Am J Cardiovasc Dis 2021;11(4):462-470
www.AJCD.us /ISSN:2160-200X/AJCD0136779

Original Article

Forearm hematoma as a complication of transradial coronary intervention: an Indian single-center experience

Sudhanshu Kumar Dwivedi*, Gyanaranjan Nayak*, Akhil Kumar Sharma*, Gaurav Kumar Chaudhary*, Sharad Chandra*, Monika Bhandari*, Pravesh Vishwakarma*, Akshyaya Pradhan*, Rishi Sethi*, Varun Shankar Narain*

*Department of Cardiology, King George's Medical University, Lucknow, Uttar Pradesh 226003, India. *Equal contributors.*

Received June 26, 2021; Accepted August 14, 2021; Epub August 15, 2021; Published August 30, 2021

Abstract: Background: Forearm hematomas are not uncommon after transradial coronary interventions. The present study describes the incidence and predictors of forearm hematoma formation after transradial coronary interventions. Methods: This was a prospective study in 1754 patients undergoing angiography/angioplasty through transradial access. Each procedure was performed using optimum levels of anticoagulation, hydrophilic sheaths, and post-procedural patent hemostasis protocols. Patients were evaluated for forearm hematoma immediately after the procedure, after radial band removal, and on the next day of the procedure. Severity of hematomas was graded according to the Early Discharge after Transradial Stenting of Coronary Arteries Study scale. Univariate and multivariate logistic regression analyses were done to determine the predictors of hematoma formation. Results: Mean age of the patients was 56.31 years and 82.2% were males. A total of 1374 (78.3%) patients underwent angioplasty while 380 (21.7%) underwent angiography. Forearm hematoma developed in 187 (10.7%) patients. Grade I hematoma was most common (3.53%) followed by Grade II (3.08%), Grade III (2.83%) and Grade IV (1.25%) hematoma. None of the patients required vascular or surgical interventions for this complication. Female gender, multiple puncture attempts, intensive antiplatelet therapy, complex procedure and longer hemostasis time were significant predictors of forearm hematoma formation post transradial coronary interventions. Conclusions: Forearm hematoma developed in substantial proportion of patients undergoing transradial coronary interventions and interventional variables were predominantly associated with hematoma formation. Pre-emptive knowledge of modifiable interventional risk factors can help in reducing the burden of this complication.

Keywords: Coronary angiography, forearm hematoma, percutaneous coronary intervention, transradial coronary intervention

Introduction

Cardiac catheterization procedures, both diagnostic and therapeutic, primarily involve the transfemoral or the transradial approach. Since last two decades, transradial coronary intervention (TCI) has become more popular owing to its minimal invasive nature, ease of performance, minimum patient discomfort, early ambulation, shorter duration of hospital-stay, and potentially low cost and reduced risk of morbidity and mortality [1-3]. Due to the superficial and readily compressible location of the radial artery, transradial access is associated with minimum complications when compared to other access sites for cardiac catheterization. However, radial artery spasm/occlusion at access site and forearm hematoma are known complications associated with TCI. Incidence of forearm hematoma after TCIs ranges from 0.3% to 33%, although the incidence rates for a large forearm hematoma requiring blood transfusions or vascular surgery are negligible [4-6]. In most of the cases, it is reported as a complication only when it requires an intervention. However, it is an established fact that even a smaller unresolved haematoma could lead to severe complications like compartment syndrome [5, 7, 8].

Well-designed trials have not been conducted to study the occurrence of forearm hematoma after TCI procedures and experience for these hematomas has largely derived from previous single-centre reports [9]. There are currently no registries for reporting this complication after TCI.

Considering the limited available data for this complication, the current study was aimed to bring in more understanding of forearm haematoma as a complication of transradial cardiac catheterization. This study was done to understand the true burden of forearm haematoma, its severity and possible predictors.

Material and methods

This was a prospective observational study conducted between October 2018 and September 2019, at the King George Medical University, Lucknow, a tertiary care centre in India. The study was conducted in compliance with the International Ethical Guidelines for Biomedical Research Involving Human Subjects, Good Clinical Practice Guidelines, the Declaration of Helsinki, and local laws. All patients or their family members provided a written informed consent. The study protocol was approved by the Institutional Ethics Committee (95th ECM 11B-Thesis/P51, 11/01 2016).

