



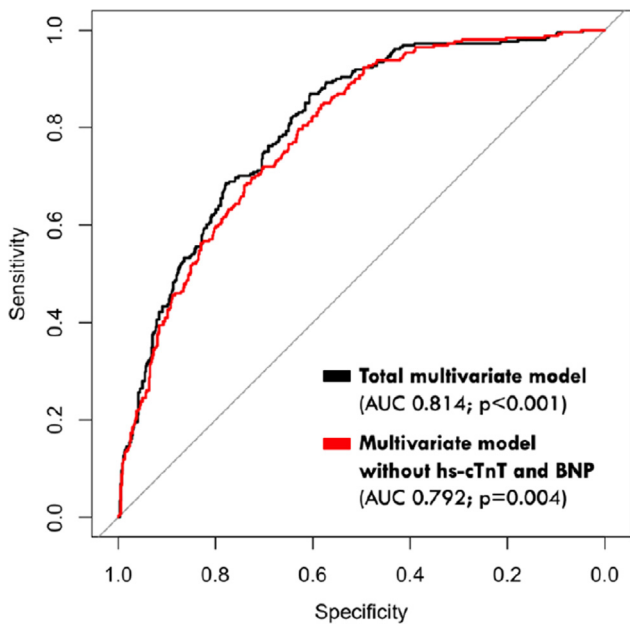
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**Methods** Between 2015 and 2019, CAC score was prospectively performed in consecutive patients with diabetes mellitus. Patients with symptoms, known coronary artery disease or history of atrial fibrillation were excluded. Within 24 h from CT examination, peripheral blood samples were taken to measure hs-cTnI and BNP. The relationship between serum hs-cTnI/BNP concentrations and zero Agatston score was assessed using univariate and multivariate binomial models. The implication of hs-cTnI and BNP in this multivariate model was evaluated using nested models associated with Chi<sup>2</sup> test of independence.

**Results** A total of 844 patients with diabetes were enrolled (61 ± 7 years, 57% men, mean duration of diabetes 18 years). In this population, 294 (35%) had a zero Agatston score, 253 (30%) an Agatston score from 1 to 100, 161 (19%) from 101 to 400, and 136 (16%) higher than 400. In univariate analysis, hs-cTnI and BNP concentrations were associated with zero Agatston score (respectively OR, 2.63 [95%CI, 1.51-5.01]; *P* < 0.001 and OR, 1.09 [95%CI, 1.01-1.22]; *P* = 0.03). In multivariate analysis, hs-cTnI and BNP concentrations were associated with zero Agatston score (respectively OR, 2.38 [95%CI, 1.51-4.76]; *P* = 0.009 and OR, 1.18 [95%CI, 1.07-1.32]; *P* = 0.001). The multivariate model included age, gender, smoking, dyslipidemia, duration of diabetes, hypertension, diabetic neuropathy, hs-cTnI and BNP concentrations, significantly discriminated the zero Agatston score (AUC = 0.81; *P* < 0.001) (Fig. 1). The most discriminant threshold was ≤ 3 ng/l for hs-cTnI and < 17 ng/l for BNP. In nested models, both hs-cTnI and BNP brought information to this multivariate model to predict a zero Agatston score (respectively *P* = 0.003 and *P* < 0.001). Moreover, removing hs-cTnI and BNP from the model results in a significant reduction in model performance (AUC = 0.79; *P* = 0.004).

**Conclusion** Cardiac biomarkers hs-cTnI and BNP are associated with zero Agatston score, which is correlated with a very low risk of cardiovascular events, in asymptomatic patients with diabetes mellitus.



**Figure 1** Receiver operating characteristic (ROC) curves demonstrating the ability of hs-cTnI and BNP to significantly predict a zero Agatston score.

