

## Case Report

# Hypokinetic Arrhythmias Arising after Several Months from ATC for Complex Atrial Arrhythmia (Atrial Fibrillation and Atrial Flutter)

Nicola Alessandri\*, Claudia Iannone, Angela Dei Giudici, Emiliano Coletta, Antonella Maddaluno, Flavia Tersigni, Michela D'Ascanio, Flavio Moscariello, Fabrizio Tufano and Bich Lien Nguyen

Università "Sapienza" Roma, Facoltà di Farmacia e Medicina (polo pontino), Via della Moletta 17 Roma -00154- Italy

## \*Corresponding author

Alessandri Nicola, Università "Sapienza" Roma, Facoltà di Farmacia e Medicina (polo pontino), Via della Moletta 17 Roma -00154- Italy, Tel: 06-5755115; Fax: 0773-6513329; Email: alessandrinicola@libero.it

Submitted: 20 February 2014

Accepted: 26 February 2014

Published: 28 February 2014

ISSN: 2333-6676

## Copyright

© 2014 Alessandri et al.

## OPEN ACCESS

## Keywords

- Catheter ablation
- Electro-anatomic mapping
- Atrial fibrillation
- Atrial flutter
- Definitive pacemaker implantation

## Abstract

In this article are reported hypokinetic arrhythmias (asystole or AVB 3°) arising after several months from ablation procedure that needed definitive pacemaker implantation. These events, coming in patients submitted to ablation of left atrial roof and cavotricuspid isthmus area could be considered, primarily, complications of ablation. However, their appearance long time after the RF-TCA let us to consider them autonomic and independent events.

The observation of these events opens a patho-physiological discussion in relation to current knowledge and we suggest these three hypothesis: a) "Electric remodelling as a result of ablation procedure"; b) "Development of the natural history of AF"; c) "Random observation".

## ABBREVIATIONS

RF-TCA: Radiofrequency Transcatheter Ablation; AF: Atrial Fibrillation; PM: Pacemaker; PT: Patient; pAF: Persistent AF; TIA: Transient Ischemic Attack; PFO: Patent Foramen Ovale; EPS: Electrophysiology Study; AV: Atrium-Ventricular; ECG: Electrocardiogram; CTI: Cavotricuspid isthmus; ECV: Electric Cardioversion; PSVT: Paroxysmal Supraventricular Tachycardia; PV: Pulmonary Veins; SSS: Sick Sinus Syndrome

## INTRODUCTION

Radiofrequency Transcatheter Ablation (RF-TCA) is one of the new strategies used for the treatment of Atrial Fibrillation (AF), its success rate ranges from 32% to 90% according to the kind of AF, the site that received radiofrequency and the therapeutic protocol used [1-4]. Early (< 7 days after the procedure) and late (> 7 < 60 days from the day of the ablation) complications after RF-TCA are known; their incidence ranges from 0,8% to 5,2% according to the ablation technique used [5]. In this article, we describe hypokinetic arrhythmias coming 11,16 ± 4,6 months after ablation procedure, that needed definitive Pacemaker

(PM) implantation. This event, coming after a RF-TCA could be considered complications of ablation. However, their appearance long time after the RF-TCA let us to consider them autonomic and independent events. The incidence and coincidence event stimulated a patho-physiological discussion in relation to current knowledge. Except the events arising after AF ablation, two other cases of arrhythmia, arising after flutter ablation, are reported to complete this topic.

Below we report the clinical history of the 6 patients (pt) under discussion.

## CASE PRESENTATION

## Case No. 1

A 59 year old man, affected by Persistent AF (pAF), with Transient Ischemic Attack (TIA), vascular cerebral disease, Patent Foramen Ovale (PFO), multinodular thyroid disease, was admitted (09/2010) because of pAF (EHRA III). An Electrophysiology Study (EPS) was performed and showed "AV node Wenckebach 450 ms, AV node refractory time 400-350-190 ms, after isoproterenol