Study population

The study enrolled consecutive patients (aged >18 years) who were diagnosed with acute coronary syndrome or stable coronary artery disease and were referred for diagnostic and/or therapeutic coronary catheterization. Patients who were planned through transradial route for coronary catheterization and had a normal Barbeau test were finally included. Patients with a negative Allen's test, failed puncture of the radial artery, post coronary artery bypass surgery, decompensated heart failure, severe valvular disease, severe sepsis, prior history of transradial procedures, local site infections, high bleeding risk, cardiogenic shock, hemodynamic instability, glomerular filtration rate (GFR) <30 ml/minute/m^2, and patients on hemodialysis and those requiring a switch to transfemoral access were excluded. For study purposes, high risk of bleeding was defined as platelet count <70000 cells/mm^3 or use of anticoagulants with INR >2.5.

Detailed medical history, demographic characteristics, biochemical parameters, echocardiographic and procedural characteristics were recorded for every patient. Routine hemogram and renal function tests were done for all patients. Serum creatinine level ≥1.5 mg/dl was defined as renal dysfunction for study purpose. Coronary interventional procedures were performed as per standard practical guidelines and were performed by well-versed interventional cardiologists with radial interventions.

Transradial access and procedure

The percutaneous access site was prepared sterile and anesthetized using 2% lidocaine. Transradial access was established by using 5F, 6F, 7F sheaths (Radifocus introducer II; Terumo, Japan). All patients received 200 µg of nitroglycerin and 5 mg of verapamil intra-arterially through the introducer sheath. Patients who were planned for diagnostic angiogram received 5000 IU unfractionated heparin (UFH) through the arterial sheath. For urgent percutaneous coronary interventions (PCI), an additional bolus of UFH was administered according to patient's weight. For elective PCIs, 100 IU/Kg of UFH was administrated through the sheath before introducing the guiding catheter. Additional boluses were then added intravenously according to the physician's choice.

After angiography or angioplasty procedures, hemostasis was achieved by following the patent hemostasis protocol [10]. A plethysmographic probe was attached to the index finger and a TR band (Terumo, Japan) with a small green box indicator was placed proximal to the radial artery puncture site in the forearm. The sheath was removed, and total occlusion of radial artery was achieved by inflating the compression balloon with 18 ml of air. The ulnar artery was manually compressed to block the flow in the palmar arch and compete loss of plethysmographic signals at this stage suggested total occlusion of antegrade flow from the radial artery. Pressure in the compression balloon of the TR band was gradually reduced by decreasing the amount of air in steps of 1 ml until the return of pulsatile plethysmographic signals or occurrence of bleeding. If bleeding occurred during this process, pressure in the compression balloon was again slightly increased by injecting about 1 ml of air to ultimately achieve radial artery compression with "patent hemostasis". Presence of pulsatile plethysmo-

graphic signals from the index finger while manually compressing the ipsilateral ulnar artery indicated patent radial artery hemostasis. The patency of the radial artery was checked at least once every 15 minutes. The TR wrist band was removed as soon as compression balloon was deflated without puncture site bleeding. A light pressure bandage was applied. All patients were encouraged to squeeze and release the hand and to keep the arm in an elevated position during the entire compression period.

Procedural details like diagnostic or therapeutic procedure, number of attempts for radial artery access, sheath size, procedure time, IV heparin dose, type of antiplatelet drugs used, use of Glycoprotein IIb/IIIa inhibitors, number of catheter exchange, type of coronary lesion (chronic total occlusion, bifurcation lesion), contrast volume, and post procedure hemostasis time were noted. Radial access in 2 or more attempts was labelled as multiple attempts. Procedure time >60 minutes, >3 catheters exchange, chronic total and bifurcation coronary lesion interventions were considered complex procedures. Intensive antiplatelet therapy was defined as use of dual antiplatelet therapy containing ticagrelor or prasugrel and/or use of Glycoprotein IIb/IIIa inhibitors.

Follow-up for hematoma

All patients were closely observed for the development of forearm or arm haematoma immediately after the procedure, after radial band removal and on the next day of the procedure. Any incidence of pain or swelling at the local site was reported. Local haematoma was graded according to the severity based on the EArly Discharge after Transradial Stenting of CoronarY Arteries Study (EASY) hematoma scale: (i) Grade-I, <5 cm in diameter (nonsignificant); (ii) Grade-II, 5-10 cm diameter (mild); (iii) Grade-III, >10 cm but distal to the elbow (moderate); (iv) Grade-IV, extending above the elbow (severe); and (v) Grade-V, anywhere with ischemic threat to the hand (compartment syndrome) [11]. All patients were followed-up every 2 weeks after discharge, till hematoma resolution.

