Internal Medicine
The Japanese Society of Internal Medicine
doi: 10.2169/internalmedicine.3888-24
Intern Med 64: 881-885, 2025
http://internmed.jp

【 CASE REPORT 】

Diagnostic Pitfalls of the Bleeding Origin after a Percutaneous Renal Biopsy

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Abstract:
A percutaneous renal biopsy (PRB) is a standard procedure for diagnosing renal disease, but can cause bleeding complications. Bleeding after a PRB can be classified as early- or late-onset, depending on the timing of the onset of the bleeding symptoms (<24 h or ≥24 h). We herein report two patients who experienced bleeding complications: one experienced early-onset bleeding from the 12th subcostal artery, and the other experienced late-onset bleeding from an arteriovenous fistula between a branch of the renal artery and renal vein. In both cases, the origin of the bleeding vessel was misjudged during the first examination. We discuss the diagnostic pitfalls of the origin of bleeding after a PRB and propose measures to avoid falling such pitfalls.

Key words: renal biopsy, bleeding complication, 12th subcostal artery, arteriovenous fistula

(Intern Med 64: 881-885, 2025)
(DOI: 10.2169/internalmedicine.3888-24)

Introduction

A percutaneous renal biopsy (PRB) is a standard procedure used to diagnose renal disease. Although considered safe, there are potential complications such as backache, hematoma, and hematuria (1). Bleeding is the most common complication of a PRB, and in rare cases, patients may require red blood cell (RBC) transfusion or surgical intervention, with a frequency of less than 1% (1, 2).

A recent Japanese survey showed that the incidence of bleeding complications after treatment is 1.0%. Specifically, the incidence rates of RBC transfusion and interventional radiology (IVR) are 0.7% and 0.2%, respectively (3). Bleeding after a PRB can be classified as early- or late-onset depending on the time of the onset of bleeding symptoms (<24 h or ≥24 h) (4). Reports indicate that early-onset bleeding is more common than late-onset bleeding, with early-onset rates ranging from 89-94% in North America (5, 6) and 62% in Japan (7). Early-onset bleeding has been reported to primarily originate from the branches of the renal artery, arteriovenous fistula (AVF), or renal capsular branch (8). However, rare cases of bleeding from other arteries have also been reported (9, 10). In contrast, late-onset bleeding has mostly been attributed to AVFs (8, 11, 12).

We herein report two patients who received RBC transfusion and arterial embolization to manage bleeding complications following a PRB. Patient 1 experienced early-onset bleeding from the 12th subcostal artery that was not detected by abdominal ultrasound but was identified using computed tomography. Patient 2 experienced late-onset bleeding from an AVF that was initially missed by the first IVR procedure. We discuss the diagnostic pitfalls of determining the origin of bleeding after a PRB and propose measures to prevent such errors.

Case Reports

Patient 1, a 56-year-old man with systemic sclerosis, polymyositis, and interstitial pneumonia, was referred to our department for the evaluation of a rapidly progressive decline in the kidney function. The patient's height and body weight were 170 cm and 62 kg, respectively. The blood pressure on admission was 156/78 mmHg, and heart rate

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Received: March 24, 2024; Accepted: June 12, 2024; Advance Publication by J-STAGE: August 8, 2024
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Table. Laboratory Date on Admission.

