Left Atrial Coagulation Activity in Patient with Mitral Stenosis in Sinus Rhythm

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Objective: *Anticoagulation therapy is strongly recommended in all patients with mitral stenosis (MS) in atrial fibrillation (AF) but this treatment is controversial in patients in sinus rhythm (SR). The objective of the present study was to investigate the coagulation activity in patients with MS in sinus rhythm compared to those in atrial fibrillation.*
Material and Method: *The authors studied the levels of biochemical markers of thrombin generation (thrombin-anti-thrombin [TAT] complex, fibrinogen, and factor XIII) and fibrinolysis (D-dimer) in specimens of blood from the atria in 35 consecutive patients with moderate to severe MS (18 in sinus rhythm and 17 in AF) who underwent percutaneous balloon mitral valvotomy.*
Results: *The levels coagulation factors in left atrium in patients with MS in SR and AF were thrombin-anti-thrombin complex = 77.21±8.87 mg/L vs. 73.48±7.78 mg/L, p = 0.755, fibrinogen = 356.57±41.86 mg/L vs. 271.62±22.47 mg/L, p = 0.089, factor XIII = 139.88±8.96 mg/L vs. 123.42±6.24 mg/L, p = 0.152, and D-dimer = 846.14±137.84 mg/L vs. 693.88±164.67 mg/L, p = 0.481. Levels of coagulation activities did not correlate with the left atrial size.*
Conclusion: *This present study demonstrates that coagulation activity is not different whether they are in SR or in AF and suggests that anticoagulation therapy should be considered in these patients.*

Keywords: *Mitral stenosis, Coagulation activity, Sinus rhythm*

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Systemic embolism is one of the major complications in patients with mitral stenosis (MS)[1,2]. This serious complication may be the first clinical manifestation of patients with MS and may occur in patients with mild MS prior the development of heart failure[3,4]. The majority of patients with MS suffering from systemic emboli are usually in atrial fibrillation (AF); however, Coulshed et al showed that systemic embolism can occur in 8% of patients in sinus rhythm (SR)[2]. Although anticoagulation therapy is strongly recommended in patients with MS and AF[5], the recommendation for oral anticoagulation therapy in patients with MS in SR is not well established. Indeed, current guidelines do not recommend anticoagulation for patients with MS in SR whose left atrial (LA) size is less than 55 mm[6]. However, the presented group has demonstrated that the risk of systemic embolism in patients with MS in sinus rhythm is not associated with LA size[7]. Because of these uncertainties, the authors thought it is important to study the coagulation activity in patients with MS in SR. Thus, the aim of the present study was to examine the coagulation activity in patients with MS in SR compared to those in AF. Moreover, the authors tested the correlation between coagulation activity and LA size to define the risk of thromboembolism.

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Material and Method

Patients

The present study enrolled prospectively thirty-five patients with symptomatic mitral stenosis who were candidates for percutaneous balloon mitral valvotomy (PBMV). The criteria for exclusion included left atrial thrombus, malignancy, moderate to severe mitral regurgitation, severe aortic regurgitation, venous thrombosis, and liver disease. All patients were on chronic anticoagulation therapy, which was discontinued three days prior to the PBMV, and the

INR on the day of the procedure was less than 1.5. The present study protocol was approved by the Institutional Ethics Committee at Khon Kaen University. Written informed consent was obtained from all patients before the PBMV procedure.

Echocardiography

Transthoracic and transesophageal echocardiography (TTE and TEE, respectively) were performed in all of the patients. TTE was performed using a 2.5- or 3.5-MHz phase array transducer (Sonos 5500, Hewlett-Packard, Andover, Massachusetts). The measurements were obtained according to the standards of the American Society of Echocardiography[8]. The authors measured the LA diameter at the end of systole (from the parasternal view), the mitral valve area (by both planimetry from two-dimensional echocardiography and the continuous wave Doppler pressure halftime method) and the Wilkins mitral valve echocardiographic score[9].

