Outflow tract obstruction and Takotsubo syndrome

We appreciated the interesting article by El Mahmoud et al on left ventricular (LV) obstruction tract (LVOT) in Takotsubo syndrome (TS).[1] We performed an observational prospective study on TS between 2003 and 2008. Takotsubo syndrome was classified according the ventriculogram as classic type with apical involvement; other variants were excluded.[2] Patients were divided into 2 groups: with significant LV septal hypertrophy (LVH+, 17 patients) by echocardiogram, defined as septal wall thickness ≥1.4 cm, and with less (LVH−, 34). Left ventricular obstruction tract gradient was only detected in LVH+ (58.8% vs 0%, $P \leq .001$). Echocardiograms showed structural differences (Table I). Left ventricular recovery was detected in both groups (mean 42.5 days), up to 44% before discharge. When gradient was specifically addressed (>30 mm Hg), its presence was previously known via echocardiogram in 4 patients (40%). During dyskinesia, the gradient increased in almost all. After LV recovery, gradient resolved in 5 patients, decreased in 2, remained unchanged in 21, and increased in 1 (Figure 1). Both groups had favorable long-term outcomes. Only 2 died, one patient each group (sudden death and sepsis).

Hypertrophic cardiomyopathy was deemed as an exclusion criterion for TS by the Mayo Clinic criteria. Recently, this controversial exclusion condition was removed from the Mayo revised criteria.[3] Although LVOT is typically associated with hypertrophic cardiomyopathy, LVOT also has been hypothesized to play a major role in the development of TS[4] but denied by others.[5] Actually, it is not an uncommon finding in TS (12/72 in the series of Tsuchihashi et al,[6] 2/13 in the series of Desmet et al,[5] and 10/51 in our series[2]). Left ventricular obstruction tract has also been demonstrated in other various clinical settings as simply LV hypertrophy, reduced LV cavity size (bleeding, dehydration), mitral valve abnormalities, and hypercontractility (stress, anxiety, and inotropic agents).[7] Our data suggest that dynamic LVOT could be related to a predisposing LV geometry (only appeared in the LVH+ cohort) and secondary to the Takotsubo physiology more than a proper cause of this syndrome. Thrombus formation in the LV apex could also be related to the presence of LVOT added to the stasis in an akinetic area.

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Iván Javier Núñez-Gil, MD, PhD
Juan Carlos García-Rubira, MD, PhD
Cardiovascular Institute
Hospital Clínico San Carlos
Madrid, Spain
E-mail: ibnsky@yahoo.es

María Luaces, MD, PhD
Cardiology Department
Hospital de Fuenlabrada
Madrid, Spain

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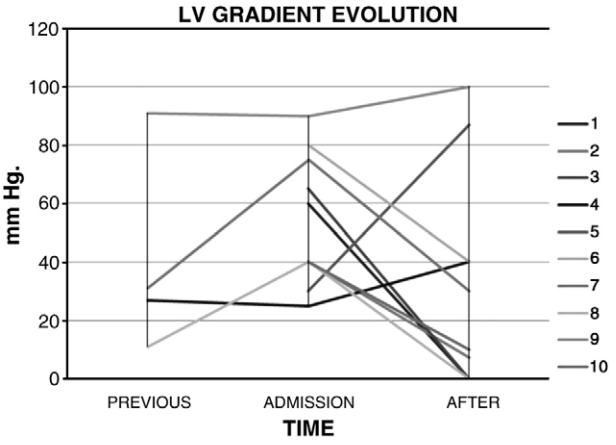
Table I. Echocardiograms showing structural differences between groups

	Overall	LVH–	LVH+	*P*
Days to cardiac catheterization	1.0 (0.0-2.0)	1.0 (0.0-2.0)	1.0 (0.0-1.0)	.58
LVEF on admission	45.94 ± 11.30	46.97 ± 12.87	43.88 ± 7.13	.36
LVEF after full recovery	63.78 ± 6.93	64.71 ± 7.52	61.94 ± 5.29	.18
Time to full recovery (days)*	42.5 (7.5-110.2)	51.0(8.0-137.5)	39.0 (5.5-104.0)	.58
Full recovery before discharge	21 (41.2%)	15 (44.1%)	6 (35.3%)	.76
Peak MR grade (≥II/IV)	7 (13.7%)	4 (11.8%)	3 (17.6%)	.673
Echocardiogram measurements (cm)				
LA dimension	4.02 ± 0.81	3.99 ± 0.92	4.06 ± 0.62	.79
LV diastolic dimension	4.63 ± 0.64	4.75 ± 0.61	4.41 ± 0.65	.09
LV systolic dimension	2.77 ± 0.49	2.92 ± 0.55	2.55 ± 0.29	.03
Septal thickness	1.24 ± 0.31	1.04 ± 0.17	1.59 ± 0.16	.000
Posterior wall thickness	1.10 ± 0.22	1.00 ± 0.18	1.27 ± 0.19	.000
Presence of LV gradient	10 (19.6%)	0	10 (58.8%)	.000
LV thrombus	3 (5.9%)	1 (2.9%)	2 (11.8%)	.25

LVEF, left ventricular ejection fraction; *MR*, mitral regurgitatic; *LA*, left atrium; *LV*, left ventricle.
*Days to control echocardiogram showing a full LV recovery.

Figure 1



Left ventricular outflow tract gradient evolution, regarding the Takotsubo episode (admission).