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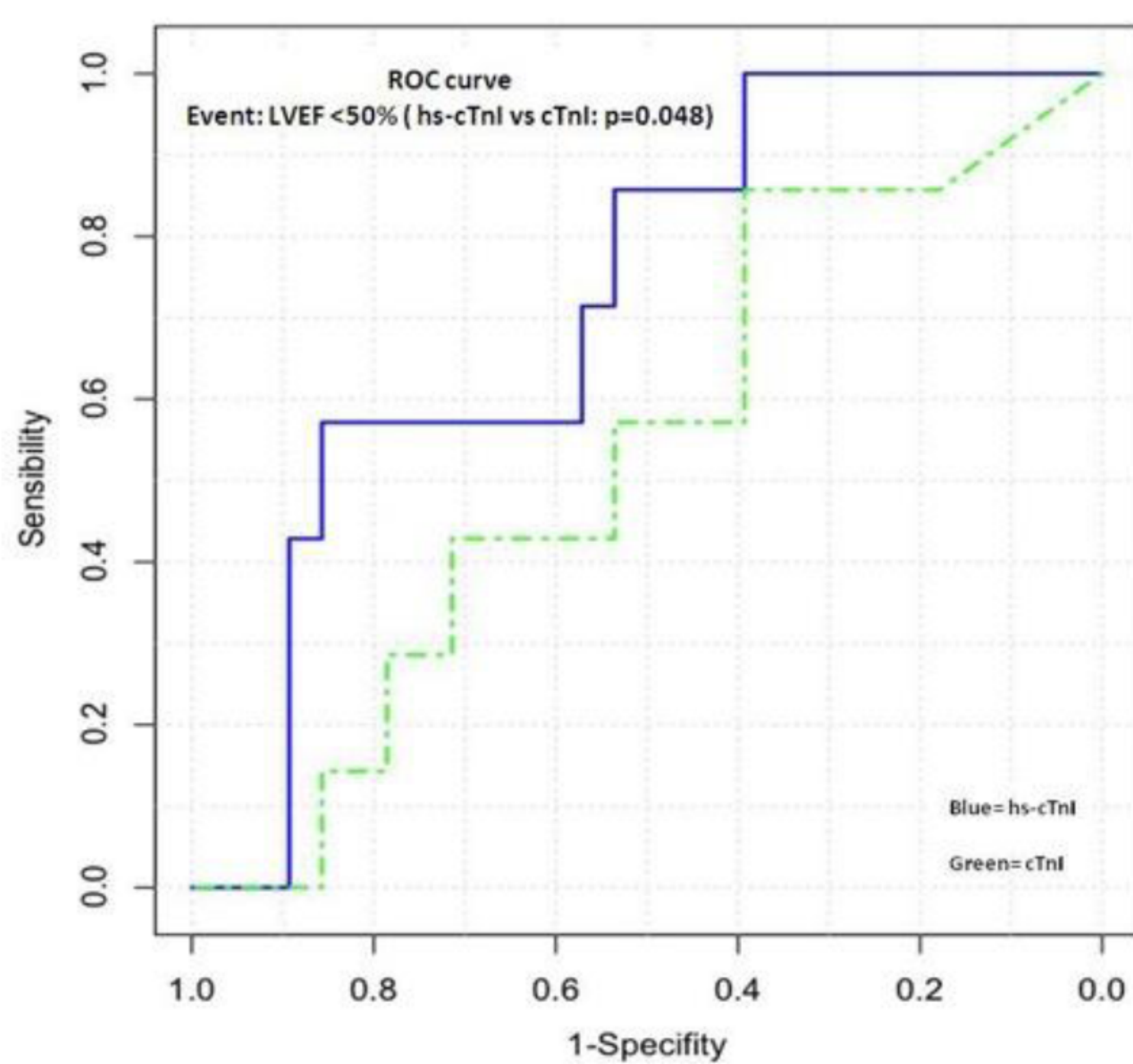
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INTRODUCTION AND AIMS

