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Cardioversion In Acute Atrial Fibrillation Without Anticoagulation

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Abstract

A major concern in cardioversion of newly detected atrial fibrillation is the risk of thromboembolic events. The vast majority of these events occur in the first week following cardioversion. Transesophageal echocardiography has demonstrated that thrombus and dense spontaneous echo contrast may occur in the left atrium and left atrial appendage in patients with acute atrial fibrillation (<48 hours) scheduled for cardioversion. Moreover, atrial function may become impaired immediately following successful cardioversion. The risk of thromboembolic events increases with the presence of stroke risk factors, such as heart failure, hypertension, diabetes, prior stroke, female sex and age above 65-75 years. Thus, the current guidelines of the ESC and ACC/AHA/Heart Rhythm Society recommend that patients with acute atrial fibrillation should undergo cardioversion under cover of unfractionated or low-molecular weight heparin followed by oral anticoagulation for at least 4 weeks in patients in patients at moderate-to-high risk for stroke. In line with the guidelines, new evidence from a large patient population suggests that after successful cardioversion of acute atrial fibrillation, patients have a low overall risk of thromboembolic events without any anticoagulation when they have no risk factors for thromboembolism. In contrast, the risk is in the range of 10% in patients with multiple classic risk factors for thromboembolism.

Thromboembolic Risk during Cardioversion of AF

A major concern in the management of AF is the risk of thromboembolic events during cardioversion of newly detected AF. The risk appears similar with either pharmacological or electrical cardioversion.[1] Previously, 6 small retrospective studies have explored the risk in a total of 1 471 patients who did not receive anticoagulation after cardioversion of acute AF.[2,3,4-5-7] In those studies, the incidence of thromboembolic events was low (0-0.9%) and all definite events occurred in elderly (age above 75 years) women and after spontaneous restoration of sinus rhythm .[2,3,4-5-7]

The recently published multicenter FinCV Study provided new insight into this clinically important "gray zone", reporting data on 5,116 successful cardioversions performed without peri-procedural and post-cardioversion anticoagulation in a total of 2,481 patients with AF lasting <48 hours.[8] In this study, we observed 38 definite embolic events within 30 days after cardioversion, corresponding to an incidence of 0.7%, and additionally 4 transient ischemic attacks.[8] The median delay was 2 days from cardioversion confirming that the first few days after cardioversion are the ones that carry the highest risk, and deserve the greatest attention. Significant independent predictors of definite embolic events were old age, female sex, heart failure, and diabetes. Accumulation of these risk factors lead to an unacceptably high risk of 9.8% in patients with heart failure and diabetes, compared with only 0.2% in those with no such risk factors.[8] Of note, the predictors of embolic events in the acute setting were the ones that also predict thromboembolic complications in the general AF population. Table 1 summarizes the thromboembolic complications in patients with acute AF who received no anticoagulation after cardioversion in previous studies

Background of Thromboembolism in AF

Most embolic events occur shortly after successful cardioversion which supports the view that conversion of AF to sinus rhythm is responsible for thromboembolism, even after short attacks of AF .[9] Thrombus formation in the left atrial appendage is usually responsible for the thromboembolic complications associated with AF. It was shown that platelet activation and increased thrombin generation in the left atrium start to develop as early as 15 minutes following induction of AF.[10] Transesophageal echocardiography has shown that left atrial thrombi - a clear contraindication to cardioversion - are found in 4% of the patients already < 48 hours of AF, and in 14% of patients when the duration is < 72 hours when no anticoagulation is used.[11,12] Importantly, however, the absence of thrombus before cardioversion does not exclude thromboembolic complications, since restoration of sinus rhythm often results in atrial stunning, and a decrease of flow velocities in the left atrial appendage.[13] It is known that atrial stunning promotes new thrombus formation and predisposes to embolization. Our recent observations on the time course of thromboembolism support the view that temporary atrial stunning may also occur after successful cardioversion of short attacks

Disclosures:
None.