Statistical analysis

Data were expressed as numbers and proportions for categorical variables and as mean ± SD for continuous variables. Data were compared for patients who developed and those who did not develop a hematoma. Continuous variables were compared using the Student's *t*-test and categorical values were compared using the Chi-square test. Potential risk factors for hematoma formation were assessed using a univariate followed by a multivariate logistic regression analysis. Odds ratio (OR) and the inclusive 95% confidence intervals (CI) were estimated for all significant variables. All tests were performed as 2-sided at 5% level of significant ($P<0.05$). All data were analysed using Statistical Package for Social Sciences (SPSS, Chicago, USA) version 21.0.

Results

Baseline and demographic characteristics

A total of 1754 consecutive patients underwent TCI, 380 (21.7%) having angiography alone. The flowchart of study patients is depicted in **Figure 1**. Mean age of the patients was 56.31±10.58 years and majority of the patients were males (82.2%). The demographic and clinical characteristics of all patients are presented in **Table 1**. About one-third of the patients were smokers (32.5%) or had diabetes (35.5%) or hypertension (38.0%).

Procedural characteristics

Table 1 shows the procedural characteristics for patients with and without the development of a hematoma after TCI. A radial sheath of 6F was used in most (77.95%) patients. About 37% patients needed multiple puncture attempts and more than two-third (77.9%) patients needed a contrast volume of 100 ml or more.

Forearm hematoma

Forearm hematoma was reported in 187 (10.7%) patients. Maximum (n=62; 3.53%) had Grade I haematoma followed by Grade II (n=54; 3.08%), Grade III (n=49; 2.83%) and Grade IV (n=22; 1.25%). None of the patients developed compartment syndrome (Grade V hematoma). All patients with hematoma formation were successfully managed conservatively by manual or device compression, reversal of anticoagulation, and occlusive haemostasis according to the grade and progression of the