Increased levels of cardiac troponins (cTn) are diagnostic for myocardial injury and are recommended by consensus guidelines for acute myocardial infarction (AMI) diagnosis (1). Stably elevated troponin concentrations are frequently observed in chronic kidney disease (CKD) patients in the absence of clinical evidence of myocardial damage, especially if newer high sensitive troponin assays are used, with increasing problems of interpretation (2-4). For end-stage renal disease (ESRD) population there is no consensus for cTnI over cTnT, although cTnI has generally been used in clinical practice because of a lower incidence of baseline elevations in asymptomatic haemodialysed patients (5-18%), but the meaning of this elevation remains unclear (5-7). Haemodialysis itself can change cardiac enzyme levels, because of haemoconcentration or clearance of cardiac troponins, but studies show different results: some groups have demonstrated a decrease in cTn levels after HD treatment (8), others don't show any effect (9-11). A 2005 meta-analysis of asymptomatic ESRD patients, that evaluated a non-high sensitive cardiac troponin assay, showed an increased mortality risk among cTnT positive patients, but conclusions related to cTnI levels could not be performed due to problems associated with the standardization of the different immunoassays (12). Newer high-sensitive cTnI (hs-cTnI) assays are characterized by a <10% coefficient of variation (CV) at the 99th percentile in a normal reference population (13); Kumar et al. demonstrated a cTnI elevation in asymptomatic chronic haemodialysed patients using a sensitive assay (CV at 99th percentile 10%) (11). Aim of this study was to evaluate basal cTnI levels in our population of asymptomatic patients undergoing chronic HD with a very sensitive assay.

