beverage. Some RA patients voiced concern about possible harmful interactions between their anti-arthritic drugs and alcohol, while others felt that their arthritis precluded 'frivolous activities', such as social drinking. This phenomenon has been noted in other groups of patients with chronic diseases.[9] Possibly some RA patients tended to understate their consumption because of fear of 'medical disapproval' of alcohol consumption with the drugs they were taking. Considering these factors together, it is clear from our study that significant numbers of RA men feel that 'alcohol and arthritis do not mix'.

Another striking feature of this study is the poor correlation between alcohol score and MCV, SUA, and GGT in all diagnostic groups of both sexes. There have been few studies attempting to correlate reported alcohol consumption with biochemical and haematological indices in patients not known to be alcoholic.[36 37] Results have been conflicting; some studies report modest correlation between the reported alcohol consumption and GGT[37 38] while in others there is apparently a better correlation.[22 23] The pattern for GGT in our cases is anyway confused because of the reported elevation of this enzyme in RA patients.[39–42] Similar controversy surrounds the value of MCV as a screening test. Some authors are impressed by a linear relationship between daily alcohol consumption and MCV;[22] others emphasise the insensitivity of MCV elevation for detecting alcoholism.[38 43] The poor ability in our study of MCV, SUA, and GGT to discriminate between heaviest and lightest drinkers emphasises that these tests are no substitute for careful questioning and a clinical impression in assessing alcohol consumption. The low consumption of alcohol per head in the UK compared with most other European countries[44] accounts for the weakness of these tests in detecting heavy alcohol consumption.[45] GGT has been shown to have only 30% sensitivity as a detector of alcohol abuse in validation studies on known alcoholics and MCV even less;[46] clearly they have not been helpful in our study of a group of patients who were not presumed alcoholic.

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