**Table 1:** Characteristics of the patients, ablation technique used and electric anomaly onset after ATC-RF.

Pt	Sex	Age	Comorbidities	Arrhythmia	Target ATC	RF (W)	Early Compl.	Late C.ompl.	Symptoms	Time post ATC	Electric anomaly
CP	M	59	TIA Hypertension High homocisteine	PAF	VP Circ. Abl.	25W	/	/	Syncope with trauma	8	Pause 12 sec. + AVB III
MA	M	82	Hypertension	AF-Flutter	CTI	45W	/	/	asthenia + dizziness	16	SAB II FC<30 bpm
ST	F	62	Hypertension Tyrhoid disease	PAF	VP. Circ. Abl with left sup ganglia	25W	/	/	asthenia + dizziness	15	Pause 5 sec.
VE	F	76	Hypertension TIA TEA carotideo dx MD type II Dyslipidemia	PAF	VP. Circ. Abl with right sup ganglia	25W	/	/	Syncope with trauma	5	Pause 7,5 sec.
DLA	F	78	Hypertension Dyslipidemia Tyrhoid disease M-A Val. disease	Flutter	CTI	40 W	/	/	Syncope with trauma	7	AV node rhythm < 35 bpm
SM	M	79	PHD A.Val. Disease PAD	Flutter	CTI	40 W	/	/	asthenia + dyspnea	6	AVB III

**Abbreviations:** A= Aortic; Abl= Ablation; AVB= Atrio-Ventricular Block; Compl= Complications; Circ= Circumferential; CTI= Cavotricuspid Isthmus; M= Mitral; MD= Mellitus Diabetes Type II; PAD= Peripheral Artery Disease; PAF= Parossistic Atrial Fibrillation; PHD= Pulmonary Heart Disease; PV = Pulmonary Disease; SAB= Sinus-Atrial Block; TEA= Thromboendarterectomy; TIA= Transient Ischemic Attack.

iv administration and right atrial burst 180 ms, ectopic beats came from right pulmonary veins and became AF with an high ventricular response." Radiofrequency energy was erogated by a 4 mm catheter ablation (Safire), M curve, obtaining encircling of pulmonary veins. Biphasic synchronized shock of 130 J had success to convert to sinus rhythm, 70 bpm.

He was discharged with the indication to follow the therapeutic protocol with flecainide, sotalol, atorvastatin, omega fatty acids, acenocumarol. He was in sinus rhythm at 1,3,6 months of follow up. During the 8th month of follow up, after TCA, he had a syncope. Holter Electrocardiogram (ECG) showed an advanced AV block, with a RR distance of 11 seconds. He had a definitive PM implantation (St. Jude Medical PM). He resulted asymptomatic and in sinus rhythm at 12, 15, 18 and 24 months follow up.

### Case No. 2

A 82 year old man, affected by Persistent AF (pAF) (first episode in 2005), with hypertension, thyroid disease treated with levothyroxine, was admitted (10/2011) because of pAF (EHRA II). An electrophysiology study was performed and showed "Sinus node recovery time (600 ms): 1387 ms, corrected 183 ms; (700 ms) 1747 ms, corrected 488 ms; (800 ms) 1664 ms, corrected 364 ms. 220 ms atrial burst, with isoproterenol iv induced a Cavotricuspid Isthmus (CTI) dependent atrial flutter (cycle 247 ms, HR 110 bpm), confirmed by an entrainment which became a left atrial flutter and then AF." Radiofrequency energy was erogated in the target point of CTI. He was discharged with the indication to follow the therapeutic protocol with flecainide and sotalol for 3 months. He had a good life until February 2013 (16 months after the ablation), then he had different episodes of

dizziness and lightheadness. He had an EPS which showed "Sinus node recovery time (600 ms): 1965 ms, corrected 651 ms; (700 ms) 1780 ms, corrected 380 ms; (800 ms) 1590 ms, corrected 340 ms. AV node Wenckebach 450 ms". An atrial- sinus disease was diagnosed. He had a definitive PM implantation (St. Jude Medical PM). He is asymptomatic and in sinus rhythm now.

### Case No 3

A 62 year old woman has affected by persistent AF (pAF) for 5 year, with hypertension. In the past (05/2006) she had a slow-fast node ablation. On 2010, she was admitted because of pAF (EHRA III). She had an EPS and then she was ablated, obtaining encircling of pulmonary veins and ablation of left superior ganglia plexi. She was discharged in sinus rhythm with an indication to follow the therapeutic protocol with flecainide, sotalol, atorvastatin, omega fatty acids, acenocumarol. She was in sinus rhythm at 1,3,6 months of follow up (EHRA I). After 15 months from the TCA, she had another episode of AF. She was admitted and an electric cardioversion (ECV) was performed with success to recover sinus rhythm. After the ECV, she had asthenia and lightheadness, and we discovered in the monitor the appearance of 5 s pause. After adequate therapy and temporary PM due to the persisting of pauses, she had a definitive PM implantation. She is asymptomatic and in sinus rhythm now.

