Clinical profile and prognostic factors of alcoholic cardiomyopathy in tribal and non-tribal population

ABSTRACT

Objective Alcoholic cardiomyopathy (ACM) is a leading cause of non-ischaemic dilated cardiomyopathy (DCM) in tribal and non-tribal population. However, no study has been done depicting the correlation between clinical profile and prognosis of ACM in tribal and non-tribal population. This study also defines the long-term outcome and prognostic markers of ACM.

Methods We studied 290 patients with ACM who were evaluated in our institute between January 2013 and December 2016. The primary endpoint of the study was all-cause mortality. Statistical analysis was done by using Kaplan-Meier survival curves for the assessment of all-cause mortality and Cox regression for the assessment of risk factors.

Results After a median follow-up period of 3.75 years (IQR: 3–4 years), 50 patients with ACM (37.3%) died among tribal population while 14 patients (9%) died among non-tribal population. Independent predictors of all-cause mortality in ACM identified by Cox regression were left ventricular ejection fraction (LVEF) (HR: 0.883; 95% CI 0.783 to 0.996; p=0.043), QRS duration (HR: 1.010; 95% CI 1.007 to 1.017; p=0.005) and Child-Turcotte-Pugh (CTP) Scoring (HR: 12.332; 95% CI 6.999 to 21.728; p<0.001) at admission. The Kaplan-Meier survival probability estimate was 95.1% at 1 year and all-cause mortality was found to be higher in patients with QRS>120 ms, LVEF ≤35%, CTP Grade B/C than patients with QRS≤120 ms, LVEF >35% and CTP Score A, respectively (log-rank χ²=55.088, p<0.001; log-rank χ²=32.953, p<0.001; log-rank χ²=139.764, p<0.001, respectively).

Conclusion Our study indicated increased morbidity and mortality in tribal population. LVEF, QRS duration and CTP Scoring at the time of presentation were found to be the independent prognostic markers of patients with ACM.

INTRODUCTION

Alcoholic cardiomyopathy (ACM) is a condition of toxic origin that causes gradual changes in the structure and function of the heart, resembling those seen in idiopathic dilated cardiomyopathy (DCM). Individuals with alcohol consumption of more than 80 g per day over a period of at least 5 years are at risk of developing ACM and heart failure (HF). Developing countries like India experience more problems with alcohol abuse than developed countries, despite equal amounts of drinking.

A disproportionate level of alcohol misuse has been reported among indigenous people all over the world, and the burden of diseases associated with alcohol misuse is almost double among them. In India, indigenous people are known as scheduled tribes, or Adivasis, and live as a group with distinctive social, cultural, historical and geographical

Key questions

What is already known about this subject?

► The literature suggests alcoholic cardiomyopathy as a leading cause of non-ischaemic dilated cardiomyopathy. However, there is a major paucity of data comparing clinical profile of alcoholic cardiomyopathy in tribal and non-tribal population.

► Many studies have discussed various prognostic markers of alcoholic cardiomyopathy, Child-Turcotte-Pugh (CTP) Scoring has been used as a prognostic marker in cirrhotic cardiomyopathy. However, none of the study has used CTP Scoring in alcoholic cardiomyopathy.

What does this study add?

► This study provides a deep insight of clinical profile of alcoholic cardiomyopathy with a special consideration of tribal population.

► This study deals with a detailed assessment of various prognostic markers of alcoholic cardiomyopathy. We have introduced CTP Score as a prognostic marker which has never been used before in any study.

How might this impact on clinical practice?

► This study provides a detailed clinical picture of alcoholic cardiomyopathy and by using basic tools like CTP Score, clinicians can make a rapid assessment of patients.

► This study will create an insight among primary caregivers, internists, physicians and cardiologists, helping them to make better decisions and thus leading to decreased morbidity and mortality of patients.
circumstances. Evidence from India also supports a high prevalence of alcohol consumption among indigenous tribes, which is associated with a wide range of health problems, high morbidity and early mortality. The data derived from National Family Health Survey suggest alcohol use among 26% of the indigenous population, while the rate was just 9% among the non-indigenous population.

