

## Ashman Phenomenon

**Dr. O. S. Ogah** MBBS (Ib), Msc(Ib) FWACP, **Dr. E. H. Aikhuele** MBBS (Ib), and **Dr. B. T. Oyebola** MBBS (Ib)

Department of Medicine, Federal Medical Centre, Idi-Aba, Abeokuta, Ogun State, Nigeria.

### SUMMARY

Ashman phenomenon was first reported in 1947 as an aberrant ventricular conduction during atrial fibrillation. It occurs as a result of a change in the length of the QRS cycle. Ashman phenomenon has also been reported in atrial tachycardia and atrial ectopy. The commonest associated conduction abnormality is RBBB although association with LBBB has been documented. The condition is by itself asymptomatic and can be diagnosed by 12 lead ECG in most cases. It can easily be misdiagnosed as ventricular premature contraction. Management includes appropriate diagnosis and treatment of the cardiac disorders associated with it such as atrial fibrillation and atrial tachycardia. Isolated complexes do not require treatment.

**Key words:** Ashman phenomenon, arrhythmia, aberrant conduction.

### INTRODUCTION

Ashman phenomenon is an aberrant ventricular conduction during a trial fibrillation. It is due to a change in the length of QRS complex. It is most often mistaken as premature ventricular contraction or ventricular tachycardia. In this short review, we present a case history; clinical features, diagnostic criteria and management of this often misdiagnosed arrhythmia.

---

### All Correspondence to Dr. O. S. Ogah

---

Department of Medicine,  
Federal Medical Centre,  
Idi-Aba, Abeokuta,  
P.M.B. 3031 Sapon, Abeokuta.  
Ogun State. E-mail: osogah56156@yahoo.com.

### CASE HISTORY

A. J. A. is an 81 year old Nigerian man. He was admitted on account of progressive bilateral leg swelling, easy fatigability, orthopnoea, paroxysmal nocturnal dyspnoea and cough productive of frothy sputum. There was associated palpitation. He was a known hypertensive of nine (9) years duration.

Essential findings on clinical examination were an elderly man, dyspnoeic and tachypnoeic, not pale, afebrile and acyanosed. He had bilateral pitting pedal oedema up to the knees, scrotal and sacral oedema.

His respiratory rate was 28 cycles/minute and he had fine bibasal crepitations.

Cardiovascular examination revealed a pulse rate of 80 beats/min with a pulse deficit of 30 beats/min. His blood pressure was 140/60mmHg and the jugular venous pulsation was elevated up to the mid neck. The apex beat was not localizable and the first and second heart sounds were heard and irregular. There was no murmur. He also had hepatomegaly and moderate ascites.

His 12-lead ECG showed atrial fibrillation with an aberrant conduction (Ashman phenomenon, figure 1), which has a right bundle branch block pattern and occurred when a short R-R interval followed a long R-R interval. Echocardiography showed normal sized heart chambers, low normal LV systolic function (Ejection fraction 55%) and mild global hypokinesia. Chest radiograph revealed aortic unfolding. The serum electrolytes and urea were essentially within normal limits.

The patient improved on heart failure medications. He had rate control with oral digoxin and later cardioverted with oral amiodarone. The latest 12 lead ECG is shown in figure 2.

He is being followed up in our medical outpatient department.

It is an intraventricular conduction anomaly due to a change in the heart rate. This is because the

**Fig. 1:** 12 lead ECG showing Ashman phenomenon (Has a RBBB morphology and occurring when a short R-R interval follows a long R-R interval).

**Fig. 2:** The 12 lead ECG after pharmacological cardioversion

## **DISCUSSION**

Ashman phenomenon is an aberrant conduction of the ventricle as a result of a change in the QRS cycle length. It was first reported in 1947 by Gouaux and Ashman [1]. They described that in atrial fibrillation, when a relatively long cycle was followed by a relatively short cycle, the beat with a short cycle often has right bundle branch morphology. Aberrant conduction with a short-long cycle order has also been reported.

electrophysiological property of the heart is strongly affected by the heart rate, which on the other hand is modulated by drugs, electrolyte abnormalities as well as metabolic problems[2].

The duration of the refractory period of the heart muscle is proportional to the R-R interval of the preceding cycle. Thus shorter duration of action potential is associated with a short R-R interval and vice versa. A longer cycle will prolong the ensuing refractory period, and, if a shorter cycle follow t

beat terminating the cycle is likely to be conducted with aberrancy. And because the refractory period of the right bundle branch is longer than the left, it will still be in the refractory period when supraventricular impulse reaches the His-Purkinje system resulting in a complex with right bundle branch morphology [2, 3].

Ashman phenomenon is a common ECG finding in the setting of atrial fibrillation and is often misdiagnosed as premature ventricular complex. It is related to the underlying heart disease. No geographical variation has been reported [4, 5].

Clinically, it is by itself asymptomatic. If symptoms are present, it is related to the underlying heart disease but not as a result of the aberrancy. No specific physical findings have been described for this condition although when it occurs in the presence of atrial fibrillation, irregularly irregular pulse, pulse deficit or fast heart rate may be seen.