**Disclosure of interest** The authors declare that they have no competing interest.

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## 260 Cardiovascular Comorbidities and Covid-19 in Women



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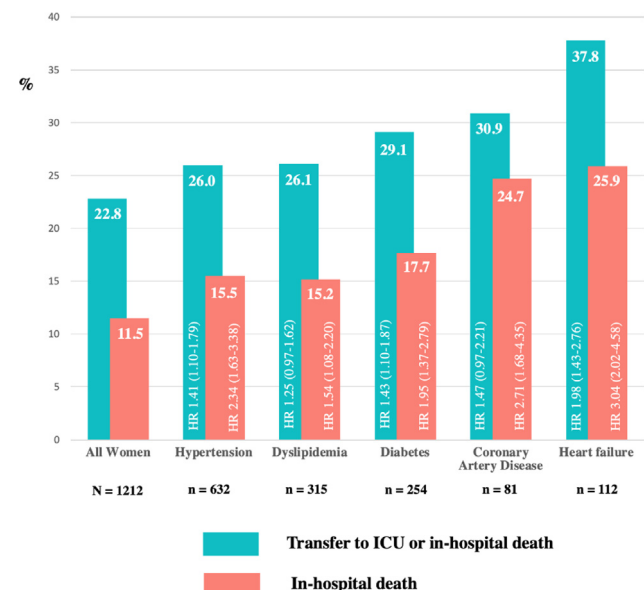
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**Background** While women account for 40-50 % of patients hospitalized for coronavirus disease 2019 (Covid-19), no specific data have been reported in this population.

**Purpose** Assess the burden of cardiovascular comorbidities on outcomes in women hospitalized for Covid-19.

**Methods** We conducted a retrospective observational multicenter study from February 26 to April 20, 2020 in 24 French hospitals including all adults admitted for Covid-19. Primary composite outcome included transfer to intensive care unit (ICU) or in-hospital death.

**Results** Among 2878 patients hospitalized for Covid-19, 1212 (42.1 %) were women. Women were significantly older (68.3 ± 18.0 vs. 65.4 ± 16.0 years, *P* < 0.001) but had less prevalent cardiovascular comorbidities than men. Among women, 276 (22.8 %) experienced the primary outcome, including 161 (13.3 %) transfer to ICU and 115 (9.5 %) deaths without transfer to ICU. The survival free from death or transfer to ICU was higher



**Figure 1** Impact of Cardiovascular Comorbidities on Outcomes in Women Hospitalized for Covid-19 ICU denotes intensive care unit; HR hazard ratio Univariate hazard ratios and corresponding 95 % confidence interval are provided in barplots.

in women (HR 0.63, 95 %CI 0.53-0.73,  $P < 0.001$ ), whereas the observed difference in in-hospital deaths did not reach statistical significance ( $P = 0.18$ ). The proportion of women that experienced the primary outcome were 37.8 % in women with heart failure ( $n = 112$ ), 30.9 % in women with coronary artery disease ( $n = 81$ ), 29.1 % in women with diabetes ( $n = 254$ ), 26.1 % in women with dyslipidemia ( $n = 315$ ), and 26.0 % in women with hypertension ( $n = 632$ ). Age (HR 1.05, 5 years increments, 95 %CI 1.01-1.10), body mass index (HR 1.06, 2 units increments, 95 %CI 1.02-1.10), chronic kidney disease (HR 1.57, 95 %CI 1.11-2.22), and heart failure (HR 1.52, 95 %CI 1.04-2.22) were independently associated with the primary outcome (Fig. 1).

**Conclusions** Women hospitalized for Covid-19 were older and had less prevalent cardiovascular comorbidities than men. While female sex was associated with a lower risk of transfer to ICU or in-hospital death, Covid-19 remains associated with considerable morbi-mortality in women, especially in those with cardiovascular diseases.

**Disclosure of interest** The authors declare that they have no competing interest.

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### Trends in in-hospital and out-of-hospital coronary heart disease mortality rates in French registers during the period 2000 to 2016



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**Purpose** To estimate the trends in- and out-hospital coronary heart disease (CHD) mortality rates from 2000 to 2016 and their respective proportions in total mortality using data of the three French CHD registries (Strasbourg, Toulouse, Lille).

**Methods** All fatal myocardial infarction (MI), coronary deaths (CD) and sudden deaths occurring between January 1st, 2000 and December 31st of 2016, were recorded for patients aged 35 to 74 years.