Excess alcohol consumption has been implicated in up to 40% of cases of DCM. ACM just like other causes of DCM is characterised by a reduced left ventricular ejection fraction (LVEF) and an increased left ventricular end diastolic diameter (LVEDD); however, no specific clinical or histological features have been defined for ACM and the diagnosis is usually made by exclusion in a patient with a long history of heavy alcohol abuse.

In spite of the fact that alcohol plays a major role in the causation of DCM, very few studies have been published on the long-term outcome of patients with ACM in India and there is a major paucity of data comparing ACM in tribal and non-tribal population. This study was undertaken to find out the long-term outcome and prognostic markers of ACM and to compare the different characteristics of the patients between the death and survival groups in tribal and non-tribal population, simultaneously.

MATERIALS AND METHODS
Study design and population
From January 2013 to December 2016, we collected data of 290 patients with ACM referred for evaluation to the Department of Internal Medicine and Department of Cardiology in our institute RIMS, Ranchi. This study was a retrospective, observational study and a diagnosis of ACM was made in accordance to the definition provided in the European Society of Cardiology consensus document on the classification of cardiomyopathies.

A diagnosis of ACM was made if the consumption of alcohol was more than 80 g per day over a duration of at least 5 years, and this alcohol abuse must have been maintained until <3 months before establishing the diagnosis of DCM.

Patients having coronary heart disease, rheumatic heart disease, diabetes, hypertension, thyrotoxic heart disease, left ventricle non-compaction, moderate to severe chronic anaemia (haematoglobin <60 g/L), peri-partum cardiomyopathy, systemic immune disease and congenital heart disease were excluded from this study.

Initial assessment of all patients included physical examination, blood tests, 12-lead electrocardiography (ECG) and 2D echocardiography. Additional studies including 24 hours ECG monitoring, coronary angiography and coronary artery CT were performed to rule out coronary heart disease.

Child-Turcotte-Pugh (CTP) scores were calculated on the basis of laboratory values obtained within 24 hours of admission. The CTP Score included two continuous variables (bilirubin and albumin) and three discrete variables (ascites, encephalopathy and international normalised ratio). This score was divided into three classes: class A (5–6), class B (7–9) and class C (10–15).

Indirect patient and public involvement
We did not directly include patient and public involvement (PPI) in this study, but the database used in the study was developed with PPI and is updated by a committee that includes patient representatives.

Follow-up
We did the follow-up until December 2019. Follow-up was done by outpatient department visit, telephonic conversation or hospital admission. All-cause mortality was the only endpoint of this study.

Statistical analysis
Categorical data were expressed as percentages and groups were compared using the chi-square (χ²) test. Student’s t test was applied to compare continuous data of two groups. Normally distributed variables were presented as the means and SD, whereas non-normally distributed variables were expressed as the medians and IQRs.

In order to identify independent predictors of all-cause mortality from baseline variables, an initial screening of all parameter values was done by univariate Cox regression at enrolment. Variables with a p<0.05 in univariate Cox regression were taken further for the forward/backward stepwise multivariable Cox proportional hazards analysis.

Continuous variables were categorised on the basis of multivariable Cox regression analysis, Kaplan-Meier method was used to calculate the survival curves, and the groups were compared using log rank test. The level of statistical significance was p<0.05. All hypothesis tests were two-tailed. SPSS software V.16.0 was used to perform the analysis.

RESULTS
Clinical profile
A total of 290 hospitalised patients with ACM participated in the study. Of the 290 patients, 134 (46.2%) belonged to tribal population and 156 (53.8%) belonged to non-tribal population. During the median follow-up period of 3.75 (IQR: 3–4) years, 50 patients with ACM (37.3%) died among tribal population while 14 patients (9%) died among non-tribal population. The clinical, ECG and echocardiographic characteristics of the patients with ACM are depicted in table 1.