TEE studies were performed with a 5-MHz omni-plane transducer (Hewlett-Packard Sonos 5500). After standard examination of the cardiac chambers and valves, the left atrial appendage (LAA) was visualized in both the basal short axis and two chamber views. Left atrial thrombus was diagnosed according to the presence of an intracavitary echogenic mass clearly distinct from the left atrial endocardium and the pectinate muscles, and visualized on at least two different planes. Left atrial spontaneous echo-contrast (LASEC) was characterized by dynamic clouds of echoes within the left atrial cavity. The severity of LASEC was graded from 0 to 4^+, as described by Fatkin et al[10].The observation of LASEC was standardized by varying both the gain and compress settings throughout their full range during each study. For analysis purposes, the presence of LASEC grade 3^+ and 4^+ was classified as a dense spontaneous echo contrast. The degree of LASEC was independently evaluated by two cardiologists (SK and CW). Any differences regarding the interpretation of LASEC were resolved by consensus.

Percutaneous balloon mitral valvotomy (PBMV) procedure

Patients were sedated with 5 mg of oral diazepam 30 minutes before the procedure. Introducer sheaths were inserted via right femoral vein and artery. A 6 F Swan Ganz catheter was inserted via femoral vein to measure right heart pressure and blood sampling from right atrium. A 6 F pigtail catheter was inserted via femoral artery to left ventricle for pressure measurement and ventriculography. The PBMV procedure was performed with transesophageal monitoring in the catheterization laboratory as described previously[12]. After atrial transseptal puncture with a Brockenborough needle, the Mulin's sheath was positioned in the left atrium for left atrium pressure measurement and blood sampling. The left ventricular-left atrial pressure gradient was recorded. Intravenous heparin was administered after all of the blood samples were collected. The efficacy of the procedure was assessed by transmitral valve gradient and 2D-echocardiography.

Blood sampling

After discarding the first 5 ml, approximately 5 ml of blood was obtained from the right and left atrium using a Swan Ganz catheter and Mulin's sheath, respectively. Blood samples were collected in a sterile syringe and immediately transferred to a 5 ml polyethyl terephthalate tube containing 0.5 ml of 3.2% trisodium citrate.

Assay procedure

Assays for biochemical markers of thrombin generation (thrombin-anti-thrombin [TAT] complex, fibrinogen, and factor XIII) and fibrinolysis (D-dimer) were performed on blood samples that were centrifuged within an hour of collection at 2,560 g at 4°C for 20 minutes to obtained platelet poor plasma. Aliquots of 300 µl of platelet poor plasma were then frozen and stored at -70°C before the assay procedure. Commercial enzyme immunoassays were used to measure plasma (atrial) levels of thrombin-antithrombin complex (TAT) (Enzygnost; Dade Berhring) and D-dimer (ASSERACHROM® D-Di; Stago, France). Both tests were assayed according to manufacturer protocols as previously described. Biotin incorporation assay was employed to determine FXIII activity. The assay was prepared in-house and the intra- and inter-assay variability was 7.83 and 7.63%, respectively. Fibrinogen concentration was measured by modified Clause method using an automated coagulometer (Sysmex, CA-1500).

Statistical analysis

Continuous variables are presented as means ± SD, while categorical variables are described with frequencies and percentages. Differences between the patient groups for categorical variables were examined using the Chi-square or Fisher's exact tests

or the Z-test. Differences in the continuous variables were assessed using the Student's t-test, Mann-Whitney U-test or Wilcoxon's rank sum test, where appropriate. A Pearson correlation coefficient linear regression model was used to examine the relationship between coagulation factor activities and left atrial size. A two-sided p-value <0.05 was required for statistical significance. All analyses were performed by using STATA version 10.0 (STATA Corporation, TX, USA).

Results

Table 1 summarizes the clinical characteristics of all of the patients. The study population was mostly female (74%) and middle-aged (42 years). The mean mitral valve area and left atrial dimension were 0.8 cm^2 and 47.8 mm, respectively. Dense left atrial spontaneous echo contrast (grade 3^+ and 4^+) was detected in 74% of the patients. The patients in the AF group tended to be older than those in SR group. The left atrial size in the AF group was significantly larger than in the sinus group and dense spontaneous contrast in the left atrium was also more often seen in the AF compared to the sinus rhythm group ($p<0.05$). There was no significant difference between the AF and sinus rhythm groups with respect to the left ventricular ejection fraction, mean pulmonary artery pressure, mean mitral valve gradient, and mitral valve area ($p>0.05$).

Complete coagulation activities in the atria were accomplished in all 35 patients (8 men, 17 women) with mitral stenosis. Coagulation activities in the left atrium were elevated in all patients. Coagulation activities in the left and right atrium (RA) in all patients are presented in Table 2. TAT and D-dimer levels in the left atrium were significantly higher than those in the right atrium. However, levels of fibrinogen and factor XIII were not significantly different between the two atria.

