Myocardial infarction with non obstructive coronary arteries (MINOCA) according to definitions of 2020 ESC Guidelines: clinical profile and prognosis

D. Nieto Ibanez[1], A. Fraile Sanz[1], B. Izquierdo Coronel[1], C. Perela Alvarez[1], R. Olsen Rodriguez[1], R. Abad Romero[1], D. Galan Gil[1], M. Espinosa Pascual[1], C. Moreno Vinues[1], J. Lopez Pais[2], P. Awamleh Garcia[1], R. Mata Caballero[1], J.J. Alonso Martin[1]

[1]*University Hospital of Getafe - Madrid Health Service, Getafe, Spain;* [2]*Complexo Hospitalario Universitario de Ourense, Ourense, Spain*

On behalf of IMACORN Group

Funding Acknowledgement: Type of funding sources: None.

Background: MINOCA's physiopathology, treatment and prognosis are yet to be completely understood. The aim of this study is to compare baseline characteristics and prognosis of MINOCA patients and those of patients with myocardial infarction (MI) and obstructive coronary arteries.

Methods: We analysed all consecutive patients with MI who underwent coronary angiography admitted in a University Hospital covering a population of 220.000 people during a period of 60 months. The database and the all the patient's angiographies were revised by a group of experts in order to adequate MINOCA to 2020 ESC Guidelines definition and the American Heart Association position paper.

Results: 680 patients, 68 of whom were MINOCA (10%) with a median of follow up of 31±16 months were analysed (see table 1). We found no differences in both groups' age. Female gender was more prevalent among MINOCA patients. The underlying mechanism in MINOCA was coronary spasm (17.6%), plaque rupture (13.2%), coronary embolism (7.4%), coronary dissection (2.9%), type II infarction (19.1%) or unknown (39.7%). Coronary arteries in MINOCA patients had no obstructions at all in 57.4%, and 30–50% obstruction in 42.6% of the cases. MINOCA patients didn't have higher prevalence of cancer, autoimmune or psychiatric diseases, dyslipidaemia, hypertension or inflammatory analytical parameters. However, we found significant differences in atrial fibrillation, migraine, connective tissue diseases, tobacco use and diabetes. We found no effect of stress in the development of MINOCA (measured with validated STAI and DS-14 scales). Symptoms at admission didn't differ between the two groups, but those with MINOCA had normal ECG more frequently. Prognosis showed relevant differences, as MINOCA patients had less major cardiovascular complications, such as inotropic requirements (0% Vs 4.8%, p=0.04), shock (0% vs 6.6%, p=0.013) and left ventricular dysfunction (11.8 vs 30.2, p=0.015). Furthermore, myocardial injury biomarkers' levels were, significantly lower in MINOCA patients. Death rates tend to be lower both in hospital (0% vs 3.1%, p=0.131) and during follow up (9.1% vs 11.5%, p=0.369).

Conclusion: Analysing MINOCA patients' clinical profile might help us understanding the underlying physiopathology, prognosis and treatment targets. In these patients, classic cardiovascular risk factors don't appear to be as important as in obstructive patients. At admission, we found no clinical differences that could help making an early diagnosis, even if those with normal ECG and lower levels of myocardial injury biomarkers are more likely to have non-obstructive coronary arteries. These patients seem to have better prognosis and lower myocardial injury than those with obstructive coronary arteries. Further research is needed to provide more evidence on the accurate treatment of these patients.

Table 1: Baseline characteristics of patients with MINOCA Vs MI with obstructive coronary arteries.

	MINOCA (68)	Obstructive (612)	p
Male sex (%)	52.9 (N = 36)	76.6 (N = 469)	< 0.01
Age median (years)	70 ± 11.5	71 ± 10	0.355
Atrial fibrillation (%)	14.7 (N = 10)	7.4 (N = 45)	0.038
Psychiatric disease (%)	19.1 (N = 13)	12.1 (N = 74)	0.079
Migraine (%)	11.8 (N = 8)	3.3 (N = 20)	0.04
Cancer (%)	14.7 (N = 10)	12.0 (N = 73)	0.139
Autoimmune disease	13.2 (N = 9)	7.0 (N = 43)	0.064
Connective tissue disease (%)	5.9 (N = 4)	1.3 (N = 8)	0.025
Smokers (%)	44.8 (N = 30)	72.3 (N = 395)	< 0.01
Diabetes (%)	20.6 (N = 14)	34.8 (N = 212)	0.011
Dyslipidaemia (%)	44.8 (N = 30)	55.8 (N = 339)	0.055
Hypertension (%)	67.6 (N = 46)	61.0 (N = 371)	0.175
Stress (%)	50.0 (N = 43)	33.5 (N = 145)	0.079
Previous treatment with acetylsalicylic acid (%)	17.6 (N = 12)	29.9 (N = 182)	0.021
Chest pain at admission (%)	76.5 (N = 52)	83.5 (N = 511)	0.102
Normal ECG at admission (%)	23.5 (N = 16)	11.3 (N = 68)	< 0.01
Reduced Left Ventricle Ejection Fraction at discharge (%)	11.8 (N = 8)	30.2 (N = 189)	0.015
Treatment with AAS at discharge (%)	63.2 (N = 43)	97.6 (N = 578)	< 0.01
Treatment with double antiplatelet therapy at discharge (%)	52.9 (N = 56)	92.4 (N = 547)	< 0.01
Treatment with IECA at discharge (%)	42.6 (N = 29)	64.3 (N = 380)	0.07
Treatment with beta blockers at discharge (%)	51.5 (N = 35)	84.7 (N = 500)	< 0.01
Treatment with statins at discharge (%)	73.5 (N = 50)	95.9 (N = 568)	< 0.01
Inotropic requirements during hospitalization (%)	0.0 (N = 0)	8.5 (N = 50)	0.04
Acute pulmonary oedema during hospitalization (%)	0.0 (N = 0)	4.8 (N = 29)	0.43
Cardiogenic shock during hospitalization (%)	0.0 (N = 0)	6.6 (N = 40)	0.013
Death during hospitalization (%)	0.0 (N = 0)	3.1 (N = 19)	0.131
Death during follow-up (%)	9.1 (N = 6)	11.5 (N = 65)	0.369
STAI scale punctuation (points)	26 ± 18	38 ± 13.5	0.537
DS-14 scale punctuation (points)	22.5 ± 12.0	18.0 ± 7.5	0.689
Creatinine at admission (mg/dL)	0.9 ± 0.15	1 ± 0.15	0.013
Troponine T hs (ng/L)	273 ± 175.5	942.5 ± 1160.5	< 0.01
CK (U/L)	199.05 ± 136.0	433±456.5	< 0.01
C- Reactive Protein (mg/L)	4 ± 4	5 ± 5.5	0.195
Heart rate (bpm)	78 ± 12.5	78 ± 11.5	0.417
Systolic blood pressure at admission (mmHg)	149 ± 21	140 ± 19	0.26

* Qualitative variables are expressed as percentage and number of cases. Quantitative variables are expressed as median and interquartile range.