

CASE REPORT

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Renal infarction caused by fibromuscular dysplasia

Gurkan Yurteri

ABSTRACT

Introduction: Fibromuscular dysplasia (FMD) is an atherosclerotic/non-inflammatory disease, usually involving the renal and internal carotid arteries. Renal infarction is a rare condition that can present with flank pain and can be caused by FMD.

Case Report: A 49-year-old female patient was admitted to the emergency department with complaints of flank pain, fever, nausea, vomiting, and chills. Her blood pressure was normal. Abdominal examination showed right costovertebral tenderness. Laboratory assessment showed hematuria, leukocytosis, increased C-reactive protein (CRP), and increased lactate dehydrogenase (LDH). Nephrolithiasis was strongly considered a cause of pain because of the nature of the pain and the presence of hematuria. The urinary system ultrasound was normal. On the computed tomography (CT), in contrast-enhanced sections, an infarct area was detected in the lower pole of the right kidney. In renal angiography, severe focal stenosis was seen in the polar renal artery, and angioplasty was made.

Conclusion: Renal infarction can be caused by FMD and may be misdiagnosed as renal colic.

Keywords: Fever, Flank pain, Fibromuscular dysplasia, Renal infarction

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INTRODUCTION

Fibromuscular dysplasia (FMD) is a disease that causes stenosis, aneurysm, dissection, and occlusion in the arteries. It is not due to atherosclerosis or inflammation and usually involves the renal and internal carotid arteries [1]. Renal infarction is a rare condition; this diagnosis is made only in 0.007% of patients in the emergency room [2]. Renal infarction is often caused by atherosclerosis and vasculitis, while it is rarely caused by fibromuscular dysplasia. Renal infarction is challenging to diagnose because it presents with non-specific abdominal pain [3]. The average time until the diagnosis of renal infarction after admission is two days [4]. In this report, we present a case of flank pain presenting to the emergency department (ED) and discuss the evaluation and overall treatment of renal infarction due to FMD.

CASE REPORT

A 49-year-old female patient without a known disease was admitted to the ED with flank pain, fever, nausea, vomiting, and chills. She did not have a history of drug use and her family history was normal. The pain was colicky and severe. Her fever was 37.4–38°C, and her blood pressure was normal. Except for right costovertebral tenderness, other system examinations were normal. Her laboratory assessment showed hematuria, leukocytosis $18.1 \times 10^9/L$, increased CRP: 126.5 mg/L (<5 mg/L), and normal kidney and liver function tests [blood urea nitrogen (BUN) 20 mg/dL, creatinine 0.6 g/dL]. Nephrolithiasis was strongly considered a cause of pain but ultrasonography was found to be normal. For differential diagnosis LDH was measured and found to be 1422.3 U/L (135–225). In contrast-enhanced CT sections, an infarct area was detected in the lower pole of the right kidney (Figure 1). Enoxaparin sodium 2×0.4 mL SC was administered as an anticoagulant. Tests were negative to exclude possible vasculitis (anti-double stranded DNA, antineutrophilic cytoplasmic antibody, anticardiolipin IgG, IgM, Venereal Disease Research Laboratory Tests).

The patient underwent renal angiography two days later. Focal severe stenosis was detected in the polar renal artery at the postero-inferior level (Figure 2). Then, balloon dilatation was performed.

On the third day of hospitalization, the patient no longer needed analgesics. On the fourth day, LDH and CRP values were normal. Her hematuria disappeared, and antiaggregant (acetylsalicylic acid 300 mg) 1×1 treatment was started. She is now being followed up in the nephrology out-patient clinic with normal glomerular filtration rate.

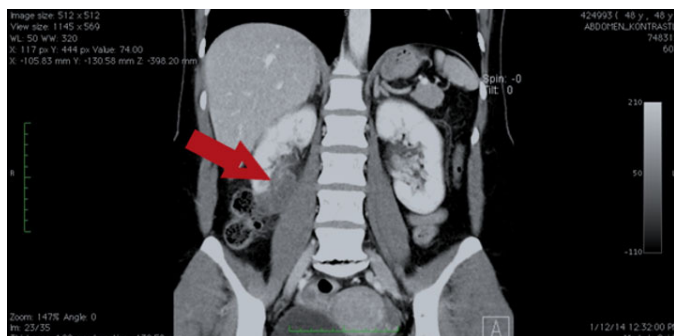


Figure 1: Computed tomography scan.



Figure 2: Angiography.

DISCUSSION

The incidence of renal infarction is very low. In a study conducted on emergency room patients, the incidence was 0.004–0.007% [5, 6]. Its frequency is 0.14% in autopsy sets [7]. Early diagnosis is difficult due to non-specific findings. The most common results in renal infarction patients are abdominal pain (53%), flank pain (50%), acute hypertension (48%), nausea (16.9%), vomiting (13%), and fever (10%) [8]. These findings can be seen in nephrolithiasis and pyelonephritis. High LDH is seen in 90.5% of renal infarctions [9]. In our case, all clinical and laboratory findings were detected except HT.

