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Management of Atrial Fibrillation Leading to BRASH Syndrome: Case Report

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Abstract **Case Report**

BRASH Syndrome is an acronym for Bradycardia, Renal failure, Atrioventricular (AV) node blocker, Shock and Hyperkalemia. This is a rare clinical entity, few medical articles are found in the literature review. The diagnosis should be suggested in patients on a combination of anti-arrhythmic drugs. We report a case of BRASH syndrome complicating the management of atrial fibrillation with high ventricular rate.

Keywords: BRASH Syndrome, anti arrhythmics, shock.

INTRODUCTION

Brash syndrome is an acronym for Bradycadia, Renal failure, atrioventricular node blocker, shock and Hyperkaliemia.

In this article, we report a case of Brash syndrome complicating the managment of atrial fibrillation with high ventricular rate.

CASE PRESENTATION

A 62-year old man presented to the emergency department with one year history of palpitations, initially intermittent, then permanent 7 days prior to admission. He developed progressively worsening dyspnea (NYHA class II-III). He had no associated chest pain, lipothymia nor syncope.

On examination, his blood pressure was 100/50 mmHg, and he had a rapid irregular heart rate at 220 beats per minute. The electrocardiogram (EKG) reveled an atrial fibrillation with a rapid ventricular response (Figure 1):

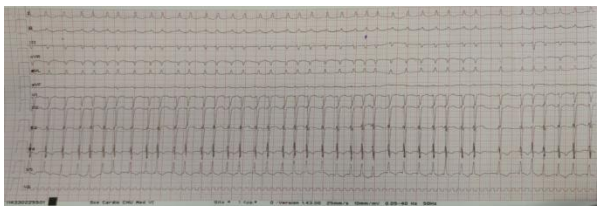

Fig-1: Initial EKG showing atrial fibrillation at high ventricular rate

Transthoracic echocardiogram showed a normal-sized left ventricle, a slightly dilated left atrium along with markedly dilated and hypertrophic right cavities. The ejection fraction (EF) per tachycardia was estimated at 40%-45%. We were not able to estimate the pulmonary artery pressure (sPAP) given the patient's tachycardia.

Chest computerized tomography (CT) paired with computed tomography angiography (CTA) were performed and showed no signs of proximal pulmonary embolism. There was, however, moderate bilateral pleural effusion, ground-glass opacities with bullous paraseptal emphysema lesions in the upper lobes of the lungs and linear atelectasis in the middle lobe. Thyroid function tests revealed a hyperthyroidism with low TSH and elevated T4. Thyroid ultrasonography revealed diffuse thyroid enlargement with multiple low risk nodules in the right lobe rated EU-TIRADS 3.

The remaining of the biological workup showed normal renal and hepatic functions, no anemia, and a negative C - reactive protein test.

Carbimazole was initiated to treat the hyperthyroidisme, along with anticoagulation with enoxaparine. We have opted for rate control given the probability of chronic atrial fibrillation, and considering that hyperthyroidism is associated with a high rate of cardioversion failure.

Following the recommendations, the patient was put on oral bisoprolol and digoxin. On the third

day, we noted no improvement in symptoms, so we switched to propranolol which has shown its effectiveness in the context of hyperthyroidism. On the fifth day, the ventricular rate remained uncontrolled. We screened the patient for atrial thrombi by transesophageal echocardiography (TEE) before attempting an electrical cardioversion that failed to normalise the heart rate. On the seventh day, our patient developed dyspnea and wheezing. We therefore decided to switch the verapamil and propranolol with atenolol which is cardioselective and has demonstrated efficacy in the context of dysthyroidism.

Amiodarone was not initiated given the risk of thyrotoxicosis, we started diltiazem with EKG and biological monitoring and the patient remained on atenolol 100 mg, digoxin 0, 25 mg and diltiazem 60 mg three times a day.

The rate control was obtained after 3 days of this association (Figure 2). The patient was then discharged symptom frees with a prescription of Atenolol 100 mg and diltiazem 60 mg three times a day. The biological workup showed normal renal function and a potassium level at 4.3 Meq/l.

