Management of Atrial Fibrillation Leading to BRASH Syndrome: Case Report
Ali Laalou*, Ikram Hazzazi, Nadia Charei, Mohammed El Jamili, Dounia Benzeroual, Saloua El Karimi, Mustapha El Hattaoui

Cardiology Department, Mohammed VI University Hospital, Marrakesh, Morocco

DOI: 10.36347/sasjm.2021.v07i04.006 | Received: 18.03.2021 | Accepted: 24.04.2021 | Published: 29.04.2021

*Corresponding author: Ali Laalou

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.

INTRODUCTION
Brash syndrome is an acronym for Bradycadria, Renal failure, atrioventricular node blocker, shock and Hyperkaliaemia.

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):

![Initial EKG showing atrial fibrillation at high ventricular rate](image)

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 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]

<table>
<thead>
<tr>
<th>Age</th>
<th>Gender</th>
<th>Medications involved</th>
<th>Potassium (mEq/L)</th>
<th>Creatinine (mg/dl)</th>
<th>Initial vitals</th>
<th>Treatments</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>70M</td>
<td>male</td>
<td>candesartan, valsartan, spironolactone</td>
<td>6.1</td>
<td>2.1</td>
<td>HR 38, 80</td>
<td>IV calcium, insulin / dextrose</td>
<td>Aziz 2011</td>
</tr>
<tr>
<td>76F</td>
<td>female</td>
<td>candesartan, spironolactone, ramipril</td>
<td>9.2</td>
<td>1.3</td>
<td>HR 28, 120</td>
<td>Transvenous pacing, insulin/glucose, bicarbonate</td>
<td>Erden 2010</td>
</tr>
<tr>
<td>78F</td>
<td>male</td>
<td>beta-blocker, ACE inhibitor calcium-channel blocker</td>
<td>7.9</td>
<td>2.1 (prior 1.1)</td>
<td>HR 33</td>
<td>Calcium, insulin furosemide, fluid</td>
<td>Unterman 2008</td>
</tr>
<tr>
<td>70M</td>
<td>male</td>
<td>metoprolol 100 mg, esmolol, spironolactone</td>
<td>6.5</td>
<td>3.3</td>
<td>HR 44, 100</td>
<td>Calcium, atropine, furosemide, transvenous pacing, dialysis</td>
<td>Isabel 2006</td>
</tr>
<tr>
<td>56F</td>
<td>female</td>
<td>atenolol 100 mg, dihydralazine 300 mg, furosemide</td>
<td>6.4</td>
<td>160 μM</td>
<td>HR 22, 80</td>
<td>External pacer, fluid, calcium, insulin</td>
<td>Bowlin 2006</td>
</tr>
<tr>
<td>57M</td>
<td>male</td>
<td>candesartan 50 mg b.i.d, digoxin spironolactone, fosinopril</td>
<td>6.8</td>
<td>2.7</td>
<td>HR 48, 120</td>
<td>Vackovic 2004</td>
<td></td>
</tr>
<tr>
<td>78M</td>
<td>male</td>
<td>metoprolol, lisinopril</td>
<td>7.5</td>
<td>8.5</td>
<td>HR 30, 120</td>
<td>Transvenous pacing, calcium, furosemide, bicarbonate</td>
<td>Zimmers 2002</td>
</tr>
<tr>
<td>66F</td>
<td>female</td>
<td>verapamil SR 360 mg</td>
<td>7.1</td>
<td>6.1</td>
<td>HR 26, 85</td>
<td>Atropine, isoproterenol, calcium bicarbonate, insulin, glucose</td>
<td>Vaquez 1996</td>
</tr>
<tr>
<td>75F</td>
<td>female</td>
<td>verapamil 120 mg, TID, captopril</td>
<td>6.9</td>
<td>2.4</td>
<td>HR 30, 80</td>
<td>Atropine, isoproterenol, calcium pacemaker</td>
<td>Joly 1991</td>
</tr>
<tr>
<td>53M</td>
<td>male</td>
<td>verapamil 120 mg, QID, propranolol 40 mg, QID</td>
<td>6.8</td>
<td>1.6</td>
<td>HR 32, 80</td>
<td>Isoproterenol, dopamine</td>
<td>Lee 1986</td>
</tr>
</tbody>
</table>

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 polyparedic 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.

REFERENCES

1. 2020 Guidelines for Management of Atrial Fibrillation. ESC Clinical Practice Guidelines
9. Isabel J, Champion JC. Junctional escape rhythm secondary to acute hyperkalemic renal failure in the
setting of concurrent beta-blocker therapy. JAAPA; 2006