The coagulation activities in patients with MS in AF and in SR are shown in Table 3. Importantly, the coagulation activities and levels—including TAT, fibrinogen, D-dimer and factor XIII—were not significantly different between the sinus rhythm and

Table 1. Baseline characteristics of all patients

Clinical variables	All patient (n = 35)	SR (n = 18)	AF (n = 17)	p-value
Age	42.20±10.45	39.55±2.27	45.00±2.62	0.125
Female, n (%)	26 (74.3)	14 (77.77)	12 (70.58)	0.626
Pre PBMV MVA (cm^2)	0.81±0.21	0.89±0.07	0.97±0.07	0.408
Post PMBV MVA (cm^2)	1.48±2.27	1.41±0.29	1.54±0.34	0.159
LA size (mm)	47.83±0.84	42.79±1.68	52.86±1.97	0.005
LVEF,%	59.74±7.71	61.6±7.02	57.7±8.09	0.134
Mild to moderate aortic regurgitation, n (%)	24 (68.6)	14 (77.70)	10 (58.80)	0.084
Mild to moderate mitral regurgitation, n (%)	19 (54.3)	13 (72.20)	6 (35.20)	0.632
Dense LASEC, n (%)	26 (74.3)	10 (55.55)	15 (88.23)	0.032
Left atrial pressure (mmHg)	24.54±8.88	24.22±2.18	24.88±2.12	0.829
Mean PAP (mmHg)	39.28±16.07	41.88±4.16	36.53±3.44	0.331
Mean LA-LV pressure gradient (mmHg)	13.93±6.56	15.19±1.75	12.51±1.31	0.240

SR = sinus rhythm; AF = atrial fibrillation; PBMV = percutaneous ballooon mitral valvotomy; MVA = mitral valve area; LA = left atrium; LVEF = left ventricular ejection fraction; LASEC = left atrial spontaneous echo contrast; LV = left ventricle; PAP = pulmonary artery pressure

Table 2. Left and right atrial coagulation factor in all patients with mitral stenosis

	Left atrium (n = 35)	Right atrium (n = 35)	p-value
Fibrinogen (mg/dL)	315.38±24.95	320.90±22.85	0.870
Thrombin-antithrombin complex (μg/L)	75.40±5.84	28.99±4.58	<0.001
D-dimer (μg/L)	772.19±106.08	430.96±77.16	0.011
Factor XIII (mg/dL)	132.09±5.68	134.95±4.95	0.750

Table 3. Left atrium coagulation factor in sinus rhythm and atrial fibrillation

	Sinus (n = 18)	AF (n = 17)	p-value
Fibrinogen (mg/dL)	356.57±41.86	271.62±22.47	0.089
Thrombin-antithrombin complex (µg/L)	77.21±8.87	73.48±7.78	0.755
D-dimer (µg/L)	846.14±137.84	693.88±164.67	0.481
Factor XIII (mg/dL)	139.88±8.96	123.42±6.24	0.152

Table 4. Left atrial coagulation factor in patients with mild and dense spontaneous echo contrast

	Mild LASEC (n = 9)	Dense LASEC (n = 26)	p-value
Fibrinogen (mg/dL)	327.98±233.94	311.36±106.57	0.780
Thrombin-antithrombin complex (µg/L)	76.21±21.75	75.12±38.42	0.936
D-dimer (µg/L)	845.64±647.17	746.76±631.43	0.690
Factor XIII (mg/dL)	132.30±49.92	132.02±26.03	0.983

AF groups (p>0.05). Moreover, the levels were similar in patients with mild vs. severe SEC (Table 4).

The correlations between the left atrial size and each coagulation activity are presented in Table 5. There was no significant correlation between the left atrial size and coagulation activity parameters (p>0.05).

Table 5. Correlation between left atrial size and coagulation activity in all patients

Coagulation factor	R	p-value
Fibrinogen (mg/dL)	-0.14	0.49
Thrombin-antithrombin complex (mg/L)	0.09	0.58
D-dimer (mg/L)	0.05	0.77
Factor XIII (mg/dL)	-0.05	0.78

Discussion

The principle findings of the present study are that 1) the left atrial coagulation activity is increased in patients with MS and is similar in those patients with AF and SR; 2) left atrial dimension is not correlated with the level of coagulation activity; and 3) coagulation activity differs in the right vs. left atrium.