In 64% of the cases in the literature, the leading cause was a thromboembolic event due to atrial fibrillation [10]. It can also be seen in coagulation disorders, vasculitis, traumas, and diseases that disrupt the vascular structure, such as Ehlers–Danlos syndrome, Marfan syndrome, and FMD [6]. In the biochemical and radio diagnostic examinations done on our patient, no reason other than FMD was found. In FMD, the fibromuscular band

folds and aneurysm enlargements may cause thrombi formation due to platelet activation [11].

Renal artery pathologies are seen bilaterally at 35–50% [12]. In our case, the left kidney vascular structure was normal.

In the past, selective renal arterial angiography was the gold standard for diagnosis. Angiographic classification includes the multifocal type, with multiple stenosis, and the “string-of-beads” appearance that is related to medial FMD, and tubular and focal types, which are not clearly related to specific histological lesions. Today, non-invasive methods such as Doppler sonography, CT scans, and magnetic resonance imaging (MRI) scans are performed [13].

The main goal of treatment is to prevent persistent parenchymal loss and control hypertension (HT). Since our patient had a loss of renal parenchyma, renal angiography and angioplasty were performed for diagnosis and treatment. Fibromuscular dysplasia patients may present with renal colic because of renal ischemia, as in our case. However, the absence of hypertension and its course with RI is a rare condition in FMD [14, 15].

CONCLUSION

In conclusion, FMD should be considered in the etiology of renal infarction in patients even if they are normotensive and presenting with the symptoms of renal colic.

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Author Contributions

Gurkan Yurteri – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all

aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

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Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Author declares no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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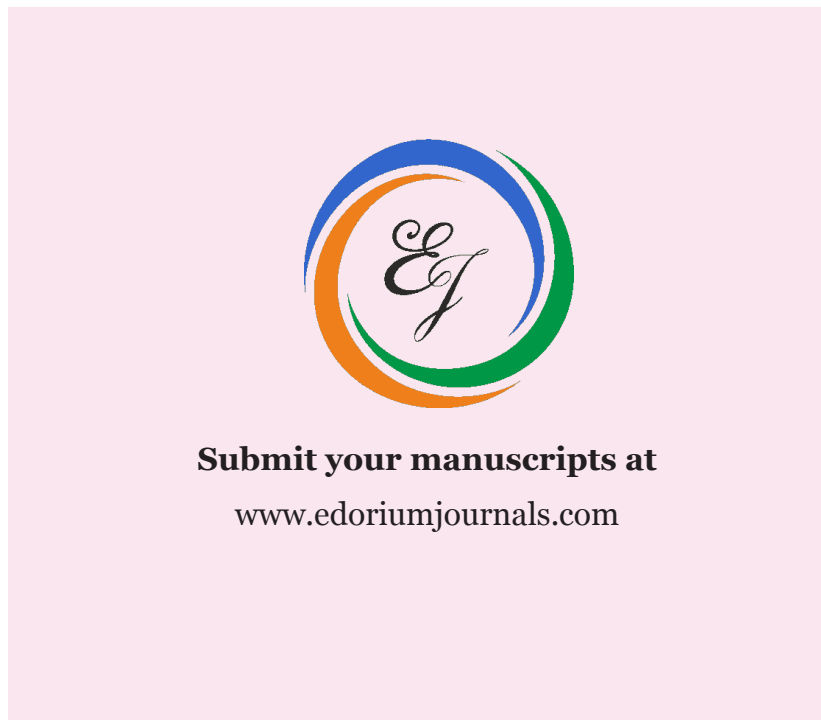
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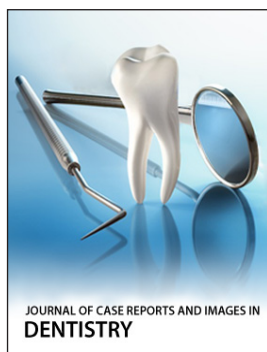
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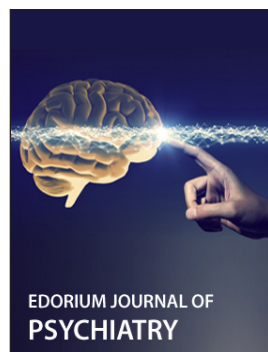
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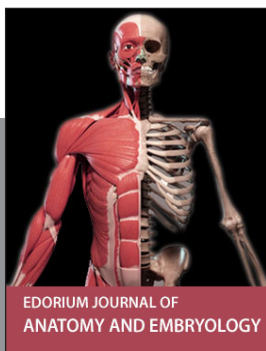
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