### Case No 4

A 62 years old woman, affected by p AF, hypertension, mellitus diabetes type 2, TIA, PFO with a no significant shunt, thromboendarterectomy right carotid artery in 2008. In the past (2009) she was admitted to our cardiology department due to

syncope, sometimes reporting trauma. In July 2010 she was admitted because of pAF (EHRA III) and a EPS was performed. She received RF-TCA and encircling of pulmonary veins, atrial roof and right superior ganglia plexi were ablated. She was discharged in AF with an indication to follow the therapeutic protocol with: flecainide, sotalol, atorvastatin, omega fatty acids, acenocumarol.

She was in sinus rhythm at 1,3 months of follow up with EHRA I class. During the 5th month of follow up after TCA, he had a syncope. She was studied and PSVT was documented. It became an AF and then it converted in Sinus rhythm, stopped by a sinus arrest of 7,3 s. She had a definitive PM implantation 5 months after the ablation. She is asymptomatic and in sinus rhythm now.

#### Case No 5

A 78 year old woman, affected by hypertension, dyslipidemia, thyroid disease. She had a cardiac surgery in 2008 to implant a bio-prosthesis in aortic position and to have a mitral plastic with a valvular ring. She was admitted in an hospital because of atrial flutter with high ventricular response in December 2012. An EPS was performed and radiofrequency energy was erogated in the left target side of CTI and arrhythmias was stopped.. After 7 months from the ablation, she was admitted in our cardiology department because of lypothymy. She had a wash out time from the therapy. Then a sinptomatic junctional escape rhythm, with HR < 30 bpm was diagnosed. She had a definitive PM implantation. She is asymptomatic and in sinus rhythm now.

#### Case No 6

A 79 year old man, with an aortic prosthesis was admitted in 12/2012 because of symptomatic atrial flutter. An EPS was performed and showed "Normal sinus node recovery time, AV node Wenckebach 500 ms, AV node refractory 550/400 ms" Radiofrequency energy was erogated in the target side of CTI and arrhythmias was stopped. He was discharged with the following therapy: amiodarone and atorvastatin.. During the follow up at 1 and 3 months he was asymptomatic and in sinus rhythm. After 6 months from the ablation he was admitted because of lypothymy. His ECG Holter showed a first grade AV block (PQ duration 400-420 ms), different daily pauses (> 2,5 < 3,1s), sometimes junctional escape rhythm. He had an EPS which showed "a third grade AV block induced by HR 80". He had a definitive PM implantation. He is asymptomatic and in sinus rhythm now.

## MATERIAL AND METHODS

### EPS before ablation

An EPS was performed in every pt (guidelines indication): Seldinger technique was used to introduce catheters (8F e 6F) from right femoral vein; 7F from right internal jugular vein and 6F from left femoral vein. The catheters were placed as following: CSL 10p in the coronary sinus; CRD on the His bundle, JSN in the right atrium. After heparin was administered, maintaining an ACT>300s, SL1 was introduced in left atrium using a ETE guided transseptal approach with BRK needle.

In case tachyarrhythmias were not spontaneous, isoproterenol (1 to 5 ug/min) was administered iv to induce them. Programmed right atrial stimulations were used to trigger AF. Left atrium was mapped to recognize the most early activation point triggering the tachyarrhythmias.

### Mapping system

Non fluoroscopic EnSite NavX St. Jude Medical mapping system was used to have a 3D left atrium geometry.

### RF-ATC

Triggers were ablated using radiofrequency energy and irrigated ablation catheter(Safire St. Jude Medical, Minnesota, MNNSA) 4 mm tip, M curve, (average power 33 W ± 9 DS, target temperature 55°C). RF created a tissue injury with coagulative necrosis.

### ATC technique

Pulmonary Veins (PV) antral isolation, left atrial roof linear lesions and lesion to isthmo between mitral anulus and left inferior PV were performer. Sometimes, after vagal reflexes were stimulated, RF was erogated in ganglia (left superior and right superior). Post ablation testing with coronary sinus stimulation 500 ms confirmed the absence of the arrhythmias and circular catheter Optima 25-15 confirmed the isolation of the pulmonary veins.

