The Journal of Arthroplasty 31 (2016) 2653–2654

Contents lists available at ScienceDirect

The Journal of Arthroplasty

journal homepage: www.arthroplastyjournal.org

Letters to the Editors

Letter to the Editor on "Balancing Thromboprophylaxis and Bleeding in Total Joint Arthroplasty: Impact of Eliminating Enoxaparin and Predonation and Implementing Pneumatic Compression and Tranexamic Acid"

To the Editor:

We had the opportunity to read last issue's article by Sharfman et al [1] regarding the use of an intermittent pneumatic compression device (IPCD) and tranexamic acid (TXA) as prophylactic measures against venous thromboembolic disease and bleeding, respectively. The authors' research on the subject is a valuable contribution to the study of relevant perioperative complications after hip or knee arthroplasty, and their stepwise approach demonstrated interesting results in patient outcomes [1]. Nevertheless, we would like to contribute some observations that could lead to a better understanding of this article, especially regarding its main conclusion.

The authors concluded and recommended that using an IPCD and TXA and discontinuing enoxaparin and preoperative autologous blood donation eliminated blood transfusion in hip or knee arthroplasty without any increase in venous thromboembolic disease [1]. However, this could be misleading due to the limitations of this study.

First, although the American College of Chest Physicians guidelines recommend the use of an IPCD in the absence of other antithrombotic therapies, it states that the best prophylactic option is enoxaparin [2,3]. It only approves the sole use of an IPCD as a first line option if the patient is at a high risk of bleeding [2]. Therefore, if the study sought to prove that the use of an IPCD is equivalent to pharmacological agents and as such could also be considered a first line option, patient follow-up should have been extended from 14 to 35 days. This is due to the fact that most venous thromboembolic disease events may occur up to 35 days after knee or hip arthroplasty [1,2].

We would also like to address the way the study divided treatment options and patients, especially in regard to the combination of TXA and an IPCD. The use of TXA by itself has been shown to reduce the need for blood transfusion [4,5], and although it did lead to favorable results when used in conjunction with an IPCD, this does not necessarily mean that this prophylactic combination is equivalent to pharmacological agents. As a result, this continuous improvement exercise does not allow adequate isolation of the different interventions in order to prove their efficacy as prophylactic agents.

Finally, this study does not have enough statistical power to challenge the gold standard which is currently pharmacological prophylaxis. In order to accomplish this objective, it would require a much larger sample size and, as previously mentioned, a thorough modification in its methodology to support this affirmation. Although the authors acknowledge these flaws, they also state that it would be wrong to ignore their conclusions and not provide this approach to patients [1]. As a result of the aforementioned limitations, this recommendation might be an overstatement.

In conclusion, this article is consistent with previous revisions regarding the efficacy of TXA and its reduction of blood loss. However, there is not enough evidence to conclude that an IPCD is equivalent to pharmacological agents and that it reduces the need for transfusions. Further studies are required to validate the use of this therapeutic approach.

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DOI of original article: http://dx.doi.org/10.1016/j.arth.2015.11.046.

One or more of the authors of this paper have disclosed potential or pertinent conflicts of interest, which may include receipt of payment, either direct or indirect, institutional support, or association with an entity in the biomedical field which may be perceived to have potential conflict of interest with this work. For full disclosure statements refer to http://dx.doi.org/10.1016/j.arth.2016.06.041.

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http://dx.doi.org/10.1016/j.arth.2016.06.041

References

1. Sharfman ZT, Campbell JC, Mirocha JM, et al. Balancing thromboprophylaxis and bleeding in total joint arthroplasty: impact of eliminating enoxaparin and predonation and implementing pneumatic compression and tranexamic acid. J Arthroplasty 2015;31:1307.
2. Falck-Ytter Y, Francis CW, Johanson NA, et al. Prevention of VTE in orthopedic surgery patients: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. Chest 2012;141:e278S.
3. Fujita Y, Nakatsuka H, Namba Y, et al. The incidence of pulmonary embolism and deep vein thrombosis and their predictive risk factors after lower extremity arthroplasty: a retrospective analysis based on diagnosis using multidetector CT. J Anesth 2015;29:235.
4. Moskal JT, Capps SG. Meta-analysis of intravenous tranexamic acid in primary total hip arthroplasty [Epub ahead of print]. Orthopedics 2016:1, http://dx.doi.org/10.3928/01477447-20160526-02.
5. Wu Y-G, Zeng Y, Yang T-M, et al. The efficacy and safety of combination of intravenous and topical tranexamic acid in revision hip arthroplasty: a randomized, controlled trial [Epub ahead of print]. J Arthroplasty 2016:1, http://dx.doi.org/10.1016/j.arth.2016.03.059.