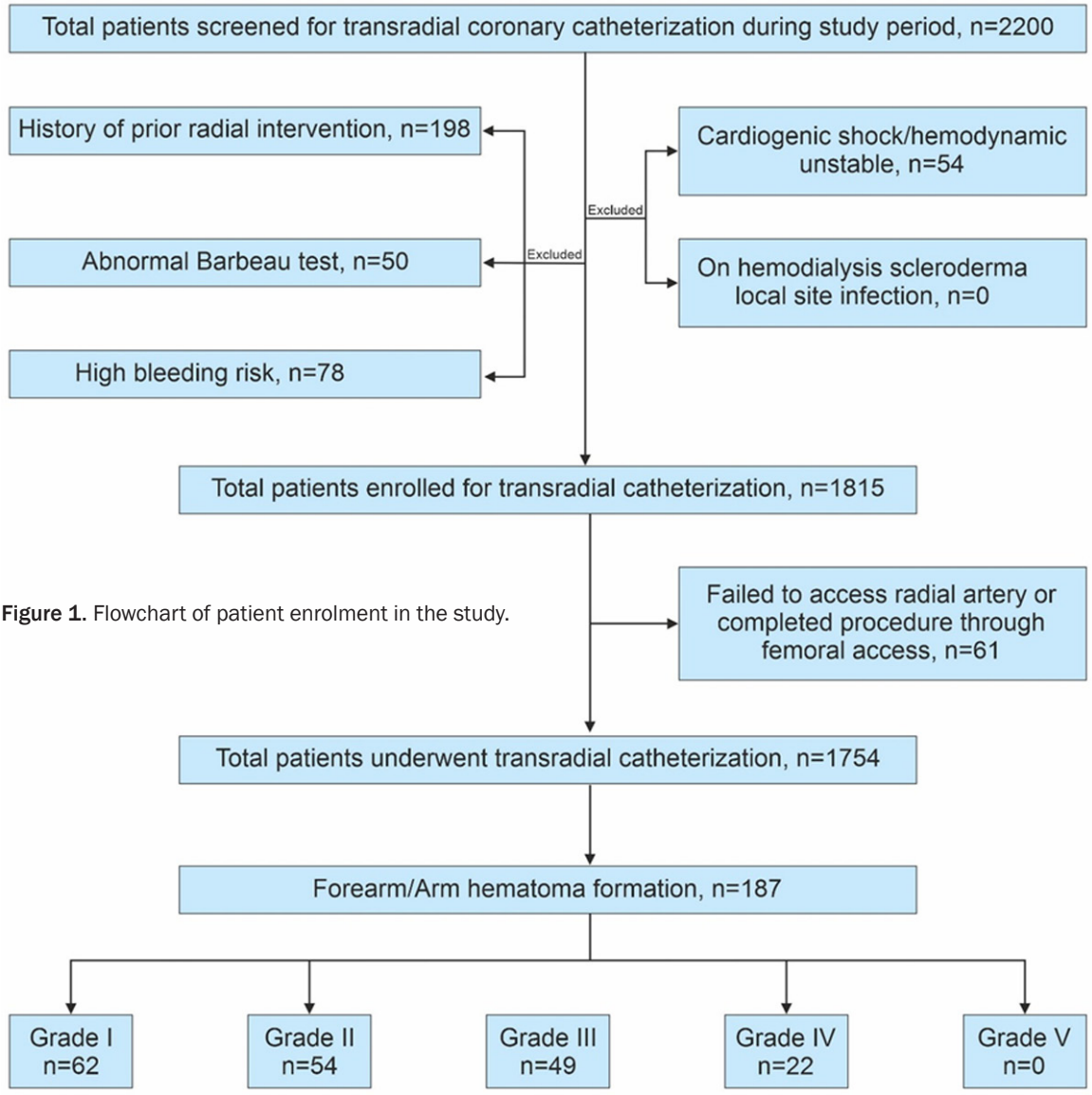

Figure 1. Flowchart of patient enrolment in the study.

hematoma. At discharge, the hematoma resolved in majority of the patients (n=158; 84.5%) and only 29 (15.5%) patients had a residual hematoma.

There were no significant age differences in patients who did and who did not develop a hematoma. Most patients who developed a hematoma were males, required multiple puncture attempts, and received a 7F sheath. More patients who developed a hematoma received intensive antiplatelet therapy and underwent a complex procedure when compared to those who did not (31.6% vs. 15.3% and 18.2% vs. 6.5%, respectively) (**Table 1**).

Predictors of forearm hematoma formation

On univariate analysis, female gender (OR: 1.936; 95% CI: 1.369-2.738), haemostasis time >360 minutes (OR: 1.004; 95% CI: 1.002-1.005), multiple puncture attempts (OR: 1.953; 95% CI: 1.440-2.649), larger sheath size (OR: 0.964; 95% CI: 0.665-1.398), intensive antiplatelet treatment (OR: 2.549; 95% CI: 1.818-3.572), and complex nature of procedure (OR: 3.192; 95% CI: 2.092-4.869) were predictors for post TCI hematoma formation (**Table 2**). Multivariate analysis yielded similar predictors (**Figure 2**). Clinical factors like age, presence of risk factors like hypertension, diabetes melli-

Table 1. Characteristics of study population and incidence of forearm hematoma with different clinico-demographic factors

Patient characteristics	Total (n=1754)	Hematoma	
		Present (n=187)	Absent (n=1567)
Age (mean ± SD, years)	56.3±10.6	56.2±11.3	56.3±10.5
Female, n (%)	312 (17.8%)	52 (27.8%)	260 (16.6%)
Body weight (mean ± SD, kg)	64.4±9.0	64.2±9.1	64.4±9.0
Hypertension, n (%)	666 (38.0%)	68 (36.4%)	598 (38.2%)
Diabetes mellitus, n (%)	622 (35.5%)	67 (35.8%)	555 (35.4%)
Smoking, n (%)	570 (32.5%)	70 (37.4%)	500 (31.9%)
Hemoglobin (mean ± SD, g/dl)	12.8±1.7	12.6±1.9	12.7±1.6
Serum Creatinine (mean ± SD, mg/dl)	1.06±0.46	1.02±0.26	1.06±0.47
GFR (mean ± SD, ml/min)	73.9±24.5	73.2+22.9	74.1±24.7
Homeostasis time, n (%)			
≤360 min	802 (45.7%)	49 (26.2%)	753 (48.1%)
>360 min	952 (54.3%)	138 (73.8%)	814 (51.9%)
GFR (ml/min), n (%)			
≤60	542 (30.9%)	58 (31.0%)	484 (30.9%)
>60	1212 (69.1%)	129 (69.0%)	1083 (69.1%)
Multiple puncture attempts, n (%)			
No	1091 (62.2%)	89 (47.6%)	1002 (63.9%)
Yes	663 (37.8%)	98 (52.4%)	565 (36.1%)
Contrast volume (ml), n (%)			
<100 ml	388 (22.1%)	40 (21.4%)	348 (22.2%)
≥100 ml	1366 (77.9%)	147 (78.6%)	1219 (77.8%)
Sheath size, n (%)			
5F	378 (21.6%)	40 (21.4%)	338 (21.6%)
6F	1367 (77.9%)	140 (74.9%)	1227 (78.3%)
7F	9 (0.5%)	7 (3.7%)	2 (0.1%)
Antiplatelet treatment, n (%)			
Conventional	1455 (83.0%)	128 (68.4%)	1327 (84.7%)
Intensive	299 (17.0%)	59 (31.6%)	240 (15.3%)
Heparin dose, n (%)			
5000 units	380 (21.7%)	40 (21.4%)	340 (21.7%)
>5000 units	1374 (78.3%)	147 (78.6%)	1227 (78.3%)
Diagnostic vs. therapeutic procedure, n (%)			
Angiography	380 (21.7%)	40 (21.4%)	340 (21.7%)
Angioplasty	1374 (78.3%)	147 (78.6%)	1227 (78.3%)
Complexity of procedure, n (%)			
Simple	1618 (92.2%)	153 (81.8%)	1465 (93.5%)
Complex	136 (7.8%)	34 (18.2%)	102 (6.5%)