**Results** A total of 20,818 events were recorded in the three registers, of which 69.4% were out-of-hospital. Age- and region-adjusted CHD mortality rate decreased with an annual percentage change (APC) of -3.6% in men and -3.9% in women. This significant decrease was approximately twice as high for in-hospital deaths compared to out-of-hospital deaths ( $P < 10^{-5}$  for both). While the out-of-hospital death rate represented 63.6% of the standardized total CHD mortality rate in 2000 for men, it was 70.1% in 2016. For women, these parts were respectively 63.9% and 70.5%. In men aged 35 to 54, the APC was twice higher for out-of-hospital deaths (-3.0%,  $P < 10^{-3}$ ) than for in-hospital deaths (-1.5%,  $P = 0.01$ ) whereas the opposite was observed in men aged 55 to 74 (-1.4%  $P = 0.01$  vs -5.2%  $P < 10^{-5}$ , respectively) (Table 1).

Table 1 Trends in CHD mortality rate, 2000-2016.

	Total		In-hospital		Out-of-hospital		
	APC	P	APC	P	APC	P	
Men	-3.6	$< 10^{-5}$	-4.5	$< 10^{-5}$	-3.1	$< 10^{-5}$	
Women	-3.9	$< 10^{-5}$	-5.3	$< 10^{-5}$	-3.2	$< 10^{-5}$	
Men	Strasbourg	-3.6	$< 10^{-5}$	-4.8	$< 10^{-5}$	-3.0	$< 10^{-5}$
	Toulouse	-4.1	$< 10^{-5}$	-4.3	$< 10^{-4}$	-4.0	$< 10^{-4}$
	Lille	-2.9	$< 10^{-5}$	-4.2	$< 10^{-5}$	-2.2	$< 10^{-3}$
Women	Strasbourg	-3.4	$< 10^{-5}$	-4.8	$< 10^{-3}$	-2.6	$< 10^{-2}$
	Toulouse	-5.4	$< 10^{-5}$	-6.6	$< 10^{-2}$	-4.9	$< 10^{-3}$
	Lille	-3.1	$< 10^{-3}$	-4.4	$< 10^{-4}$	-2.4	$< 10^{-2}$

**Conclusion** More than two-thirds of the deaths from CHD occurred outside the hospital. The CHD mortality rates significantly decreased during the 2000-2016 period, for both genders. However, this decline was slighter for out-of-hospital deaths than for in-hospital deaths, pointing out the need to strengthen primary and secondary prevention of CHD.

**Disclosure of interest** The authors declare that they have no competing interest.

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### What can we learn from cardiac and genetic screening of relatives in families with sacromeric hypertrophic cardiomyopathy?



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**Background** Hypertrophic cardiomyopathy (HCM) is a genetic disease with delayed cardiac expression. Family screening strategy includes cardiac screening that may be guided by predictive genetic testing (PGT). However, little is known about the impact of PGT and the risk factors present at the very early stage of the disease.

**Purpose** Our study aims to evaluate the impact of predictive genetic testing on the rate of initiation of cardiac screening, to estimate the penetrance of HCM and to determine the frequency of risk factors (RF) for sudden cardiac death in individuals at preclinical stage without HCM.

**Results** We studied 60 consecutive relatives in a single center (31 women and 29 men, mean age:  $34 \pm 16.3$  years) without history of HCM before genetic testing and who were mutation carriers after PGT. Cardiac screening was initiated before PGT in only 37%, and was started after PGT in 63%. Echocardiographic criteria for HCM were observed in 17% (28% of men but 6% of women, 17% of MYH7 mutation carriers but 0% in MYBPC3 carriers). Among mutation carriers without hypertrophy ( $n = 50$ ), 1 had non-sustained ventricular tachycardia, 2 had non-explained syncope and 5 presented with family history of sudden death. In addition, 14% had dilated left atrium, 2% a high risk mutation and 8% practiced intense physical activity. Moreover, 10% (2/21) of carriers who performed MRI had cardiac fibrosis and 3% (1/32) of carriers who performed Holter monitoring had significant premature ventricular beat (PVB > 240/day).

**Conclusion** The assessment of HCM causal mutations in relatives seems to be an important step to initiate cardiac screening. The