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Table 1: Thromboembolic complications in patients with acute (<48 hours) atrial fibrillation who received no anticoagulation after cardioversion in previous studies

Authors	Number of patients	Mean age	Male gender	Success rate	Thromboembolism
Weigner et al. 1997 [2]	224	68	NA	95%	0.9%*
Michael et al.1999 [7]	217	64	54	86%	0.5
Burton et al. 2004 [4]	314	61	55	86%	0†
Gallagher et al. 2002 [3]	198	63	68	100%	0.5%‡
Stiell et al. 2010 [5]	414	65	56	92%	0
Xavier et al. 2010 [6]	104	57	92	96%	0
Airaksinen et al. 2013 [8]	3143&	61	64	95%	0.7%

*All 3 thromboembolic events after spontaneous cardioversion and in old (>75 years) ladies; †Follow-up of 7 days; ‡Plus one probable thromboembolic event; &7660 cardioversions

of AF, and predispose to embolism, especially when the patient has clinical features favouring thrombus formation. The timing of these events is characteristic of temporary stunning, with the vast majority occurring within 1 week after cardioversion, and most events taking place in the first 3 days.[4] The decrease of thromboembolic risk after this time interval is probably due to gradual recovery of left atrial function after cardioversion.[5]

Current Practice Guidelines

The current 2010 ESC practice guidelines recommend that in patients presenting with acute AF, cardioversion can be performed immediately under cover of intravenous unfractionated or weight-adjusted therapeutic-dose low-molecular weight heparin, followed by either long-term oral anticoagulation (INR 2.0-3.0) in those at high risk of stroke (Class I, level of evidence B), or without post-cardioversion oral anticoagulation in those with no thromboembolic risk factors (Class IIb, level of evidence C) 14. In the 2011 update of the ACC/AHA/ESC practice guidelines on AF, the need for anticoagulation before and after cardioversion of acute AF is based on the individual patient risk for thromboembolism; nevertheless, with a lower level of evidence (Class IIa, level of evidence C) .[15] A recent compilation of the latter guidelines with those of the 2011 ACC/AHA/Heart Rhythm Society retained the same class of recommendation with the same level of evidence for patients with acute AF.[16] Likewise, the 2012 clinical practice guidelines of the American College of Chest Physicians recommended that for patients with acute AF should start anticoagulation at presentation, and after successful cardioversion they recommend therapeutic anticoagulation for at least 4 weeks regardless of the baseline risk of stroke, rather than no anticoagulation (grade 2C).[17] So, there has been a divergence of the class of recommendation between the European and North American clinical practice guidelines for considering long-term anticoagulation after successful cardioversion in patients with acute AF, with a C level of evidence in most cases, supported only by small retrospective studies.[14,15,17] (Table 2).

Table 2: Current practice guidelines on the recommendations for anticoagulation during cardioversion of acute atrial fibrillation

Authority	Practice guidelines	Class of recommendation	Level of evidence
ESC [14]	For patients with AF <48 h and at high risk of stroke, i.v. heparin or weight-adjusted therapeutic dose LMWH is recommended peri-cardioversion, followed by OAC therapy with a VKA (INR 2.0–3.0) long term.	I	B
ESC [14]	For patients with AF duration that is clearly <48 h and no thrombo-embolic risk factors, i.v. heparin or weight-adjusted therapeutic dose LMWH may be considered peri-cardioversion, without the need for post-cardioversion oral anticoagulation.	IIb	C
ACC/AHA/ ESC [15]	During the first 48 h after onset of AF, the need for anticoagulation before and after cardioversion may be based on the patient's risk of thromboembolism.	IIa	C
ACCP [17]	For patients with AF of documented duration of 48 h or less undergoing elective cardioversion (electrical or pharmacologic), we suggest starting anticoagulation at presentation (LMWH or unfractionated heparin at full venous thromboembolism treatment doses) and proceeding to cardioversion rather than delaying cardioversion for 3 weeks of therapeutic anticoagulation or a TEE-guided approach.	Grade 2	C
ACCP [17]	After successful cardioversion to sinus rhythm, we recommend therapeutic anticoagulation for at least 4 weeks rather than no anticoagulation, regardless of baseline stroke risk.	Grade 2	C

ACC indicates American College of Cardiology; ACCP, American College of Chest Physicians; AF, atrial fibrillation; AHA, American Heart Association; ESC, European Society of Cardiology; h, hour; INR, international Normalized Ratio; iv, intravenously; LMWH, low-molecular weight heparin; OAC, oral anticoagulants; TEE, trans-esophageal echocardiography; VKA, vitamin K antagonists.

Conclusions:

To prevent thromboembolism as a result of atrial stunning, effective peri- and post-cardioversion anticoagulation is of paramount importance also in acute attacks of AF. Without anticoagulation the risk of stroke is unacceptably high in patients with classic clinical risk factors for thromboembolism. It is reassuring, however, that patients without risk factors for stroke and with a brief and definite duration of preceding AF have a very low rate of thromboembolic events even without anticoagulation.

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