Clinical conditions that will give rise to Ashman phenomenon are those leading to altered duration of the myocardial refractory period. These will include atrial fibrillation, atrial tachycardia and atrial ectopy [6, 7].

The differential diagnosis includes ventricular premature complexes and ventricular tachycardia (series of consecutive aberrantly conducted supraventricular complexes). It is important to understand Ashman phenomenon because it will be useful in differentiating aberrantly conducted supraventricular impulses from wide complex arrhythmia of ventricular origin as their prognosis and treatment are entirely different [6, 7].

Ashman phenomenon is principally diagnosed by 12 lead surface ECG. But in difficult cases, and where facilities are available, invasive electrophysiological studies will be required to establish the source of an arrhythmia whether ventricular or supraventricular [4, 5, 8, 9, 10, 11].

In 1983, Fisch published a set of diagnostic criteria for Ashman phenomenon [12]. These include:

1. A relatively long cycle immediately preceding the cycle terminated by the aberrant QRS complex: A short-long-short interval is even more likely to initiate aberration. Aberration can be LBBB and

RBBB even in the same patient.

2. RBBB- form aberrancy with normal orientation of the initial QRS vector: concealed perpetuation of aberration is possible, such that a series of wide QRS supraventricular beats is possible.
3. Irregular coupling of aberrant QRS complexes.
4. Lack of a fully compensatory pause (which is never seen in atrial fibrillation).

Presence of Ashman phenomenon does not really differentiate aberrancy versus ventricular rhythm. A morphological feature that favours the diagnosis of arrhythmic of ventricular origin is showing in Table 1 [13].

**TABLE 1:** Morphological Features that favour the Diagnosis of Wide Complex Arrhythmias of Ventricular Origin.

- 
- Left bundle branch morphology with notched or slurred down stroke in V1 or V2
  - QS pattern in V6
  - Right bundle branch morphology with monophasic R, biphasic QRS, or rSR<sup>l</sup> pattern in V1.
  - QRS duration > 140ms in RBBB morphology and > 160ms in LBBB morphology.
  - R-to-S interval > 100ms in a precordial lead.
  - Marked Left axis deviation (-90 and 180)
- 

Treatment of Ashman phenomenon involves the treatment of the cardiac condition causing it for example atrial fibrillation and atrial tachycardia. An isolated complex of Ashman phenomenon does not warrant any treatment.

## REFERENCES

1. Gouaux JL, Ashman R. Auricular fibrillation with aberration simulating ventricular paroxysmal tachycardia. *Am Heart J* 1947; 34: 366.

2. Nave C, Nardi S, Gaudino M, Curcio N, Cirillo T and Iacono A. [The electrophysiological basis of aberrant intraventricular conduction during atrial fibrillation]. *Cardiologia* 1996; 41(12): 1193-1198.
3. Schamroth L and Jacobs ML. A study in intracardiac conduction with special reference to the Ashman phenomenon. *Heart Lung* 1982; 11(4): 381-382.
4. Kennedy LB, Leefe W and Leslie BR. The Ashman phenomenon. *J La State Med Soc* 2004; 156(3):159-62.
5. Nelson WP, and Fletcher RD. 11—The “Ashman phenomenon”. *Med Times* 1978; 106(9): 96-100.
6. Heimonas ET, Cokkinos DV, Hatzivasiloglou C, Papoulis S, Ioannou N, and Chaniotakis M. A study of the factors influencing the appearance of the Ashman phenomenon. *Acta Cardiol* 1982; 37(3): 175-181.
7. Chung EK. Atrial fibrillation with aberrant ventricular conduction. *W V Med J* 1970; 66(7): 227-228.
8. Akiyama T, Richeson JF, Faillace RT, Lockhart J and Scherer JC. Ashman phenomenon of the T wave. *Am J Cardiol* 1989; 63(12): 886-890.
9. Bellanca G, Cataldo C, D’Antonio E, Di Stefano P, Picarella B, and Terzo S. [Atrial fibrillation with high ventricular rate with aberrant conduction simulating ectopic ventricular tachycardia. Presentation of a case]. *Minerva Cardioangiol* 1969; 17(12): 1132-1138.
10. Quaal S and Schamroth L. Aberrant ventricular conduction during atrial fibrillation. *Heart Lung* 1985; 14(1): 101.
11. Spodick DH. Electrocardiology teacher analysis and review: 4:3 atrioventricular wenckebach exit block with (probable) Ashman phenomenon during junctional tachycardia. *Am J Geriatr Cardiol* 2004; 13(5): 285.
12. Fisch C. Electrocardiography of arrhythmias:from deductive analysis to laboratory confirmation—twenty years of progress. *J Am Coll Cardiol* 1983;1(1):306-16.
13. Marriot HJL and Sandler JA. Criteria, old and new, for differentiating between ectopic ventricular beats and aberrant ventricular conduction in the presence of atrial fibrillation. *Prog Cardiovasc Dis* 1966; 9: 18.