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Low Dose Aspirin: An Effective Chemoprophylaxis for Preventing Venous Thromboembolic Events

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INTRODUCTION

The available guidelines, endorsed by Surgical Care Improvement Project (SCIP), have advocated that aspirin (ASA) is a safe and effective strategy for venous thromboembolic events (VTE) prophylaxis following total joint arthroplasty (TJA). The optimal dose of aspirin for this purpose is not known. The first guidelines for prevention of VTE that were issued by the American Academy of Orthopedic Surgeons recommended 325 mg Bis in die (twice a day) (bid) for this purpose with the recommendation having a 1C grade (little evidence to support the recommendation). It is known that platelet aggregation inhibition occurs at lower doses. Traditionally, ASA 81mg has been used as a cardioprotective medication. Additionally, all available randomized studies, including the sentinel study on Pulmonary Embolism Prevention (PEP) trial¹⁻⁴ have used lower doses of ASA. It was our hypothesis that lower dose aspirin is likely to be as effective as higher dose aspirin while reducing the gastrointestinal side effects associated with the higher dose aspirin.

MATERIALS AND METHODS

We analyzed a cohort of 2,880 primary TJA patients. All patients were treated with post-operative intermittent pneumatic compression while hospitalized. Of these, 2,138 patients with an average age of 64.6 years [Standard desviation (SD) ±10.4] received enteric coated ASA 325mg by mouth, bid for 4 weeks. In the other group, 742 patients with an average age of 64.1 years (SD±12.0) received plain ASA 81mg by mouth bid for 4 weeks. Gender, body mass index (BMI), and comorbidities assessed by the Charlson comorbidity index (CCI) were recorded (Table 1). There was no difference in age, gender, CCI, or BMI between the patient populations. Patients were evaluated for the development of symptomatic VTE in the post-operative period using International Classification of Diseases version 9 (ICD-9) codes, specifically deep vein thrombosis (DVT) and pulmonary embolism (PE). Statistical analysis was performed using Wilcoxon and Fisher's tests.

RESULTS

There was no significant difference in the incidence of VTE between the two groups; 0.1% in the 81mg ASA group (one DVT), compared to 0.2% in the 325mg ASA group (2 DVT and 2 PE). Two episodes of gastrointestinal (GI) bleeding occurred in the 325mg ASA group, compared to none in the 81mg ASA group.

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Investigation performed at the Rothman Institute at Thomas Jefferson University, Philadelphia, PA.

TABLES 1 & 2

		Aspirin 81mg bid	Aspirin 325mg bid	p-value	Complication	ASA 81mg bid	ASA 325mg bid	p-value
Age (years) mean (SD)		64.1 (12.0)	64.6 (10.4)	0.295	Complication	(n=742)	(n=2138)	
CCI mode (SD)		3 (± 2)	3 (± 1)	0.082	DVT	1 (0.1%)	2 (0.1%)	0.764
BMI (kg/m ²) mean (SD)		29.7 (6.2)	29.5 (5.1)	0.738	Pulmonary Embolism	0 (0%)	2 (0.1%)	0.405
LOS (days) mean (SD)		1.9 (1.1)	1.9 (2.2)	0.591	Gastrointestinal Bleeding	0 (0%)	2 (0.1%)	0.405
Sex	Male	46.2%	45.1%	0.654	Acute Infection	0 (0%)	5 (0.2%)	0.187
	Female	53.8%	54.9%		90-day Mortality	1 (0.1%)	2 (0.1%)	0.764
Primary THA		40.2%	51.6%	<0.001	Table 2: Complication rates in patients receiving ASA 81mg bid vs. 325mg bid.			
Primary TKA		59.8%	48.4%	<0.001	mg=milligrams; bid= <i>Bis in die</i> (twice a day); DVT=Deep venous thrombosis.			

Demographics and procedures in patients receiving ASA 81mg bid vs. 325mg bid.

mg=milligrams; bid=Bis in die (twice a day); SD=Standard deviation; CCI=Charlson comorbidity index; Kg=Kilograms; m²=Square meter; THA=Total hip arthroplasty; TKA=Total knee arthroplasty.

FIGURE 1

Aspirin Evidence: Dose and Efficacy Indirect comparisons of aspirin doses on vascular events in								
Aspirin Dose	No. of Trials	(%)	Odds Ra Vascula	atio for r Events				
500 – 1500 mg	34	19	-					
160 – 325 mg	19	26						
75 – 150 mg	12	32						
< 75 mg	3	13		_				
Any aspirin	65	23	•					
					P<0.0001			
		0	0.5 1	.0 1.5	2.0			
Antiplatelet Better Antiplatelet Worse								

Figure 1: Forest plot from the Antitrombotic Trialists' Combination study⁴ depicting the protective effect againts vascular events among different aspirin dosages. Studies using between 75 and 150 mg of aspirin exhibit a greater antiplatelet effect than studies with aspirin regimens in between 160 and 325 mg. No.=Number; mg=milligrams.

Acute infection rate was also higher in the 325mg ASA group at 5 cases (0.2%) compared to none in the 81mg ASA group. Finally, there were two mortalities in the 325mg ASA group (one in-hospital, one post-discharge) compared to one mortality in the 81mg ASA group (post-discharge).

Our ongoing study demonstrates that low dose ASA (81mg bid for four weeks) is as effective of a prophylaxis agent as high dose ASA (325mg) following TJA. This is not surprising as all available literature, including many publications related to VTE prophylaxis following TJA, demonstrate that low dose aspirin has better antiplatelet aggression properties. Continued evaluation of the safety and efficacy of ASA as a prophylactic agent and the comparison of the doses continues at our in our prospective study.

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RESULTS

DISCUSSION

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