### Follow-up after-ATC

All the pts had a clinical and strumental 1, 3, 6, 12, 18 and 24 month follow up. The first consisted of: analysis of symptoms (EHRA score), lifestyle, examination and therapeutic protocol modifications according to pt clinic characteristics. The second one consisted of: ECG, Monitoring Holter ECG and trans-thoracic echocardiogram.

After ATC therapy: Therapeutic protocol with flecainide (50 mg x 2), sotalol (20 mg x 3), atorvastatin (80 mg), omega fatty acids (1000 mgx3) and acenocumarol was followed for 3 months.

### The implant procedure for PaceMaker

The procedure was performed under local anesthesia and creating a pocket in the upper chest, under left clavicle region. A bipolar catheter was introduced in the apical septal right ventricle through the left cephalic vein; a bipolar catheter was introduced in the right atrial auricle through the left axillary vein. The bicameral PM was inserted in the pocket beneath the skin.

### After PM controls

All the pts were visited to control the incision healing and the PM on the seventh day, and to repeat PM control and echocardiogram at 1-3-6 months.

## DISCUSSION

ATC-RF is a therapeutic approach reporting high success rate, but early and late complications are described. Focusing our attention on early (< 7 days after ATC) arrhythmic complications, Bezold Jarish reflex could be the pathogenetic mechanism of the ipokinetic arrhythmias. It consists of a vagal stimulation and an adrenergic inhibition, after cardiac mechanoreceptors activation, with symptoms related to low heart rate, low blood pressure due to vasodilatation [6-8] (Table).

Reversible atrial ventricular block after RF erogation, with a slowing down of the heart rate until to temporary asystole, is caused by a neurocardiogenic reflex. The effects

of the autonomic disfunction during RF ablation used to treat supraventricular arrhythmias has been described by different authors [9]. Another early complication during EPS is linked to the endocardial stimulation of autonomic ganglia which leads to immediate vagal response, such as RR decline, asistole and AV block. These responses disappear after isolation of pulmonary veins [10]. AV advanced block, sometimes irreversible, is one of the complications after ablation of cavotricuspid-isthmus-dependent atrial flutter. It is caused by direct RF injury to the CTI, or abnormal flogistic response in the area near AV node, or an acute occlusion of the right coronary artery [11]. Instead the reversible block is caused by an increased vagal tone due to pain or stimulation of epicardial vagal fibres or ganglia. To our knowledge the arrhythmias, arisen during RF erogation, or until 30 days after the procedure, or until 60 days after ATC have a pathogenesis linked to the procedure (technique, time and power).

The patients described in this study are, just in appearance, affected by autonomic and independent ipokinetic arrhythmias. After a deep study, we found out that a RF-ATC was performed to treat refractory to pharmacological therapy supraventricular arrhythmias (4 AF and 2 Flutter) with a EHRA II-III class, a lot of months before the arising of the events. Definitive PM implantation was performed after  $9,5 \pm 4,7$  months from the RF-ATC procedure. The brady-arrhythmias cannot be explain as a result of RF-ablation, because the late onset ( $9,5 \pm 4,7$  months) is too much to think to a isto-morpho-anatomic correlation.

This report consists of an heterogeneous group of 6 different arrhythmias: 6 cases of pAF and 2 cases of flutter, different ablated areas (the left atrial roof and ICT respectively) and technique used; all the pts shared RF-ATC and the late bradyarrhythmia onset.

A) Based on this observation we suggest three different hypothesis: "Electric remodelling as a result of ablation procedure"

B) "Development of the natural history of AF"

C) "Random observation".

A) RF erogated during ablation is responsible for a modification of the autonomic fibers and of the anatomic substrate. The close relation between Pulmonary Veins (PV) and ganglionated plexi shows that RF erogation causes not only pulmonary vein disconnection, but also a modification of the autonomic vagal tone [12]. Preganglionic parasympathetic vagal fibers to sinus atrial node, reach fatty tissue near PV, fibers to AV node terminate in fatty tissue near inferior vein cava [13]. These areas, full of afferent vagal receptors, are ablated during the ATC procedure to treat AF and isthmo- cavo-tricuspid dependent Flutter. Furthermore, "C fibers", which induce bradycardia and hypotension, are located in the ostium of the 4 PV, in the roof of left atrium, in the lateral right atrium and in the left posterior ventricle [14]. The RF erogation, basing on power and timing, induces a flogistic reaction characterized by interstitial oedema, microhemorrhage with coagulative necrosis, a flogistic cellular response which resolve in fibrotic tissue; this response depend on

the characteristic of the pt. RF erogation could create a subclinical injury on autonomic cardiac fibers, although not so important to induce early bradyarrhythmias, on anatomic substrate with modifications that induce late atrial conduction for AF (increased critic mass) and late atrium-ventricular conduction (Bundle of His A-H-V-V); as a result of this modification could be lose the physiological ability to maintain SR [15,16].