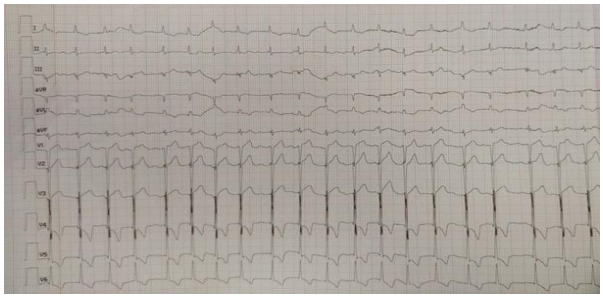

Fig-2: EKG after antiarrhythmic drugs

One week later, the patient presented with signs of shock: heavy sweating and severe hypotension. EKG showed high degree heart block. The patient underwent temporary cardiac pacing Figure 4-5.

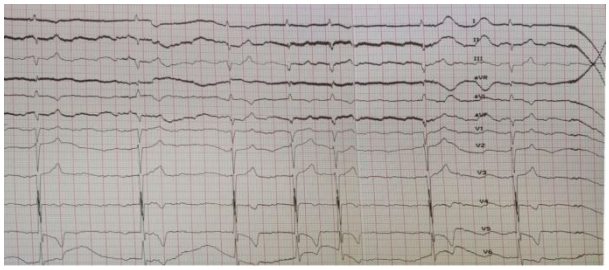

Fig-3: EKG showing a high degree heart block

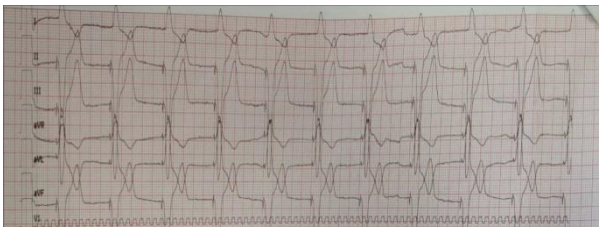

Fig-4: EKG after pacing

Laboratory investigations revealed a moderate hyperkalemia at 6,8 meq/l, acute renal failure with anuria. Dialysis could not be carried out considering the hemodynamic instability. Unfortunately, the patient died a few hours later.

DISCUSSION

The rate control management in atrial fibrillation is essentially based on intravenous betablockers, calcium channel blockers and cardiac glycosides which must be available in every hospital [1]. Unfortunately, in Morocco those emergency drugs are unavailable, leading to the association of many antiarrhythmics with risk of interaction and increased side effects.

We suggest that our patient developed a BRASH syndrome after his discharge from the hospital, which is an acronym for Bradycardia, Renal failure, Atrioventricular (AV) node blocker, Shock and Hyperkalemia.

The pathophysiology leading to BRASH syndrome was described in different articles (Figure 5).

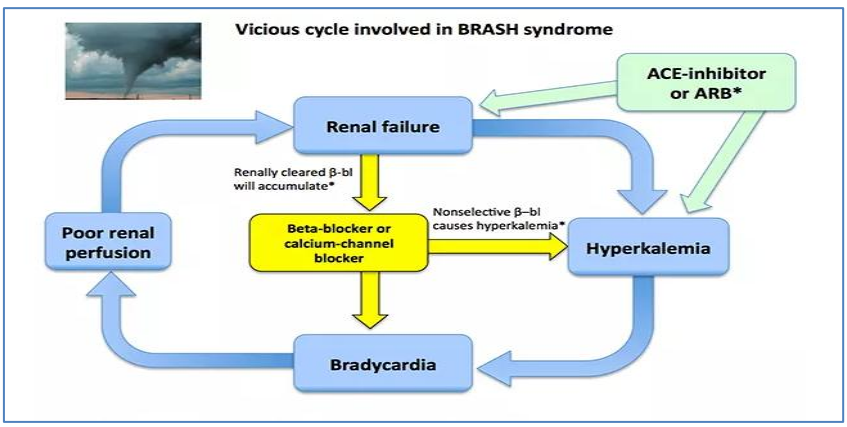

Fig-5: BRASH (Bradycardia, Renal failure, AV blockade, Shock, and Hyperkalemia) pathophysiology (7). ACE = angiotensin converting enzyme; ARB = angiotensin-receptor blocker [4-16]

BRASH syndrome is a rare entity. The cases linked to the use of betablockers and borderline renal function are reported in the articles listed below (Table 1) [5,6,7,8,9,10,11,12,13,14].

