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INTERVENTIONAL RADIOLOGY

LETTER TO THE EDITOR

Reply: Impact of biopsy route, muscle pathway, and cortex target on safety and diagnostic yield in ultrasound-guided renal parenchymal biopsy

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Dear Editor,

We thank the author for the thoughtful comments on our study.[1] We appreciate the opportunity to clarify several methodological aspects.

First, we agree that blood pressure and impaired renal function may influence bleeding risk after percutaneous renal biopsy.[2-4] As described in the Methods, biopsy was performed only when blood pressure was < 160/90 mmHg. Nevertheless, in response to the author's comments, we evaluated whether these variables contributed to the observed findings by comparing them between groups. Baseline blood pressure, serum creatinine, and estimated glomerular filtration rate (eGFR) were retrieved from the medical and laboratory records. Independent-samples t-tests showed no significant differences between the lateral-to-medial and medial-to-lateral groups in systolic blood pressure (125.9 ± 12.2 vs. 125.4 ± 11.1 mmHg, $P = 0.676$), diastolic blood pressure (81.4 ± 8.6 vs. 81.9 ± 7.5 mmHg, $P = 0.514$), or eGFR (74.2 ± 43.4 vs. 73.6 ± 45.2 mL/min/1.73 m^2, $P = 0.886$). Because serum creatinine was non-normally distributed, the groups were compared using the Mann–Whitney U test; mean creatinine levels were likewise similar (1.80 ± 1.86 vs. 1.88 ± 1.90 mg/dL, $P = 0.653$). These findings do not support blood pressure or renal function as alternative explanations for the observed difference in hematoma size.

As stated in the Methods, all biopsies were performed using a coaxial technique, and two core tissue samples were obtained from every patient. Therefore, the number of biopsy passes cannot explain the differences in bleeding or diagnostic yield. Regarding tract hemostasis, whenever bleeding was observed through the coaxial introducer, the inner stylet was first reinserted to achieve temporary tract tamponade. If bleeding persisted, an autologous blood clot or an absorbable gelatin sponge was used. This was not a prophylactic intervention but rather a standardized institutional algorithm applied identically by both operators. We agree that documenting these interventions would have strengthened the study; however, these data were not systematically recorded because of the retrospective design.

Operator-specific biopsy approaches represent an inherent limitation, as acknowledged in the Discussion. Nevertheless, both interventional radiologists had more than 10 years of experience, had each performed more than 1,000 renal biopsies, and consistently used their respective standardized techniques. Moreover, suboptimal diagnostic yield was comparable between the lateral-to-medial and medial-to-lateral groups (95.4% vs. 95.3%, $P = 0.958$) as was pathologist-based diagnostic adequacy (96.6% vs. 98.0%, $P = 0.335$). Although optimal diagnostic yield differed (73.0% vs. 85.0%, $P = 0.001$), mean glomerular counts within biopsy adequacy categories were similar (all $P \geq 0.058$), suggesting that the difference reflects the predefined adequacy threshold rather than a clinically meaningful reduction in diagnostic performance.

Finally, we agree that bleeding originates from renal and capsular vessels.[5] Our discussion did not suggest that the traversed muscles alter bleeding initiation but rather proposed a plausible explanation for the smaller non-transfusion-requiring perirenal hematomas observed

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in one group, analogous to the tamponade effect of surrounding tissues described in other percutaneous biopsy settings.[6] Major bleeding and clinically significant complications were comparable between the groups. As emphasized in the Discussion, this interpretation was intentionally presented as a hypothesis rather than as evidence of a direct causal mechanism.

We thank the author for the valuable comments, which have helped clarify our methodology and interpretation.

Footnotes

Conflict of interest disclosure

The authors declared no conflicts of interest.

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