Among tribal population, no differences between the survival and death groups were observed at the baseline in terms of age, sex, chest pain, basal crepts, orthopnoea, PND (paroxysmal nocturnal dyspnoea), JVP (jugular venous pressure), oedema and New York Heart Association (NYHA) classification. The frequencies of atrial fibrillation (AF), atrioventricular block (AVB) and CTP (Score B, C) were higher in the death group and sinus.
### Table 1 Clinical, electrocardiographic and echocardiographic characteristics of patients tabulated according to survival and death in tribal and non-tribal population

|                  | Tribal n=134 |                  | Non-tribal n=156 |                  | Tribal vs non-tribal |
|------------------|--------------|------------------|-------------------|------------------|----------------------|
|                  | All patients | Death (n=50)     | Alive (n=84)      | P value          | All patients         | Death (n=14) | Alive (n=142) | P value | P value |
| Age (years)      | 52.6±21.9    | 43.9±24.2        | 57.8±18.8         | 0.544            | 53.6±16.1            | 37±17.8     | 55.1±15       | 0.765   | 0.674   |
| Sex              |              |                  |                   |                  |                      |              |               |        |
| Male             | 89 (66.4%)   | 29               | 60                | 0.111            | 75 (48.1%)           | 8           | 67            | 0.477   | 0.587   |
| Female           | 45 (33.6%)   | 21               | 24                |                   | 81 (51.9%)           | 6           | 75            |         |
| Chest pain       |              |                  |                   |                  |                      |              |               |        |
| Present          | 86 (64.2%)   | 34               | 52                | 0.477            | 54 (34.6%)           | 9           | 45            | 0.453   |         |
| Absent           | 48 (35.8%)   | 16               | 32                |                   | 102 (65.4%)          | 5           | 97            |         |
| Basal crepts     |              |                  |                   |                  |                      |              |               |        |
| Present          | 132 (98.5%)  | 50               | 82                | 0.272            | 137 (87.8%)          | 14          | 123           | 0.144   |         |
| Absent           | 2 (1.5%)     | 2                |                   |                   | 19 (12.2%)           | 0           | 19            |         |
| Orthopnoea       |              |                  |                   |                  |                      |              |               |        |
| Present          | 90 (67.2%)   | 38               | 52                | 0.127            | 58 (37.2%)           | 12          | 46            | 0.587   |         |
| Absent           | 44 (32.8%)   | 12               | 32                |                   | 98 (62.8%)           | 2           | 96            |         |
| PND              |              |                  |                   |                  |                      |              |               |        |
| Present          | 91 (67.9%)   | 40               | 51                | 0.061            | 76 (48.7%)           | 11          | 65            | 0.19    |         |
| Absent           | 43 (32.1%)   | 10               | 33                |                   | 80 (51.3%)           | 3           | 77            |         |
| JVP              |              |                  |                   |                  |                      |              |               |        |
| Present          | 112 (83.6%)  | 35               | 77                | 0.071            | 113 (72.4%)          | 10          | 103           | 0.93    | 0.023   |
| Absent           | 22 (16.4%)   | 15               | 7                 |                   | 43 (27.6%)           | 4           | 39            |         |
| Oedema           |              |                  |                   |                  |                      |              |               |        |
| Present          | 107 (79.9%)  | 42               | 65                | 0.356            | 90 (57.7%)           | 11          | 79            | 0.097   |         |
| Absent           | 27 (20.1%)   | 8                | 19                |                   | 66 (42.3%)           | 3           | 63            |         |
| NYHA             |              |                  |                   |                  |                      |              |               |        |
| I                | 1 (0.7%)     | 0                | 1 (1.2%)          | 0.107            | 13 (8.3%)            | 0           | 13 (9.2%)     | 0.001   |         |
| II               | 8 (6%)       | 0                | 8 (9.5%)          | 0.001            | 44 (28.2%)           | 0           | 44 (30.9%)    |         |
| III              | 31 (23.2%)   | 11 (22%)         | 20 (23.9%)        | 0.36             | 56 (35.9%)           | 4 (28.6%)   | 52 (36.6%)    |         |
| IV               | 94 (70.1%)   | 39 (78%)         | 55 (65.4%)        | 0.001            | 43 (27.6%)           | 10 (71.4%)  | 33 (23.3%)    |         |

