IMPACT OF ATRIAL FIBRILLATION TYPE IN ACUTE CORONARY SYNDROME AND THE ANTITHROMBOTIC STRATEGY

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BACKGROUND: Atrial fibrillation (AF) is an adverse prognostic factor during acute coronary syndrome (ACS). Current evidence recommends dual antithrombotic therapy (DAT), 1 antiplatelet drug and 1 anticoagulant drug, as the default strategy after nonST elevation ACS.

AIM: **To identify the clinical differences and prognosis of AF type-new onset (nAF) or pre-existing (pAF)- during ACS, to evaluate antithrombotic strategy at hospital discharge (HD) and its impact on haemorrhagic and ischemic events.**

METHODS AND RESULTS:

Retrospective observational cohort study

3241 patients with ACS
6-year period
12-months follow-up

364 AF
11.2%

230 (63.2%) with new-onset AF

134 (36.8%) with pre-existing AF

Table 1. Baseline characteristics of the study population according to the type of AF.

BASELINE CHARACTERISTICS	Pre-existing AF (n=134)	New-onset AF (n=230)	p-value
DEMOGRAPHICS			
Age (years), mean ±SD	74.9±10.4	73.02 ±11.6	p=0.133
Male, n(%)	97(72.4)	160(69.6)	p=0.569
BMI (kg/m²), median±IQR	27.1 ±5.3	26.4 ±4.8	p=0.420
CV RISK FACTOS			
Hypertension, n(%)	114(85.1)	172(74.8)	***p=0.021***
Mellitus diabetes, n(%)	51(38.1)	83(36.1)	*p=0.707*
Dyslipidemia, n(%)	87(66.4)	126(58.9)	*p=0.162*
Smoking habits, n(%)	40(29.9)	82(35.7)	*p=0.258*
COMORBIDITIES			
Previous MI, n(%)	36(26.9)	40(17.5)	***p=0.034***
Previous PCI, n(%)	23(17.3)	22(9.6)	***p=0.033***
Previous CABG (n(%)	19(14.3)	20(8.7)	*p=0.097*
Valvular disease, n(%)	17(12.8)	13(5.7)	***p=0.017***
Previous Sroke, n(%)	22(16.5)	22(9.6)	***p=0.05***
PAD, n(%)	10(7.5)	11(4.8)	*p=0.282*
Renal failure, n(%)	15(11.3)	17(7.4)	*p=0.208*
COPD, n(%)	9(6.8)	11(4.8)	*p=0.425*
CLINICAL PRESENTATION			
STEMI, n(%)	38(28.4)	129(56.1)	***p<0.001***
Killip Class≥3, n(%)	11(8.2)	29(12.6)	*P=0.196*
BLOOD TESTS			
Peak Creatinine (mg/dL), median±IQR	1.2 ±0.7	1.3 ±0.7	*p=0.075*
Nadir Hemoglobin (g/dL), mean±SD	12.2±1.6	11.7 ±2	***p=0.015***
ECHOCARDIOGRAPHY			
LVEF ≤40%,n(%)	54(45.0)	109(52.9)	p=0.168
Left atrial enlargement at least moderate, n(%)	55(41.7)	38(16.9)	***p<0.001***
MR>II/IV, n(%)	32(24.2)	44(19.6)	*p=0.296*
CORONARY ANGIOGRAPHY			
Absence significant CAD, n(%)	10(7.5)	7(3)	p=0.05
Multivessel disease, n(%)	85(63.4)	138(60)	p=0.517

Table 2. In-hospital complications according to the type of AF.

In-Hospital complications	Without AF (n=2877)	Pre-existing AF (n=134)	New-onset AF (n=230)	p-value (NAF- PAF)
Death, n(%)	49(1.7)	5(3.7)	21(9.1)	p<0.001
Re-infarction, n(%)	42(1.5)	0(0)	8(3.5)	p=0.028
Stroke, n(%)	17(0.6)	3(2.2)	6(2.6)	p=0.005
Cardiogenic shock, n(%)	131(4.6)	6(4.5)	34(14.8)	P=0.002
In-hospital composite endpoint*, n(%)	181 (6.3)	9(6.7)	46(20)	p<0.001

*In-hospital composite endpoint (death, stroke, reinfarction and cardiogenic shock).

Compared with the population without AF, nAF was a predictor of in-hospital death (OR 2.9, p=0.03) and in-hospital composite endpoint (OR 2.5, p=0.001) in multivariate analysis, but pAF wasn't.

During 12-months follow-up of pts with ACS and AF, there was no difference regarding death or follow-up composite endpoint (death, stroke and ACS) between the AF types.

Table 3. Antithrombotic strategy at hospital discharge.

ANTITHROMBOTIC STRATEGY AT HOSPITAL DISCHARGE*	AF (n=338)	pAF (n=129)	nAF (n=209)	*p-value* (pAF-nAF)
Triple antithrombotic therapy (TAT), n(%) (2 antiplatelet drugs and 1 anticoagulant drug)	161(47.6)	77(59.7)	84(40.2)	*p<0.001*
Dual antithrombotic therapy (DAT), n(%) (1 antiplatelet drug and 1 anticoagulant drug)	42(12.4)	31(24)	11(5.3)	*p<0.001*
Just anticoagulant therapy, n(%)	10(3)	7(5.4)	3(1.4)	*p<0.001*
Just dual antiplatelet therapy, n(%)	121(35.8)	12(9.3)	109(52.2)	*p<0.001*
Stop the 2ª antiplatelet in patients with TAT, n(%)				
1-month after hospital discharge	23(16.8)	9(13)	14(20.6)	
3-month after hospital discharge	19(13.9)	10(14.5)	9(13.2)	*p=0.294*
6-month after hospital discharge	60(43.8)	28(40.6)	32(47.1)	
12-month after hospital discharge	35(25.5)	22(31.9)	13(19.1)	

*Loss of follow-up of 4 patients.

During follow-up, pts discharged with TAT had trend, not statistically significant, towards more haemorrhagic and both groups had similar ischaemic events. In multivariate analysis the choice of TAT or DAT wasn't a predictor of ischaemic events.

CONCLUSION:

In ACS, pts with nAF had worst in-hospital outcomes. Regarding antithrombotic strategy at HD pts with nAF were less often anticoagulated and less often treated with TAT.
In our study the choice between DAT or TAT had no statistical impact on follow-up outcomes.

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