Parameter	Value	
	Patient 1	Patient 2
Complete blood cell count		
White blood cells, /μL	10,030	7,080
Hemoglobin, g/dL	10.2	9.7
Platelets, $\times 10^4$/μL	14	21.1
Blood chemistry		
Aspartate aminotransferase, IU/L	24	11
Alanine aminotransferase, IU/L	27	9
Alkaline phosphatase, IU/L	60	89
Total protein, g/dL	5.6	6.8
Albumin, g/dL	3.4	3.2
Triglycerides, mg/dL	358	50
Total cholesterol, mg/dL	328	161
Low-density lipoprotein cholesterol, mg/dL	205	93
Blood urea nitrogen, mg/dL	48.6	30.4
Creatinine, mg/dL	3.09	2.08
eGFR, mL/min/1.73 m^2	18	26
Uric acid, mg/dL	8.7	7.4
Sodium, mEq/L	143	141
Potassium, mEq/L	4.7	4.7
Chloride, mEq/L	110	107
Calcium, mg/dL	8.5	8.5
Inorganic phosphate, mg/dL	3.7	3.1
C-reactive protein, mg/dL	0.23	4.22
Hemoglobin A1c, %	5.8	5.5
Blood glucose, mg/dL	120	124
Coagulation system		
PT, %	104	74
PT-INR	0.98	1.21
APTT, %	89	39.1
D-dimer, μg/mL	<1.0	1.1
Urinalysis		
pH	5.5	6
Protein, g/g・creatinine	2.03	0.45
Red blood cells, /μL	7	691.8
Creatinine, mg/dL	61	111.4
β2-microglobulin, μg/L	1,820	
α1-microglobulin, mg/L		36
N-acetyl-β-D-glucosaminidase, U/L		18.2

eGFR: estimated glomerular filtration rate, PT: prothrombin time, PT-INR: international normalized ratio, APTT: activated partial thromboplastin time

was 82/min. Laboratory data are presented in Table. The patient did not exhibit any bleeding tendencies. The antiplatelet agents sarpogrelate and beraprost, which had been prescribed for the treatment of Raynaud's disease, were discontinued three days before a renal biopsy according to the kidney biopsy guidebook published by the Japanese Society of Nephrology (7). Ultrasonography revealed no apparent renal atrophy, with the major axis and cortical thickness measuring 88 mm and 15 mm, respectively. A renal biopsy was performed with 18-G needle punctures, inserted caudally towards the lower pole of the left kidney, three times.

The clinical course of the PRB is shown in Fig. 1a. Only a small amount of hematoma near the biopsied kidney was detected immediately after the biopsy by ultrasound. The patient had transient hypotension (91/74 mmHg) and nausea 3 hours after the biopsy. The patient had not reported any pain at that time. A blood test at that time showed a mild drop in hemoglobin (Hb) level (from 10.4 to 9.0 g/dL). Ultrasonography did not detect any increase in the hematoma surrounding the biopsied kidney (data not shown), and macrohematuria was absent, thus ruling out excessive bleeding from the kidney. A blood test was performed again 5 h after the biopsy, and the Hb level was further declined (from 9.0 to 8.3 g/dL). To determine the bleeding site, contrast-enhanced computed tomography (CT) was performed, and extravasation from the 12th subcostal and pelvic arteries was suspected (Fig. 2a). Angiography revealed extravasation from the left 12th subcostal artery (Fig. 2b, c), and the artery was embolized. The patient received an RBC transfusion, and the Hb level did not decrease again. A diagnosis of thrombotic microangiopathy with 80-90% tubulointerstitial damage was made.

Patient 2, a 71-year-old man diagnosed with IgA nephropathy and rheumatoid arthritis four years ago was admitted to our department for a re-biopsy to evaluate progressive decline in kidney function. He was treated with prednisolone, salazosulfapyridine, and tacrolimus for rheumatoid arthritis. Over the course of four years, his serum creatinine level had increased from 1.00 mg/dL to 2.08 mg/dL. The patient's height and body weight were 168 cm and 74.3 kg, respectively. Upon admission, his blood pressure was 93/52 mmHg and heart rate was 72/min. Given his low blood pressure, the antihypertensive drugs and diuretics were discontinued. On the second day of admission, his blood pressure stabilized at-130-150/80-90 mm Hg. The laboratory data are presented in Table. Ultrasonography revealed no apparent renal atrophy, with the major axis and cortical thickness measuring 92 mm and 14 mm, respectively. Renal biopsy was performed with 18 G needle punctures, inserted caudally towards the lower pole of the left kidney, four times. The clinical course of a PRB is shown in Fig. 1b. Ultrasonography detected a very small hematoma surrounding the kidney soon after a PRB, but it did not increase the following day. However, the patient experienced transient loss of consciousness, left-sided back pain, low blood pressure (91/73 mmHg), and a decrease in Hb level (from 11.4 to 9.2 g/dL) three days after a PRB. Macrohaematuria was not observed. Contrast-enhanced CT revealed an increased hematoma on the dorsal side of the kidney and extravasation of contrast medium within the hematoma (Fig. 2d). Although angiography of the renal capsular artery and renal artery did not reveal any apparent extravasation, embolization was performed on the renal capsular artery based on contrast-enhanced CT findings, and the patient received RBC transfusion. The Hb level, which initially increased after embolization, decreased again on day 6, and plain CT revealed an increase in the he-