Continuous variables are reported in mean (standard deviation). GFR: Glomerular filtration rate; SD: Standard deviation. Intensive antiplatelet therapy: Use of dual antiplatelet therapy containing ticagrelor or prasugrel and/or use of Glycoprotein IIb/IIIa inhibitors.

tus, smoking, angiogram or angioplasty, low GFR (<60 ml/min/1.73 m^2), body weight, haemoglobin and creatinine levels were not predictors for hematoma formation.

Discussion

Forearm hematomas are a unique complication of TCI. To the best of our knowledge, present

Table 2. Binary logistic regression to evaluating predictors of forearm hematoma as a complication of transradial coronary intervention

Patient characteristics	Univariate Analysis OR (95% CI)	*P*-value
Age (mean ± SD, years)	0.999 (0.985-1.013)	0.889
Female, n (%)	1.936 (1.369-2.738)	<0.001
Body weight (mean ± SD, kg)	0.998 (0.981-1.015)	0.807
Hypertension, n (%)	0.926 (0.676-1.269)	0.632
Diabetes mellitus, n (%)	1.018 (0.742-1.397)	0.912
Smoking, n (%)	1.277 (0.932-1.749)	0.127
Hemoglobin (mean ± SD, g/dl)	0.953 (0.871-1.042)	0.287
Serum Creatinine (mean ± SD, mg/dl)	0.722 (0.423-1.231)	0.273
GFR (mean ± SD, ml/min)	0.998 (0.992-1.005)	0.629
Homeostasis time, n (%)		
≤360 min		
>360 min	2.605 (1.853-3.663)	<0.001
GFR (ml/min), n (%)		
≤60	1.006 (0.725-1.396)	0.971
>60		
Multiple puncture attempts, n (%)		
No		<0.001
Yes	1.953 (1.440-2.649)	
Contrast volume (ml), n (%)		
<100 ml		0.799
≥100 ml	1.049 (0.725-1.518)	
Sheath size, n (%)		
5F	0.989 (0.684-1.432)	0.955
6F	0.825 (0.581-1.173)	0.285
7F	30.431 (6.274-147.594)	<0.001
Antiplatelet treatment, n (%)		
Conventional		<0.001
Intensive	2.549 (1.818-3.572)	
Heparin dose, n (%)		
5000 units		0.923
>5000 units	1.018 (0.704-1.474)	
Diagnostic vs. therapeutic procedure, n (%)		
Angiography		0.923
Angioplasty	1.018 (0.704-1.474)	
Complexity of procedure, n (%)		
Simple		<0.001
Complex	3.192 (2.092-4.869)	

GFR: Glomerular filtration rate; OR: Odds ratio; SD: Standard deviation. Intensive antiplatelet therapy: Use of dual antiplatelet therapy containing ticagrelor or prasugrel and/or use of Glycoprotein IIb/IIIa inhibitors.

study was largest single-centre prospective study which enrolled real-world patient population to assess post TCI hematoma formation. In our study, forearm hematoma occurred in 10.7% patients who underwent TCI. Similar findings have been reported in a recent study from North India by Garg et al. who used the EASY hematoma scale and reported a similar incidence of about 10% in 520 patients who underwent TCI [9]. In previous studies, forearm hematoma has been reported with wide variability ranging from 0.3% to 33%; this is explained by the varying and subjective definitions of hematoma, lack of prospective multicentre studies, inclusion of highly variable patient population, and use of different type and level of anticoagulation/antiplatelet therapy [4-6, 12-14].