B) The late onset of the arrhythmias, months after the ATC procedure, could be the evolution of the natural history of the complex arrhythmia AF, due to degenerative sclerotic myocardium. It is described the close relation between AF and sick sinus syndrome (SSS): it is caused by both, the reduced ability of the sinus node to rule and synchronize electric heart activity and the electric-anatomic atrial remodeling in AF [17-21]. Bradycardia due to SSS and structural (critical mass), electrical (focus, rotors, triggers), functional (Autonomic Nervous System) modifications support the tachyarrhythmia onset with RR variability. The RF-ATC aims to eliminate primary source and ectopic focus of AF and their spread, altering atrial structure. The suppression of the arrhythmia could let the sick sinus node to rule again. Can be a degenerative fibrotic sick node the physiological pacemaker? This phenomenon, also diffused to AV node, could explain the onset of bradyarrhythmias of our patients.

C) The third hypothesis is the possibility of "accidental coincidence" or "random observation".

These arrhythmias could not have connections with AF disease and could not be the consequence of a previous procedure technique. They could be just an accidental observation due to an accurate follow-up after ATC. The arrhythmias after ATC developed in 11 % of our population, versus 2% of the percentage linked to a random observation.

So it is difficult thinking to an absence of linkage between the previous ablative procedure and the onset of the arrhythmia.

## CONCLUSION

SSS and AV node disease developed in our 6 pts cannot be direct complications of RF-ATC, on the contrary of the early or late bradyarrhythmias described in literature. Io our opinion these arrhythmias are caused by the unsuccessful heart to maintain an artificial balance after ATC. The histological heart remodeling after the inflammation and an unbalance on ANS induced by RF enhance "critic mass". Sometimes, the inability to find a good balance between these factors, determines the onset of arrhythmias in special pts.

An accurate analysis of the individual anatomy and of the risk factors, associated with a limited RF energy erogation are not able to prevent this specific response. Instead a deep follow-up after ATC could prevent permanent injuries and this kind complications.

## REFERENCES

1. Pappone C, Augello G, Sala S, Gugliotta F, Vicedomini G, Gulletta S, et al. A randomized trial of circumferential pulmonary vein ablation