Continued...
Tribal Non-tribal Tribal vs non-tribal

|                      | All patients | Death (n=50) | Alive (n=84) | P value | All patients | Death (n=14) | Alive (n=142) | P value | P value |
|----------------------|--------------|--------------|--------------|---------|--------------|--------------|---------------|---------|---------|
| **CTP Scoring**      |              |              |              |         |              |              |               |         |         |
| A                    |              |              |              |         |              |              |               |         |         |
|                      | 50 (37.3%)   | 0            | 50           | <0.001  | 125 (80.1%)  | 0            | 125           |         | <0.001  |
| B                    | 56 (41.8%)   | 22           | 34           |         | 23 (14.7%)   | 6            | 17            | <0.001  |         |
| C                    | 28 (20.9%)   | 28           | 0            |         | 8 (5.2%)     | 8            | 0             |         |         |
| **ECG**              |              |              |              | <0.001  |              |              |               |         |         |
| Sinus rhythm (%)     | 62           | 66           | 76           | 0.04    | 66           | 60           | 70            | 0.039   |         |
| AF (%)               | 38           | 34           | 24           | 0.03    | 34           | 40           | 30            | 0.027   |         |
| AVB (%)              | 24           | 20           | 16           | 0.01    | 22           | 23           | 17            | 0.034   |         |
| QRS (ms)             | 120.5±31.3   | 130.1±32.9   | 112.8±31.8   | <0.001  | 118.5±30.3   | 133.1±33.9   | 110.4±28.8    | <0.001  |         |
| **Echocardiographic finding** | | | | | | | | | |
| LVEF (%)             | 30.6±10.8    | 23.8±4.5     | 29.3±5.7     | <0.001  | 33.4±10.8    | 23.2±5.8     | 35.9±6.6      | <0.001  | <0.001  |
| LVESD (cm)           | 4.9±0.7      | 5.3±0.3      | 5±0.6        | 0.001   | 4.1±0.4      | 5.3±0.4      | 4.3±0.6       | <0.001  | <0.001  |
| LVEDD (cm)           | 6.0±0.8      | 6.4±0.4      | 6.1±0.6      | 0.006   | 5.8±0.4      | 6.4±0.4      | 5.9±0.3       | <0.001  | <0.001  |
| Mortality (%)        | 50 (37.3%)   | 84 (62.7)    | 14 (9)       |         | 142 (91)     |             |               |         |

Data were expressed as mean±SD or as percentages. P values were calculated from independent samples t tests or χ² tests for categorical data. Bold data indicate p<0.05.

AF, atrial fibrillation; AVB, atrioventricular block; CTP, Child-Turcotte-Pugh; JVP, jugular venous pressure; LVEDD, left ventricular end diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; NYHA, New York Heart Association; PND, paroxysmal nocturnal dyspnoea.
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rhythm was observed to be more in the survival group. The QRS duration, LVESD (left ventricular end-systolic dimension) and LVEDD were higher in the death group but the LVEF was lower in the patients of death group than those patients in the in the survival group.

Among non-tribal population, no differences in the death and survival groups were observed at the baseline in terms of age, sex, chest pain, basal crepts, orthopnoea, PND, JVP and oedema. The frequencies of CTP (Grade B, C), NYHA (class III/IV) classification, AF and AVB were higher in the death group and sinus rhythm was observed to be more in the patients of survival group. The QRS duration, LVESD and LVEDD were higher in the death group and LVEF was observed to be lower in the patients of death group than those in the survival group.

On comparing tribal with non-tribal population, no differences were observed at baseline in terms of age and sex. The frequencies of chest pain, basal crepts, orthopnoea, PND, JVP, oedema, NYHA (class III/IV) classification, CTP (Score B, C), AF and AVB were higher in tribal population but sinus rhythm was more in non-tribal population. The QRS duration, LVEDD and LVESD were higher but the LVEF was lower in the tribal population.

Prognostic factors assessment in ACM

Univariate analysis of the data suggested that ethnicity of belonging to tribal population, chest pain, orthopnoea, PND, NYHA, CTP Scoring, LVEF, LVEDD and LVESD were the prognostic predictors of ACM. Multiple Cox regression analysis revealed LVEF (HR: 0.883; 95% CI 0.783 to 0.996; p=0.043), QRS (HR: 1.010; 95% CI 1.007 to 1.017; p=0.005) and CTP Scoring (HR: 12.332; 95% CI 6.999 to 21.728; p<0.001) as the independent predictors of all-cause mortality at admission (table 2).