In our study, most hematomas were closer to the puncture area (Grade-I: 3.53% and Grade-II: 3.08%). Fewer patients developed a hematoma in the proximal forearm and arm (Grade-III: 2.83% and Grade-IV: 1.25%) and no patients had a Grade-V hematoma. Access site hematomas are said to occur after the removal of arterial introducer sheath and the proximal extension of bleeding from the access sites. Multiple puncture attempts and inadequate haemostasis can possibly explain the occurrence of access site hematomas. More proximal forearm and arm hematomas (Grades III and IV) can be caused by perforation of the radial artery or one or more of its small branches. Tortuosity, spasm, or atherosclerotic lesions in the radial artery may increase the likelihood of a radial artery puncture and the subsequent development of a hematoma. Contrary to the study by Garg et al., we report a

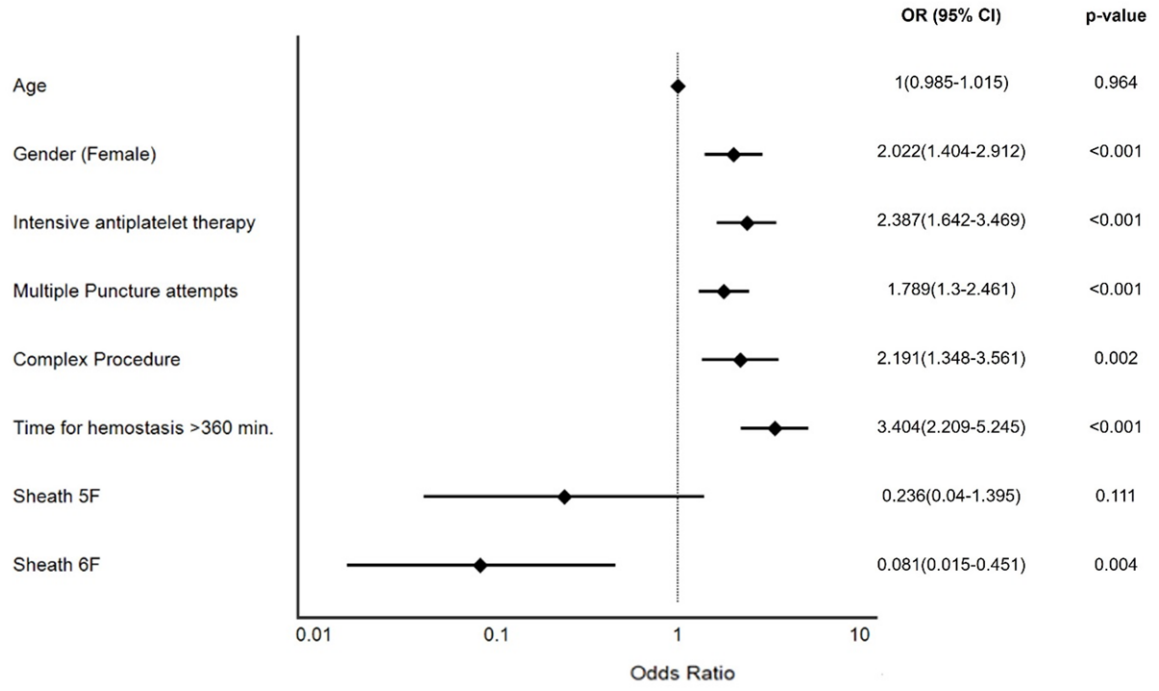

Figure 2. Forest plot based on multivariate logistic regression analysis showing predictors of forearm hematoma after transradial coronary intervention. CI: Confidence interval; OR: Odds ratio. Intensive antiplatelet therapy: Use of dual antiplatelet therapy containing ticagrelor or prasugrel and/or use of Glycoprotein IIb/IIIa inhibitors.

higher incidence of Grade-IV hematomas (0.8% vs. 1.25%) [9].

We observed a high-resolution rate (84.5%) of haematoma after conservative management during the in-hospital period and only 15.5% patients were discharged with grade I/II residual hematoma. In all these patients, the hematomas completely resolved without further complications at follow-up after two weeks of discharge. Thus, findings of the present study are similar to those reported in previous studies with almost negligible rates of major forearm hematoma formation [4, 6, 15].

Predictors of forearm hematoma

Factors like clinical characteristics of patients and interventional features impact the development of forearm hematoma after TCI. Both of these factors should be crucially considered before selection of radial artery access for coronary catheterizations. Clinical factors like age [4, 9, 16], female gender [4, 17], body mass index [9], and renal dysfunction [4, 11] are reported as predictors for forearm hematoma formation after TCI in prior studies. Our study also found female gender as an independent predictor for hematoma formation; however, age, body weight, and renal dysfunction were not found to be significant predictors.