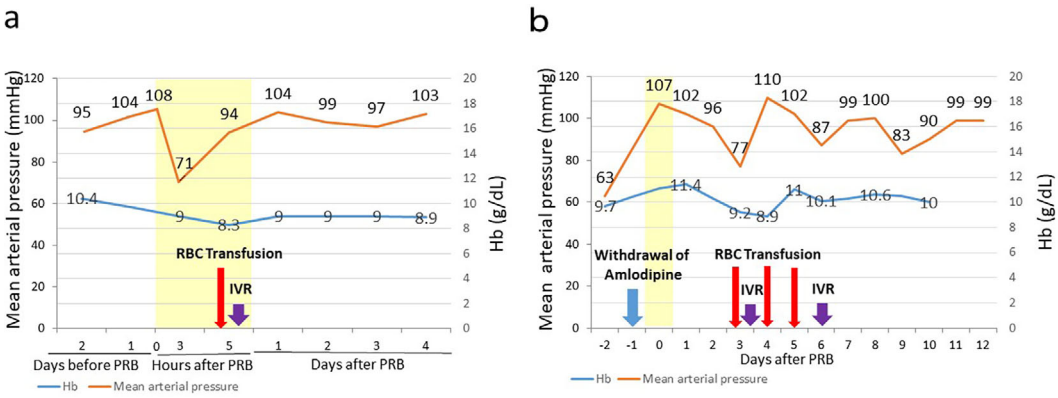

Figure 1. **Clinical course after admission in Patient 1 (a), and Patient 2 (b). Levels of mean arterial pressure (red line) and Hb (blue line) are shown. The yellow background represents the date of the biopsy. Hb: hemoglobin, IVR: interventional radiology, RBC: red blood cell, PRB: percutaneous renal biopsy**