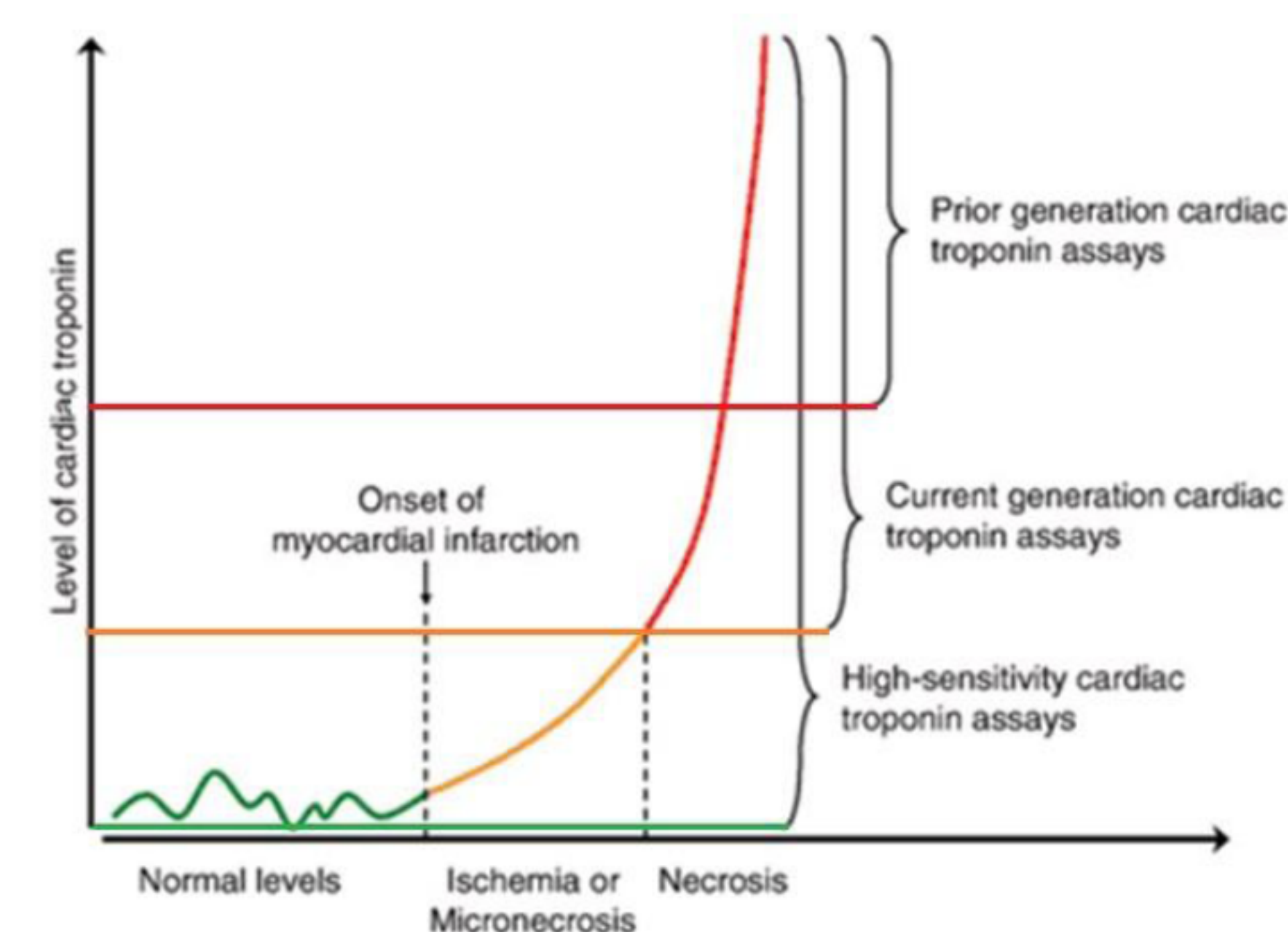
METHODS

We enrolled 35 patients who underwent chronic haemodialysis for more than 3 months; all patients were asymptomatic for chest pain and had no history of acute coronary syndrome in the past 2 months. For every patient we measured before HD: hs-cTnI (Abbott Architect, detection limit 1.2 ng/L, CV 5.6%), cTnI (Beckman Access, detection limit 0,01 µg/L, CV 14%), brain natriuretic peptide (BNP), haemoglobin (Hb), calcium (Ca⁺⁺), phosphorus (P), parathyroid hormone (PTH). We evaluated demographic, anamnestic, dialytic and echocardiographic characteristics of the examined population. Demographic and clinical characteristics were compared between the group of patients who had elevated hs-cTnI and the group with normal values. Data were examined for normality (D'Agostino and Person normality test). Categorical data were presented as percentages and compared with the Fisher's exact test; continuous normally distributed data were presented as mean standard deviation (SD) and analysed with the independent samples t-test. Data that were not normally distributed were analysed by the Mann-Whitney U-test and presented as median values (Interquartile ranges, IQR). Receiver Operating Characteristics (ROC) curve were used to compare two groups of patients who had hs-cTnI and cTnI respect to left ventricular ejection fraction (LVEF) <50%. A p-value < 0.05 was considered a priori to indicate statistical significance.



	Results		p-value
	hs-cTnI positive (n=12)	hs-cTnI negative (n=23)	
age, mean (SD)	64.2(9.32)	61.1(14.7)	0.51
sex, male(%)	2(17)	19(83)	0.0003
sex, female(%)	10(83)	4(17)	0.0003
hypertension, n(%)	8(67)	10(43)	0.28
diabetes, n(%)	4(33)	7(30)	0.99
hyperlipidaemia, n(%)	6(50)	10(43)	0.73
PAD, n(%)	3(25)	6(26)	0.99
CAD, n(%)	3(25)	5(22)	0.99
LVEF <50%, n(%)	5(42)	2(9)	0.03
LVH, n(%)	10(83)	8(35)	0.01
HD duration(months), median(IQR)	50.5(39.5-120)	61(24.75-126.75)	0.42
PTH,median(IQR)	182.5(64.2-1654)	309(162.25-718.75)	0.65
BNP(pg/mL), median(IQR)	407.1(114.8500-1728.3)	140.2(78.5250-253.12)	0.10
Hb(g/dL), mean(SD)	10.43(1.25)	11.23(1.00)	0.048
Ca ⁺⁺ (mg/dL), mean(SD)	9.4(1.26)	8.8(0.62)	0.15
P(mg/dL), mean(SD)	5.14(1.0)	5.00(1.46)	0.78

*reference ranges: 0-15.6 pg/ml for female, 0-34.2 pg/ml for male; SD: standard deviation; PAD: peripheral artery disease; CAD: coronary artery disease; IQR: interquartile range



RESULTS

Pre-HD hs-cTnI levels were elevated in 12 of 35 patients enrolled (34.2%), while pre-HD cTnI levels were increased in 4 of 35 (11.4%) patients. Only 3 of 35 patients (8.5%) had a contemporary hs-cTnI and cTnI elevation. Multivariate analysis showed a significant correlation between hs-cTnI and left ventricular hypertrophy (LVH, p=0.01) and LVEF < 50% (p=0.03). Furthermore hs-cTnI was more predictive of LVEF<50% than standard cTnI (p=0.048).

CONCLUSIONS

In agreement with the literature, this study confirmed that a sensitive assay detects more asymptomatic HD patients compared to previously used methods. Furthermore, our assay is more sensitive than that of Kumar et al. (CV 5.6% vs 10%). Our data suggest that hs-cTnI levels are correlated with cardiac dysfunction in a small population of asymptomatic patients undergoing chronic haemodialysis. Future aim is to extend the population of patients enrolled and to repeat further measurements of hs-cTnI to gain a better definition of the prognostic value of increased hs-cTnI basal levels.

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