Correlation between independent predictors and all-cause mortality

The Kaplan-Meier survival probability estimate was 95.1% at 1 year. According to the clinical experience, the optimal cut-off value was taken as 120 ms for QRS duration and 35% for LVEF. The endpoint was reached by 130 patients with a QRS>120 ms and 160 patients with a QR≤120 ms; 204 patients with LVEF ≤35% and 86 patients with LVEF >35% and 115 patients classified as CTP Score B/C and 175 patients classified as CTP Score A. In our study, patients with QR≤120 ms, LVEF ≤35% and CTP Grade B/C had higher occurrence of all-cause mortality as compared with patients with a QR≤120 ms, LVEF >35% and CTP Score A, respectively (log-rank χ²=55.088, p<0.001; log-rank χ²=32.953, p<0.001; log-rank χ²=139.764, p<0.001; figure 1A–C).

DISCUSSION

As per our research, this is the first ever study conducted to compare ACM in tribal and non-tribal population. The study findings point towards clinically comparable groups. Patients from both the tribal and non-tribal groups had a similar age pattern and sex distribution. However, the groups differ on various sociodemographic characteristics. Non-tribals were better educated, from urban areas, involved in business or a professional job and were from

| Variable | Univariate analysis | Multivariate analysis |
|----------|---------------------|-----------------------|
|          | HR                  | 95% CI                | P value | HR                  | 95% CI                | P value |
| Age      | 0.965               | 0.952 to 0.978        | 0.673   | 1.002               | 0.979 to 1.025        | 0.867   |
| Sex      | 0.928               | 0.565 to 1.524        | 0.768   |                     |                       |         |
| Ethnicity| 0.209               | 0.116 to 0.379        | <0.001  | 0.704               | 0.371 to 1.334        | 0.282   |
| Chest pain | 2.282              | 1.354 to 3.846        | 0.002   | 0.325               | 0.164 to 0.641        | 0.547   |
| Basal crepts | 2.832              | 1.428 to 3.217        | 0.123   |                     |                       |         |
| Orthopnoea | 4.087              | 2.215 to 7.541        | <0.001  | 0.732               | 0.732 to 0.327        | 0.448   |
| PND      | 0.315               | 0.171 to 0.579        | <0.001  |                     |                       |         |
| NYHA     | 3.417               | 2.109 to 5.537        | <0.001  | 1.208               | 0.564 to 2.586        | 0.627   |
| CTP Scoring | 14.925             | 9.537 to 23.356       | <0.001  | 12.332              | 6.999 to 21.728       | <0.001  |
| QRS      | 1.008               | 1.006 to 1.015        | 0.003   | 1.010               | 1.007 to 1.017        | 0.005   |
| JVP      | 0.638               | 0.373 to 1.090        | 0.1     |                     |                       |         |
| LVEF     | 0.787               | 0.746 to 0.830        | <0.001  | 0.883               | 0.783 to 0.996        | 0.043   |
| LVESD    | 7.137               | 3.820 to 13.334       | <0.001  | 2.259               | 0.469 to 10.890       | 0.31    |
| P value  | 6.562               | 3.599 to 11.965       | <0.001  | 0.485               | 0.091 to 2.575        | 0.395   |

PND was not entered into multiple analysis because of its significant collinearity with orthopnoea (r=0.645, p<0.001). Bold data indicate p<0.05.

CTP, Child-Turcotte-Pugh; JVP, jugular venous pressure; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic dimension; NYHA, New York Heart Association; PND, paroxysmal nocturnal dyspnoea; QRS, QRS duration.

Table 2 Univariate and multivariate analysis showing the independent predictors of all-cause mortality

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the middle economic status, whereas tribals are less educated, many of them were from rural areas and most were involved in agricultural activities, with a higher proportion of patients coming from the lower economic status. This reflects the prevailing deprived condition of the tribals.\textsuperscript{19} Due to all mentioned factors, tribals tend to seek hospital facility very late and they seek medical advice only when their symptoms flare up, as reflected in our study that the incidence of chest pain, basal crepts, orthopnoea, PND, oedema, raised JVP, NYHA grade III, IV and CTP Score B, C were significantly higher in tribal group. Even investigations revealed higher incidences of AF, AVB, increased QRS duration, reduced LVEF, increased LVESD and LVEDD in tribal group. All these findings point towards the fact that tribals are already in advanced stage of ACM by the time they seek medical advice, which ultimately leads to high mortality as substantiated in our study.