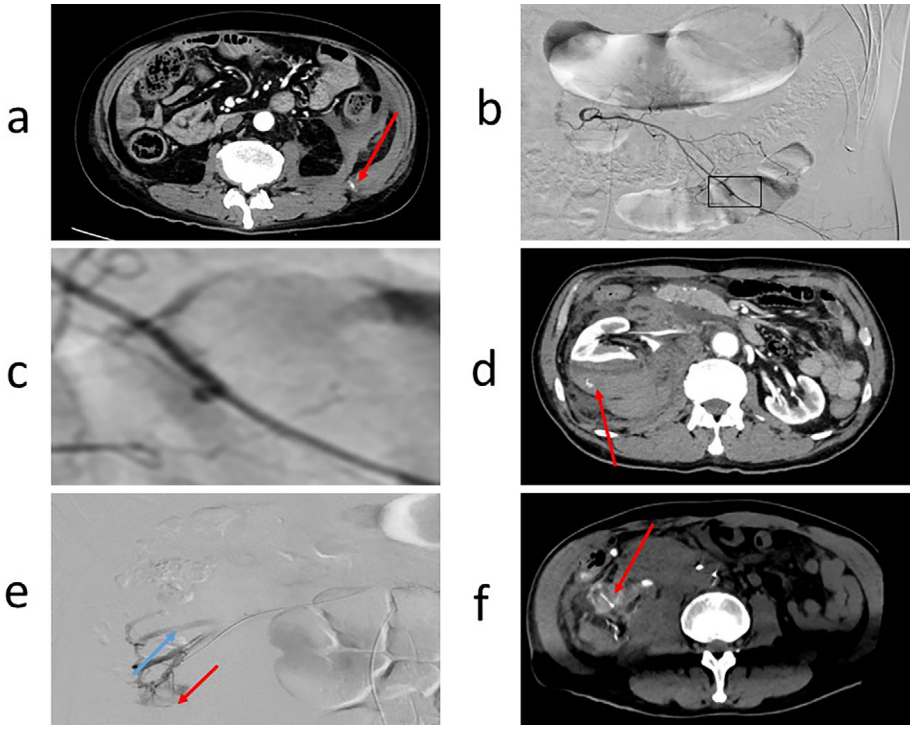

Figure 2. **Computed tomography (CT) and angiography images in Patients 1 (a-c) and 2 (d-f). Patient 1: (a) Contrast-enhanced CT showing extravasation from the artery outside the kidney (red arrow). (b) Angiography revealing bleeding from the 12th subcostal artery. (c) Higher magnification of the fragment shown in (b). Patient 2: (d) Contrast-enhanced CT revealed hemorrhage in the hematoma (red arrow). (e) Angiography showing AVF formation between the branch of the renal artery feeding the lower pole (red arrow) and renal vein (blue arrow). (f) Contrast-enhanced CT during the second interventional radiology procedure. The vessel surrounding the right kidney is enhanced soon after injecting contrast media into the renal artery (red arrow). AVF: arteriovenous fistula**

matoma size, suggesting continuous or recurrent bleeding. Angiography detected an AVF between a branch of the renal artery and the renal vein (Fig. 2e); thus, the artery was embolized. The blood vessels in the lower pole of the kidney were also imaged immediately after injection of the contrast agent into the branch of the renal artery (Fig. 2f), suggesting the formation of an AVF between a branch of the renal artery and the renal capsular vein. In addition, the renal capsular artery running in the lower pole of the kidney that was embolized previously was enhanced after contrast media injection into the renal artery, and the artery was embolized again in this case. After IVR, the Hb level stabilized, and the patient was discharged on day 12. A diagnosis of advanced sclerosing glomerulopathy due to IgA nephropathy

with 50-60% tubulointerstitial damage and apparent arteriosclerosis was made.

Discussion

There are several directions of puncture for a renal biopsy, and we puncture the kidney caudally toward the lower pole of the kidney. In addition to the hematoma surrounding the puncture site in the kidney, which originated from bleeding from the renal parenchyma, we encountered two possible bleeding patterns using this approach: bleeding due to injury to the subcostal artery, which may run near the puncture route, and hemorrhaging around the lower pole of the kidney due to injury to the capsular arteriovenous vessels, which could occur when the puncture tip penetrates the vessel during the puncture and reaches the kidney. Since the cause of this hemorrhaging was AVF formation in the capsular arteriovenous vessel, no gross hematuria was observed, so this case was considered to be one of late-onset bleeding. Both hemorrhaging points tend to be difficult to identify on echography during the initial bedside examination, so further investigation into these possibilities is important.

In Patient 1, a decrease in blood pressure suggested bleeding complications, but ultrasonography could not detect an increase in perirenal hematoma three hours after a PRB. The principal bleeding vessels after a PRB have been reported to originate from a branch of the renal artery, AVF, or renal capsular branch (8). However, some cases of extrarenal vascular hemorrhaging have been reported, such as those in the mesenteric artery or lumbar artery as the potential origin of bleeding after a PRB (9, 10).

