Management of Atrial Fibrillation Leading to BRASH Syndrome: Case Report
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Abstract

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.

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INTRODUCTION

Brash syndrome is an acronym for Bradycardia, Renal failure, atrioventricular node blocker, shock and Hyperkalemia.

In this article, we report a case of Brash syndrome complicating the management 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) revealed an atrial fibrillation with a rapid ventricular response (Figure 1):

[Image: 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 parasetal 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 enoxaparin. 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
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 free 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.

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.

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).
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 values | Treatments                          | Reference     |
|-------------|-------------------------------------------|-------------------|--------------------|---------------|-------------------------------------|---------------|
| 70M         | candesartan, valsartan, spironolactone     | 6.1               | 2.1               | HR 38, 88/66/50 | IV calcium, insulin / dextrose       | Aziz 2011     |
| 76F         | candesartan, spironolactone, ramipril      | 9.2               | 1.3               | HR 28, 120/59  | Transvenous pacing, insulin/glucone, bicarbonate | Ender 2010   |
| 76F         | 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, 100/56  | Calcium, albuterol, furosemide, transvenous pacing, dialysis | Isabel 2006 |
| 56F         | aterozol 100 mg, diltiazem 300 mg, ibesartan | 6.4             | 160 uM            | HR 22, 60/50   | External pacer, fluid, calcium, insulin | Bonini 2006 |
| 57M         | candesartan 50 mg bid, digoxin, spironolactone, lisinopril | 6.8             | 2.7               | HR 48, 120/60  | Vackovic 2004                      |
| 78M         | metoprolol, lisinopril                     | 7.5               | 8.5               | HR 30, 120/60  | Transvenous pacing, calcium, furosemide, bicarbonate | Zimmer 2002 |
| 66F         | verapamil SR 360 mg                        | 7.1               | 6.1               | HR 26, 85/60   | Bisoprolol, dopamine, calcium, bicarbonate, insulin, glucose | Vaquez 1996 |
| 75F         | verapamil 120 mg TID captopril             | 6.9               | 2.2               | HR 30, 120/70  | Atropine, bisoprolol, calcium, pacemaker | Jolly 1991 |
| 53M         | verapamil 120 mg QID propranolol 40 mg QID | 6.8               | 1.6               | HR 32, 80/70   | Bisoprolol, 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 atrioventricular 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 management 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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