Various interventional factors are key predictors of hematoma formation after TCI. In our study, intensive antiplatelet therapy, multiple puncture attempts, complex procedure, and longer time for haemostasis were significant independent predictors of hematoma formation (**Figure 2**). Earlier studies report similar predictors of forearm haematoma after TCI including intensive antiplatelet therapy [9], multiple puncture attempts [9], and longer duration or complexity of the procedure [4, 11], Dose of anticoagulation has shown variable effects on post TCI hematoma formation. A multicenter, randomized study compared a high dose (100 IU/kg body weight) and a standard dose (50 IU/kg body weight) of heparin during 5F or 6F coronary angiographies and reported no significant differences for the formation of local hematomas [12]. In this study, local hematomas in the radial artery were reported in 23.6% and 23.0% with the high (n=908) and standard (n=928) heparin doses, respectively. Similarly in our study, dose of heparin was not a predictor for forearm hematoma after TCI. However,

we reported dose of heparin in form of >5000 units or less than it. We did not record data for accurate total dose of heparin and any additional doses of heparin that may have been administered during prolonged procedures. On the contrary, Mattea et al. reported high intraprocedural heparinization as well as higher value of post procedure activated clotting time (ACT) as a potential risk factors associated with post TCI haematoma formation [4]. Similarly, ACT values of <150 s (n=153), 150-249 s (n=528), and ≥250 s (n=156) were significantly (P<0.0004) associated with local hematoma formation in 5.8%, 4.7%, and 16.1% patients, respectively [13]. Cumulative dose of heparin (OR: 1.017, 95% CI: 1.011-1.023, P<0.0001) and ACT values (OR: 1.004, 95% CI: 1.001-1.006, P=0.0004) were independent predictors of occlusive hemostasis after transradial catheterizations for coronary procedures in an analysis of data collected prospectively for 837 patients in an observational cohort registry [14]. Effect of sheath diameter on hematoma formation after TCI has been variably reported in different studies [4, 9, 11, 12]. We observed a significant association between use of 7F sheath and hematoma formation on univariate analysis (**Table 1**) but not on multivariate analysis (**Figure 2**).

Study limitations

Our study has certain limitations for procedural and other factors which can potentially impact hematoma formation. In our study, radial artery angiogram or colour Doppler study was not done to assess the anatomy or diameter of the radial artery. We did not follow up on the progress of hematoma from beginning to discharge and have reported only maximum grade of hematoma during the in-hospital period. Use of thrombolytic agents before TCI, history of bleeding disorders, and periprocedural activated clotting time value were not reported. Rate of radial artery spasm was not assessed which could influence the rate of hematoma formation.

Conclusions

Forearm hematoma is an easy to treat as well as preventable complication of TCIs but negligence in identification and management may lead to catastrophic complications like compartment syndrome. Our study reported 10.7% cases of forearm hematoma but none of them required vascular or surgical interventions. Female gender, multiple puncture attempts, intensive antiplatelet therapy, complex procedure, and longer hemostasis time were independent predictors of forearm hematoma formation after TCI.

The knowledge of possible risk factors can help to prevent hematoma formation after TCI and to formulate treatment strategies for hematomas of different grades. Furthermore, appropriate training, careful technique selection, timely recognition and immediate institution of corrective measures might reduce the risk of development of severe forearm hematoma. In future, larger studies are required to validate and potentiate the findings of present study as well as to identify other possible risk factors.

Acknowledgements

The authors thank Dr. Tarveen Jandoo for medical writing and editorial support in the preparation of this manuscript.

Disclosure of conflict of interest

None.

Address correspondence to: Dr. Akhil Kumar Sharma, Department of Cardiology, King George's Medical University, Shah Mina Road, Chowk, Lucknow, Uttar Pradesh 226003, India. Tel: +91-9198882220; Fax: 0522-2255830; E-mail: akhil.med@gmail.com

References

[1] Brueck M, Bandorski D, Kramer W, Wieczorek M, Höltgen R and Tillmanns H. A randomized comparison of transradial versus transfemoral approach for coronary angiography and angioplasty. JACC Cardiovasc Interv 2009; 2: 1047-1054.

[2] Mann T, Cubeddu G, Bowen J, Schneider JE, Arrowood M, Newman WN, Zellinger MJ and Rose GC. Stenting in acute coronary syndromes: a comparison of radial versus femoral access sites. J Am Coll Cardiol 1998; 32: 572-576.