Our study indicated that the variables determining the prognosis of ACM were LVEF (%), QRS duration, CTP Scoring at admission. Various ACM studies have suggested other prognostic factors; however, those studies were having a drawback of small sample size and had conflicting conclusions. The first paper that determined the prognostic factors of ACM was published by McDonald\textit{ et al} and they found that the only factors responsible for poor outcome were excessive consumption of alcohol and the duration of HF symptoms before admission.\textsuperscript{20} Other published articles revealed that alcohol abstinence and an increased left ventricular end-systolic diameter were the only independent poor prognostic markers of ACM.\textsuperscript{3,12}

It is a known fact that QRS duration increases with age,\textsuperscript{21} but it should be kept in mind that QRS duration increases in organic heart disease due to bundle branch blocks and the prolonged QRS on ECG has been used traditionally as a prognostic marker in a wide variety of clinical scenario.\textsuperscript{22,23} QRS prolongation has been proposed in several studies as a significant predictor of arrhythmias and mortality in HF and DCM.\textsuperscript{24,25} A study done by Lelakowski\textit{ et al} reported that a QRS complex of <118 ms provided an independent protective factor for sudden cardiac death in patients with DCM.\textsuperscript{26} Another study done by Guzzo-Merello\textit{ et al} concluded that a shorter distance in the 6 min walking test, QRS complex duration >120 ms, AF, absence of treatment with beta-blockers and
the use of digoxin were the factors associated with the occurrence of poor cardiac outcome (heart transplantation or cardiovascular death) in patients with ACM.25 Our study revealed that prolonged QRS complex duration (>120 ms) had a higher occurrence of all-cause mortality and the QRS duration was found to be an independent prognostic predictor in patients with ACM.

Echocardiography is an indispensable tool in assessment of ACM; it is under Class I recommendation of updated ‘Echocardiography Indication Guidelines’ in accordance with the ‘Standards for the Elaboration of Guidelines, Positions and Normations’ sanctioned by the Brazilian Society of Cardiology in evaluation of a suspected DCM.27 A study conducted by Fang et al reported LVEF as an independent significant predictor of mortality in univariate analysis of patients with ACM; however, in multivariate analysis, it was found to be insignificant.28 In our study, patients with an LVEF ≤35% had a higher occurrence all-cause mortality as compared with patients with LVEF >35%, and LVEF was found to be an independent prognostic predictor in patients with ACM.

Many studies were conducted on using CTP Score as a prognostic marker in cirrhotic cardiomyopathy. A study conducted by Baik et al proposed that the majority of cirrhotic patients in Child-Pugh class B and C presented at least one feature of cirrhotic cardiomyopathy namely QTc prolongation and diastolic dysfunction.29 To our knowledge, no study used CTP Scoring in assessing mortality in patients with ACM. In our study, patients assessed as having CTP Score B, C had a higher mortality as compared with the patients having CTP Score A, and CTP Score was found to be one of the independent prognostic predictors in the multivariate Cox regression analysis.

CONCLUSION
Our study indicated increased morbidity and mortality of patients with ACM in tribal population; this study has direct implications on the prevention and treatment of ACM in the Indian context in general and the Jharkhand state in particular. A comprehensive upbringing of tribals in the social, economic and educational aspects would show direct implications in the primary prevention of problems related to alcohol. Educating the people about alcohol, its consequences like ACM, symptoms of ACM, focusing especially on the rural areas is the need of hour. Timely visit to the medical facility and with proper management, morbidity and mortality of patients with ACM in tribal population would be reduced. Prolonged QRS, reduced LVEF, and CTP score were found to be significant mortality predictors for patients with ACM.

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Correction notice Author name Ajit Dungdung has been corrected to Ajit Dungdung.

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