Thrombogenicity in mitral stenosis results in part from the atrial remodeling owing to atrial pressure and volume overload and AF. Systemic thromboembolism is a well-known complication in patients with MS and AF; the risk of stroke in patients with MS and AF is increased 18-fold compared with matched control subjects[12-15]. Thus, anticoagulation therapy is strongly recommended in patients with MS and AF or LA thrombus and history of stroke[6,13]. Guidelines support anticoagulation for patients with MS and SR only when additional risk factors are present - i.e. prior thromboembolism, dense spontaneous echo contrast, and at least moderate left atrial enlargement (>50 or 55 mm)[6,13]. However, the level of evidence in these guidelines is low without supporting evidence from randomized controlled clinical trials. In that regard, our group has, demonstrated previously that the risk of systemic embolism in patients with MS in sinus rhythm was not associated with LA size[7].

Yamamoto et al[14] first reported a hypercoagulable state in the left atrium contributing to the pathogenesis of systemic thromboembolism (especially in patients with AF) in MS, even during anticoagulation. Subsequently, biochemical markers of platelet activity and the status of thrombin generation, fibrinolysis, and endothelial function have been reported by many investigators[14-17]. For example, Boyaci et al[17] measured the coagulation activity in 46 patients with MS and found that left atrial tPA, plasmin, PAI-I, antiplasmin, PF4, and vWF levels were elevated significantly and exceeded the corresponding peripheral venous levels, especially in those with AF. The increase of systemic and regional coagulation activity has been reported previously in patients with MS and SR[15-21]. Topaloglu et al[15] noted that if SEC was present, there was no difference in coagulation and platelet activation and endothelial dysfunction in patients with AF and SR. Similarly, Atak et al[16] reported that patients with severe MS and SR had increased regional coagulation activity in those with and without left atrial SEC, but only the former had peripheral blood activity, suggesting peripheral spillover in the former. Jafri et al[22] reported that the plasma concentration of fibrinogen and D-dimer were

elevated among female patients with MS and SR. The present study confirms that the markers of coagulation activity including fibrinogen, TAT complex, D-dimer, and Factor XIII were significantly increased in the LA in patients with MS, irrespective of the cardiac rhythm and the presence of severe SEC.

Interestingly, the authors found no correlation between LA dimension and the level of coagulation factors. The risk factor of systemic thromboembolism in patients with mitral stenosis in sinus rhythm has not been well established, although recommendations are based on LA size. Daimee et al[19] reported a subgroup of patients with MS in sinus rhythm at risk of thromboembolism. In that study, the incidence of systemic embolism was increased in patients with low left atrial contraction velocity (<25 cm/s). Keenan et al[20] demonstrated that LA volume >60 ml/m^2 in MS and SR was more precisely related to markers of thromboembolism (SEC, low LAA velocities) than LA diameter. However, similar to our findings, Topaloglu et al[15] found no significant difference between their patients with SR and AF with regard to LA diameter, mitral valve area, left ventricular ejection fraction, and coagulation parameters.

An interesting finding in the present study was the greater coagulation activity in the LA than RA. In contrast, Li-Saw-Hee et al[23] found no difference in coagulation activity between right and left atria in patients with chronic AF. However, while Yamamoto et al[14] reported that the levels of fibrinopeptide A and thrombin-antithrombin III complex in the left atrium were significantly higher than in the right atrium, the plasma levels of platelet factor 4, D-dimer, and plasmin-alpha$_2$-plasmin inhibitor complex were not. Although it has been suggested that factor XIII—a fibrin clot stabilizer—could facilitate the growth of new thrombus and may contribute to the atherogenesis involving the coronary artery and cerebrovascular disease[18], the level of factor XIII in the LA was not significantly higher than in the RA in the present study. Cianciulli et al[24] reported that patients with MS and SR had reduced right and left atrial appendage velocities, but that dysfunction and SEC were less in the RA than LA.