Table-1: Reported Cases of BRASH Syndrome [5, 6, 7, 8, 9, 10, 11, 12, 13, 14]

Age, gender	Medications involved	Potassium (mEq/L)	Creatinine (mg/dL)	Initial vitals	Treatments	Reference
70M	carvedilol valsartan, spironolactone	6.1	2.1	HR 38, Bp 86/50	IV calcium insulin / dextrose	Aziz 2011
76F	carvedilol spironolactone, ramipril	9.2	1.3	HR 28, Bp 120/59	Transvenous pacing insulin/glucose, bicarbonate	Erden 2010
78F	beta-blocker, ACE inhibitor calcium-channel blocker	7.9	2.1 (prior 1.1)	HR 33	Calcium, insulin furosemide, fluid	Unterman 2008
70M	metoprolol XL 100 mg enalapril, spironolactone	6.5	3.3	HR 44, Bp 100/56	Calcium, albuterol, kayexalate, transvenous pacing, dialysis	Isabel 2006
54F	atenolol 100 mg, diltiazem 300 mg irbesartan	6.4	160 uM	HR 22, Bp 60/30	External pacer, fluid, calcium, insulin	Bonvini 2006
57M	carvedilol 50 mg bid, digoxin spironolactone, fosinopril	6.8	2.7	HR 48, Bp 100/60		Vuckovic 2004
78M	metoprolol, lisinopril	7.5	8.5	HR 30, Bp 120/60	Transvenous pacing, calcium, furosemide, bicarbonate	Zimmers 2002
66F	verapamil SR 360 mg	7.1	6.1	HR 26, Bp 85/60	Isoproteranol, dopamine, calcium Bicarbonate, insulin, glucose	Vazquez 1996
75F	verapamil 120 mg TID captopril	6.9	2.4	HR 30, Bp 70/	Atropine, isoproterenol, calcium pacemaker	Jolly 1991
53M	verapamil 120 mg QID propranolol 40 mg QID	6.8	1.6	HR 32, Bp 70/	Isoproterenol, dopamine	Lee 1986

This syndrome is frequently underdiagnosed, leading to delayed treatment. Treatment of BRASH syndrome includes the stabilisation of the hemodynamic status with fluid resuscitation and vasopressors, and hyperkalemia therapies.

The BRASH syndrome in our patient was probably due to the association of beta blockers and digoxin. Within the patients presenting atrioventricual node block (AV block), high risk patients for developing a BRASH syndrome are those of advanced age, with moderate renal failure and episodes of dehydration. Prognosis is good with early recognition and management of this rare clinical entity as reported in Golchin and al paper of an 84-year-old man with a medical history of hypertension who presented with weakness and polyuria. The patient was on beta-blockers; the examination showed hypotension and bradycardia. Laboratory values revealed acute renal failure and hyperkalemia of 7.1. The patient was given intravenous calcium, intravenous fluids, and insulin with dextrose and put on dopamine drip. The patient received emergent dialysis with a good evolution [15].

CONCLUSION

BRASH syndrome is a process resulting in a combination of hyperkalemia and medications blocking the AV node.

This syndrome should be suggested in polymedicated old patients as it are frequently underdiagnosed, leading to delayed treatment. Timely diagnosis and early managment of this rare clinical entity enables better outcomes.

We insist on the fact that the injectable treatments must be available in Morocco to avoid the side effects of drug combinations potentially leading to complications such as BRASH syndrome.

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