Brugada Syndrome: Differential diagnosis with variant angina or Prizmetal angina

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In the following table the main differential features between both entities are explained:

DIFFERENTIAL CHARACTERISTICS BETWEEN BRUGADA SYNDROME AND VASOSPASTIC ANGINA

	BRUGADA SYNDROME	VASOSPASTIC ANGINA
Precordial pain:	No.	Yes.
Tendency to VT/VF:	High.	High.
Structural heart disease:	Absent.	Could exist.
Response to nitrates and nitroglycerine:	Null.	Improves or suppresses clinical/electrocardiographic manifestations.
Permanence of ST segment elevation:	Persistent (or fluctuating) and without pain.	Brief, transitory and accompanied by pain.
Cause:	Genetic alteration of Na^+channel.	Possible alteration in production nitrous oxide in vascular wall.
Presence of image in mirror or reciprocal in ECG:	Could be present.	Present.
Topography of ST elevation:	Right precordial leads of V1 to V3; it could rarely be observed in inferior wall and triggered or increased by antiarrhythmic agents of the IC[1]and IA classes .	Variable. It could alternate between precordial leads and inferior ones. It could be triggered by hyperventilation.
Dromotropic disorders:	AV block of the first degree by	It could happen in a transitory way until a

	extension of H-V in 50% of cases and in carriers of the mutation.	high degree of AV block during the episode; it is associated with a higher risk of arrhythmia and SCD.
Persistent T wave inversion:	Negative T wave in precordial leads from V_1 to V_3, characteristic of type 1.	Inverted and deep T waves from V_1 to V_4 associated to anterior hypokinesia, suggesting myocardial “stunning” that indicates critical lesion of the anterior descending artery: “LAD-T wave pattern”.
Presence of transitory Q wave:	No.	It could happen.
Effort test:	It could normalized the variation during effort.	Variable response.
Myocardial scintigraphy with thallium 201:	Normal.	Transitory transmural hypo -uptake.
Response to test of maleate of ergonovine in doses of 0.05 to 0.40 mg (stimulant of alpha adrenergic and seratoninergic receptor)	There could be a mild diffuse reduction of caliber without spasm when doses are equal to or less than 0.40 mg are used.	Intense coronary spasm accompanied by pain and ST elevation. Possible cardiac block, asystole and VT.
Response to hyperventilation:	It does not modify.	Severe spasm and reproduction of clinical electrocardiographic manifestations.

Response to intracoronary acetylcholine, each dose given in a time above one minute, in doses of **10, 25, 50 and 100μg doses separated by five minute intervals:**	It could worse the ST elevation with paradoxical dilation of coronary vessels.	Severe spasm and reproduction of clinical electrocardiographic manifestations.
Response to magnesium sulfate:	Not mentioned.	Suppresses attacks induced by hyperventilation and exercise.
Treatment:	Automatic implantable cardioverter defibrillator in association with amiodarone, a drug that contributes to diminish the number of shocks. Isoproterenol indicated in elctric storm associated with general anesthesia and cardiopulmonary "bypass" or Amiodarone?	Calcium antagonists, such as nifedipine, diltiazem, verapamil, and felodipine associated to nitrates. Benefit with prazosin is mentioned.

Nishizaki et al. **Nishizaki M, Fujii H, Ashikaga T, Yamawake N, Sakurada H, Hiraoka M.ST-T wave changes in a patient complicated with vasospastic angina and Brugada syndrome: differential responses to acetylcholine in right and left coronary artery.Heart Vessels. 2008; 23: 201-205.** presented a 49-year-old man with chest pain and syncope with saddleback or occasionally coved type ST elevation from V1 to V3. Coronary spasm in the left anterior descending artery was induced by acetylcholine injection and ST elevation changed from saddleback to coved type in V2-V3 together with ST depression in V4-V5 (mirror image), whereas acetylcholine injection into the right coronary artery did not provoke spasm, but induced augmented and coved type ST elevation in V2 without ST-T changes in V4-V5. These ECG changes in response to acetylcholine administration into each coronary artery are compatible with pathogenesis of vasospastic angina and Brugada syndrome, respectively.

Yagihara et al**. Yagihara N,Sato A,Furushima H,Chinushi M,Hirono T,Aizawa Y. Ischemia-induced prominent J waves in a patient with Brugada syndrome.Intern Med.2010;49:1979-1982.)** presented a

comproved case with concomitant BrS (Extrastimuli induced VF.) and Prinzmetal angina (intracoronary ergonovine maleate induced spasms) treated with ICD, and Ca antagonist and isosorbide dinitrate.

Nakamura W, Segawa K, Ito H, et al. Class IC antiarrhytmic drugs: flecainide and pilsicainide, produce ST segment elevation simulating inferior myocardial ischemia. J Cardiovasc Electrophysiol 1998; 9: 855-85.