[3] Tewari S, Sharma N, Kapoor A, Syal SK, Kumar S, Garg N and Goel PK. Comparison of transradial and transfemoral artery approach for percutaneous coronary angiography and angioplasty: a retrospective seven-year experience

from a north Indian center. Indian Heart J 2013; 65: 378-387.
[4] Mattea V, Salomon C, Menck N, Lauten P, Malur FM, Schade A, Steinborn F, Costello-Boerrigter L, Neumeister A and Lapp H. Low rate of access site complications after transradial coronary catheterization: a prospective ultrasound study. Int J Cardiol Heart Vasc 2017; 14: 46-52.
[5] Chugh SK, Chugh Y and Chugh S. How to tackle complications in radial procedures: tip and tricks. Indian Heart J 2015; 67: 275-281.
[6] Patel T, Shah S, Sanghavi K and Pancholy S. Management of radial and brachial artery perforations during transradial procedures--a practical approach. J Invasive Cardiol 2009; 21: 544-547.
[7] Sugimoto A, Iwamoto J, Tsumuraya N, Nagaoka M and Ikari Y. Acute compartment syndrome occurring in forearm with relatively small amount of hematoma following transradial coronary intervention. Cardiovasc Interv Ther 2016; 31: 147-150.
[8] Jue J, Karam JA, Mejia A and Shroff A. Compartment syndrome of the hand: a rare sequela of transradial cardiac catheterization. Tex Heart Inst J 2017; 44: 73-76.
[9] Garg N, Umamaheswar KL, Kapoor A, Tewari S, Khanna R, Kumar S and Goel PK. Incidence and predictors of forearm hematoma during the transradial approach for percutaneous coronary interventions. Indian Heart J 2019; 71: 136-142.
[10] Pancholy S, Coppola J, Patel T and Roke-Thomas M. Prevention of radial artery occlusion-patent hemostasis evaluation trial (PROPHET study): a randomized comparison of traditional versus patency documented hemostasis after transradial catheterization. Catheter Cardiovasc Interv 2008; 72: 335-340.
[11] Bertrand OF, De Larochellière R, Rodés-Cabau J, Proulx G, Gleeton O, Manh Nguyen C, Déry JP, Barbeau Gr, Noël B and Larose E, Poirier P and Roy L; Early Discharge After Transradial Stenting of Coronary Arteries Study Investigators. A randomized study comparing same-day home discharge and abciximab bolus only to overnight hospitalization and abciximab bolus and infusion after transradial coronary stent implantation. Circulation 2006; 114: 2636-2643.
[12] Hahalis GN, Leopoulou M, Tsigkas G, Xanthopoulou I, Patsilinakos S, Patsourakos NG, Ziakas A, Kafkas N, Koutouzis M, Tsiafoutis I, Athanasiadis I, Koniari I, Almpanis G, Anastasopoulou M, Despotopoulos S, Kounis N, Dapergola A, Aznaouridis K and Davlouros P. Multicenter randomized evaluation of high versus standard heparin dose on incident radial arterial occlusion after transradial coronary angiography: the SPIRIT OF ARTEMIS study. JACC Cardiovasc Interv 2018; 11: 2241-2250.
[13] Pacchioni A, Bellamoli M, Mugnolo A, Ferro J, Pesarini G, Turri R, Ribichini F, Saccà S, Versaci F and Reimers B. Predictors of patent and occlusive hemostasis after transradial coronary procedures. Catheter Cardiovasc Interv 2021; 97: 1369-1376.
[14] Pacchioni A, Ferro J, Pesarini G, Mantovani R, Mugnolo A, Bellamoli M, Penzo C, Marchese G, Benedetto D, Turri R, Fede A, Benfari G, Saccà S, Ribichini F, Versaci F and Reimers B. The activated clotting time paradox: relationship between activated clotting time and occlusion of the radial artery when used as vascular access for percutaneous coronary procedures. Circ Cardiovasc Interv 2019; 12: e008045.
[15] Hildick-Smith DJ, Lowe MD, Walsh JT, Ludman PF, Stephens NG, Schofield PM, Stone DL, Shapiro LM and Petch MC. Coronary angiography from the radial artery-experience, complications and limitations. Int J Cardiol 1998; 64: 231-239.
[16] Soomro NA, Khan MN, Naseeb K, Batra MK, Majeed H, Bhatti U, Ashraf T and Karim M. Outcomes of primary percutaneous coronary intervention through a transradial approach in a tertiary care cardiac center. Cureus 2019; 11: e6484.
[17] Tizón-Marcos H, Bertrand OF, Rodés-Cabau J, Larose É, Gaudreault V, Bagur R, Gleeton O, Courtis J, Roy L, Poirier P, Costerousse O and De Larochellière R. Impact of female gender and transradial coronary stenting with maximal antiplatelet therapy on bleeding and ischemic outcomes. Am Heart J 2009; 157: 740-745.