Limitations

There are several limitations of the present study that should be acknowledged. First, the authors used left atrial dimension not volume. However, guideline recommendations are based on left atrial diameter. Left atrial appendage velocities were not reported in the present study, as these were generally low and poor quality. Second, while the authors demonstrate important rhythm, site, and size-specific changes in biochemical markers of coagulation, the authors' survey is far from comprehensive; however, ultimately these markers are all surrogates for thrombus formation and only indirectly assess coagulation activity. Third, there is a selection bias as these patients were deemed suitable for valvuloplasty and all were on anticoagulants, which were discontinued three days before the procedure. Fourth, only certain coagulation activity parameter (that were available) were compared which may not represent clotting potential in mitral stenosis. Finally, this is a cross-sectional study and therefore lacks prospective follow-up and evaluation of hard events.

Despite these limitations, this present study demonstrates that left atrial coagulation activity in patients with moderate to severe mitral stenosis in sinus rhythm is increased irrespective of left atrial dimension, and, as in patients with mitral stenosis in atrial fibrillation, anticoagulation therapy may be warranted to reduce the risk of systemic embolism.

Potential conflicts of interest

None.

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ปัจจัยการแข็งตัวของเลือดในหัวใจห้องบนซ้ายในผู้ป่วยลิ้นไมทรัลตีบที่มีการเต้นหัวใจปกติ

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ภูมิหลัง: ผู้ป่วยโรคลิ้นไมทรัลตีบที่มีหัวใจเต้นผิดจังหวะชนิด atrial fibrillation ควรได้ยาต้านการแข็งตัวของเลือดทุกราย เพื่อป้องกันภาวะลิ่มเลือดหลุดอุดหลอดเลือด แต่ในผู้ป่วยลิ้นไมทรัลตีบที่มีการเต้นของหัวใจปกติยังไม่มีแนวทางการใช้ยาต้านการแข็งตัวของเลือดที่ชัดเจน

วัตถุประสงค์: เพื่อหาระดับปัจจัยการแข็งตัวของเลือดในหัวใจห้องบนซ้ายของผู้ป่วยลิ้นไมทรัลตีบที่มีการเต้นของหัวใจปกติ เปรียบเทียบกับในผู้ป่วยลิ้นไมทรัลตีบที่มีการเต้นหัวใจผิดปกติชนิด atrial fibrillation

วัสดุและวิธีการ: ทำการศึกษาระดับปัจจัยการแข็งตัวของเลือด โดยการตรวจหาค่าของ thrombin-anti-thrombin complex, fibrinogen, factor XIII และ D-dimer ในหัวใจห้องบนของผู้ป่วยลิ้นไมทรัลตีบขั้นปานกลางและรุนแรงที่เข้าทำการรักษาด้วยบอลลูนขยายลิ้นหัวใจจำนวน 35 ราย โดยมีเป็นผู้ป่วยที่มีหัวใจ เต้นปกติ 18 ราย และ atrial fibrillation 17 ราย

ผลการศึกษา: พบว่าระดับปัจจัยการแข็งตัวของเลือดในผู้ป่วยลิ้นไมทรัลตีบที่มีการเต้นหัวใจปกติ และ atrial fibrillation มีค่าตามลำดับดังนี้ ระดับ thrombin-anti-thrombin complex เท่ากับ 77.21±8.87 mg/L และ73.48±7.78 mg/L ค่า p เท่ากับ 0.755 ระดับ fibrinogen เท่ากับ 356.57±41.86 mg/L และ 271.62±22.47 mg/L ค่า p เท่ากับ 0.089 ระดับแฟคเตอร์ XIII เท่ากับ 139.88±8.96 mg/L และ 123.42+6.24 mg/L ค่า p เท่ากับ 0.152 ระดับ D-dimer เท่ากับ 846.14±137.84 mg/L และ 693.88±164.67 mg/L ค่า p เท่ากับ 0.481 และระดับของปัจจัยการแข็งตัวของเลือดไม่มีความสัมพันธ์กับขนาดของหัวใจห้องบนซ้าย

สรุป: การศึกษานี้แสดงให้เห็นว่าระดับของปัจจัยการแข็งตัวของเลือดในผู้ป่วยลิ้นไมทรัลตีบที่มีหัวใจเต้นปกติ ไม่แตกต่างจากผู้ป่วยที่มี atrial fibrillation และไม่มีความสัมพันธ์กับขนาดของหัวใจห้องบนซ้าย ผู้ป่วยลิ้นไมทรัลตีบที่มีการเต้นของหัวใจปกติอาจจะมีความจำเป็นที่ต้องให้ยาต้านการแข็งตัวของเลือด เหมือนกับผู้ป่วยที่มี atrial fibrillation