- versus antiarrhythmic drug therapy in paroxysmal atrial fibrillation: the APAF Study. *J Am Coll Cardiol*. 2006; 48: 2340-2347.
2. Jaïs P, Cauchemez B, Macle L, Daoud E, Khairy P, Subbiah R, et al. Catheter ablation versus antiarrhythmic drugs for atrial fibrillation: the A4 study. *Circulation*. 2008; 118: 2498-2505.
  3. Cappato R, Calkins H, Chen SA, Davies W, Iesaka Y, Kalman J et al. Updated worldwide survey on the methods, efficacy, and safety of catheter ablation for human atrial fibrillation. *Circ Arrhythm Electrophysiol* 2010; 3: 32-38.
  4. Brooks AG, Stiles MK, Laborderie J, Lau DH, Kuklik P, Shipp NJ, et al. Outcomes of long-standing persistent atrial fibrillation ablation: a systematic review. *Heart Rhythm*. 2010; 7: 835-846.
  5. Calkins H, Brugada J, Packer DL, Cappato R, Chen SA, Crijns HJ, et al. HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: recommendations for personnel, policy, procedures and follow-up. A report of the Heart Rhythm Society (HRS) Task Force on Catheter and Surgical Ablation of Atrial Fibrillation developed in partnership with the European Heart Rhythm Association (EHRA) and the European Cardiac Arrhythmia Society (ECAS); in collaboration with the American College of Cardiology (ACC), American Heart Association (AHA), and the Society of Thoracic Surgeons (STS). Endorsed and approved by the governing bodies of the American College of Cardiology, the American Heart Association, the European Cardiac Arrhythmia Society, the European Heart Rhythm Association, the Society of Thoracic Surgeons, and the Heart Rhythm Society *Europace*. 2007; 9: 335-379.
  6. Tsai CF, Chen SA, Tai CT, Chiou CW, Prakash VS, Yu WC, et al. Bezold-Jarisch-like reflex during radiofrequency ablation of the pulmonary vein tissues in patients with paroxysmal focal atrial fibrillation. *J Cardiovasc Electrophysiol*. 1999; 10: 27-35.
  7. Duru F, Bauersfeld U, Candinas R. Autonomic effects of radiofrequency catheter ablation. *Europace*. 2000; 2: 181-185.
  8. Friedman PL, Stevenson WG, Kocovic DZ. Autonomic dysfunction after catheter ablation. *J Cardiovasc Electrophysiol*. 1996; 7: 450-459.
  9. Friedman PL, Stevenson WG, Kocovic DZ. Autonomic dysfunction after catheter ablation. *J Cardiovasc Electrophysiol*. 1996; 7: 450-459.
  10. Verma A, Saliba WI, Lakkireddy D, Burkhardt JD, Cummings JE, Wazni OM et al. Vagal responses induced by endocardial left atrial autonomic ganglion stimulation before and after pulmonary vein antrum isolation for atrial fibrillation. *Heart Rhythm*. 2007; 4: 1177-1182.
  11. Bernard Belhassen, Aharon Glick, Raphael Rosso, Yoav Michowitz, Sami Viskin. Atrioventricular block during radiofrequency catheter ablation of atrial flutter: incidence, mechanism, and clinical implications. *Europace*. 2011; 13: 1009-1014.
  12. Raviele A, Natale A, Calkins H, Camm JA, Cappato R, Ann Chen S, et al. Venice chart international consensus document on atrial fibrillation ablation: 2011 update. *Journal of cardiovascular electrophysiology*. 2012; 23: 890-923.
  13. Pappone C, Santinelli V, Manguso F, Vicedomini G, Gugliotta F, Augello G, et al. Pulmonary vein denervation enhances long-term benefit after circumferential ablation for paroxysmal atrial fibrillation. *Circulation*. 2004; 109: 327-334.
  14. Marron K, Wharton J, Sheppard MN, Fagan D, Royston D, Kuhn DM, et al. Distribution, morphology, and neurochemistry of endocardial and epicardial nerve terminal arborizations in the human heart. *Circulation*. 1995; 92: 2343-2351.
  15. Nath S, Wayne JG, Kaul S, Goodman NC, Jayaweera AR, Haines DE. Effects of radiofrequency catheter ablation on regional myocardial blood flow. Possible mechanism for late electrophysiological outcome. *Circulation*. 1994; 89: 2667-2672.
  16. Canpolat U, Ařahiner L, Aytimir K, Oto A. Recovery of atrioventricular block with teophylline and methylprednisolone occurring few days after slow pathway radiofrequency ablation. *Int J Cardiol*. 2012; 160: e33-34.
  17. Engelmann MD, Svendsen JH. Inflammation in the genesis and perpetuation of atrial fibrillation. *Eur Heart J*. 2005; 26: 2083-2092.
  18. Everett TH 4th, Olgin JE. Atrial fibrosis and the mechanisms of atrial fibrillation. *Heart Rhythm*. 2007; 4: S24-27.
  19. Aldhoon B, Melenovský V, Peichl P, Kautzner J. New insights into mechanisms of atrial fibrillation. *Physiol Res*. 2010; 59: 1-12.
  20. Hocini M, Sanders P, Deisenhofer I, Jaïs P, Hsu LF, Scavée C, et al. Reverse remodeling of sinus node function after catheter ablation of atrial fibrillation in patients with prolonged sinus pauses. *Circulation*. 2003; 108: 1172-1175.
  21. Khaykin Y, Marrouche NF, Martin DO, Saliba W, Schweikert R, Wexman M, et al. Pulmonary vein isolation for atrial fibrillation in patients with symptomatic sinus bradycardia or pauses. *J Cardiovasc Electrophysiol*. 2004; 15: 784-789.

#### Cite this article

Alessandri N, Iannone C, Dei Giudici A, Coletta E, Maddaluno A, et al. (2014) Hypokinetic Arrhythmias Arising after Several Months from ATC for Complex Atrial Arrhythmia (Atrial Fibrillation and Atrial Flutter). *J Cardiol Clin Res* 2(3): 1030.