To our knowledge, bleeding complications after a PRB from the 12th subcostal artery have not been reported. The 12th subcostal artery arises directly from the thoracic aorta and passes behind the kidneys (13). The lumbar arteries run parallel to the intercostal and subcostal arteries (14), increasing the risk of puncturing these arteries during a PRB. In Patient 1, we performed a renal biopsy of the left kidney. Anatomically, the left kidney tends to be located cranial to the right kidney (15), increasing the risk of puncturing the 12th subcostal artery during the procedure. In this case, the left kidney was located cranial to the right. In addition, the puncture site was slightly lateral, and the 12th subcostal artery descended along the 12th costa. Therefore, operators should recognize the possibility that the extrarenal arteries may lie in the route of the biopsy needle. Color Doppler ultrasound has been reported as a useful tool for detecting arteries around the kidneys (16). Therefore, we propose adding a check for arteries around the kidneys as part of the routine screening before a PRB. In the current case, we could not detect an increase in the size of the hematoma around the kidney by ultrasound because the hematoma was located far from the kidney. Hematoma does not necessarily form in the perirenal area during extrarenal bleeding. Therefore, CT should be considered if bleeding is clinically suspected but ultrasonography cannot detect signs of bleeding after a PRB.

In Patient 2, symptoms of bleeding, such as a decline in blood pressure and an increase in back pain appeared three days after a PRB. The size of the hematoma around the kidney did not increase, and the Hb level did not decrease one day after a PRB. However, on day 3, an increase in hematoma was observed on ultrasonography, along with a decline in Hb levels. These findings suggest that bleeding may have continued slowly and/or that there may have been a transient spasm, leading to gradual progression of the hematoma. According to recent surveys in Japan (3), in cases of severe bleeding requiring IVR after a PRB, bleeding occurring beyond 24 h accounts for 35%, while bleeding occurring after 7 days accounts for 20%, indicating that late-onset bleeding is not uncommon among severe bleeding cases. Therefore, caution should be exercised regarding late-onset bleeding after a PRB.

CT is reportedly more effective than ultrasound in detecting renal biopsy-related hemorrhaging (17). In addition to the higher hematoma detection ability, CT might be useful for predicting the vessels responsible for bleeding using contrast media. However, this finding is not direct evidence but merely a suggestion. In the present case, contrast-enhanced CT showed extravasation of contrast medium within the hematoma, suggesting bleeding from the vessels surrounding the kidney. Therefore, we performed the first IVR procedure to determine the origin of bleeding mainly in the renal capsular arteries, resulting in insufficient exploration of the branches of the renal artery and missing bleeding from the AVF between a branch of the renal artery and renal capsular vein. It has been reported that 20-30% of initial IVR procedures fail to resolve iatrogenic renal bleeding, thus necessitating repeat IVR (7, 18). The bleeding site is difficult to locate because of the tamponading effect of the hematoma, hypotension, or transient spasm (7). In particular, subtle, slowly progressive bleeding, such as late-onset bleeding, may be difficult to detect using IVR.

In Patient 2, the estimated glomerular filtration rate calculated from serum creatinine on admission was 26 mL/min/1.73 m^2, and histology revealed advanced glomerulosclerosis/tubulointerstitial damage with apparent arteriosclerotic change. Some risk factors for bleeding complications after a PRB have been reported (19-21). This patient had a risk factor of advanced renal insufficiency, notably a glomerular filtration rate <30 mL/min/1.73 m^2 (2). The bleeding risk after surgery has been reported to increase as the renal function decreases (22). In addition, a report demonstrated that the presence of significant (>40%) interstitial fibrosis is a risk factor for bleeding complications after a PRB (23). Arteriosclerosis has also been suggested as a risk factor for bleeding complication (24). These factors may have contributed to the onset of bleeding complications in this case.

We encountered two patients who experienced bleeding complications after a PRB; however, the initial examination failed to identify the origin of the bleeding vessels in both cases. The limitation of the ability to detect the bleeding ori-

gin by ultrasound and CT should be recognized. Although IVR can be an appropriate treatment to resolve active bleeding, the origin of bleeding can be difficult to detect, especially in cases of late-onset bleeding where AVF formation should be suspected.

The authors state that they have no Conflict of Interest (COI).

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