Response to Letter to the Editor on "Balancing Thromboprophylaxis and Bleeding in Total Joint Arthroplasty: Impact of Eliminating Enoxaparin and Predonation and Implementing Pneumatic Compression and Tranexamic Acid"

In Reply:

We thank Dr. Bonilla et al for their interest in our article and their thoughtful comments. While we agree with many of their points, we would like to highlight a few points in response to their commentary.

In response to their first point, we respectfully disagree with their read of the ACCP guidelines. Guideline 2.1.1 clearly states "In patients undergoing total hip arthroplasty (THA) or total knee arthroplasty (TKA), we recommend use of one of the following for a minimum of 10 to 14 days rather than no antithrombotic prophylaxis: low-molecular-weight heparin (LMWH), fondaparinux, apixaban, dabigatran, rivaroxaban, low-dose unfractionated heparin (LDUH), adjusted-dose vitamin K antagonist (VKA), aspirin (all Grade 1B), or an intermittent pneumatic compression device (IPCD) (Grade 1C)." [1]. Importantly, the IPCD specifically recommended within the guidelines is the device used in our study. Furthermore, though Guideline 2.3.1 does state "we suggest the use of LMWH in preference to the other agents…" [1], the strength of this recommendation is a lower grade (2B-2C). In addition, the ACCP recommendation does not "only approve[s] the sole use of an IPCD as a first line option if the patient is at a high risk of bleeding." Rather Guideline 2.6 states "In patients undergoing major orthopedic surgery and increased risk of bleeding, we suggest using an IPCD or no prophylaxis rather than pharmacologic treatment (Grade 2C)." [1].

With regard to the point that "the study sought to prove that the use of an IPCD is equivalent to pharmacologic agents…," we respectfully underscore that this was not our intention, nor did we make this claim. Our study did not set out to prove equivalence nor did its retrospective design permit such proof. We are in agreement that this would require a much larger cohort of patients and an entirely different study design. We do, however, believe that within our series there are no signals to suggest inferiority (increased risk of venous thromboembolic disease [VTED]), and this point is further supported by an article currently submitted for publication reporting the use of this protocol in 179 patients followed for more than 3 months demonstrating the same outcomes.

We also agree with Dr. Bonilla et al that our study does not necessarily allow adequate isolation of the different interventions in order to prove their efficacy as prophylactic agents. However, it should be clarified that the tranexamic acid was not used as a prophylactic against VTED, but rather to reduce bleeding. The benefit of tranexamic acid is that it indeed reduces blood loss without any demonstrated increase in VTED in our study or in the broader literature. In addition, the Active Care while specifically used as a prophylaxis against VTED has also been demonstrated to reduce blood loss. The combined use has for the first time enabled effective VTED without an increased risk of bleeding, a claim that no pharmacologic agent can make. It is for this reason that although we agree that our study does not contain the statistical power to challenge the efficacy of pharmacologic prophylaxis, we nevertheless reaffirm our conclusion that it would be wrong to ignore the findings of our study and the potential benefit without detectable increased risk this protocol provides to our patients.

Most of all, we agree that further studies are required to validate the use of this therapeutic approach, and we hope that our submitted work and ongoing research will continue to contribute to this worthy discussion.

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http://dx.doi.org/10.1016/j.arth.2016.06.042

Reference

1. Falck-Ytter Y, Francis CW, Johanson NA, et al. Prevention of VTE in orthopedic surgery patients: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest 2012;141(2 Suppl):e278S.

DOI of original article: http://dx.doi.org/10.1016/j.arth.2016.06.041.

One or more of the authors of this paper have disclosed potential or pertinent conflicts of interest, which may include receipt of payment, either direct or indirect, institutional support, or association with an entity in the biomedical field which may be perceived to have potential conflict of interest with this work. For full disclosure statements refer to http://dx.doi.org/10.1016/j.arth.2016.06.042.