

CASE REPORT

Open Access



# Renal artery stenosis presenting as preeclampsia

Michael Brandon Omar<sup>1\*</sup>, William Kogler<sup>1</sup>, Satish Maharaj<sup>2</sup> and Win Aung<sup>1</sup>

## Abstract

**Background:** Renal artery stenosis is a notorious cause of secondary hypertension which classically presents as chronic refractory hypertension, recurrent flash pulmonary edema or renal insufficiency after initiation of an angiotensin converting enzyme inhibitor. Rarely, there have been reported cases of pregnant patients presenting with new onset or superimposed preeclampsia secondary to renovascular hypertension. In this subset of patients, renovascular hypertension carries significantly higher risks including obstetric, fetal and medical emergencies and death. Prompt treatment is required. However, the teratogenic risks of radiological investigations and antihypertensive medications limit diagnostic and management options thus posing quite a dilemma.

**Case presentation:** A 38-year-old female, at 33 weeks of gestation, was hospitalized for preeclampsia with severe features. A viable neonate had been expeditiously delivered yet the patient's post-partum blood pressures remained severely elevated despite multi-class anti-hypertensive therapy. Renal artery dopplers revealed greater than 60% stenosis of the proximal left renal artery and at least 60% stenosis of the right renal artery. Renal angiography showed 50% stenosis of the left proximal renal artery for which balloon angioplasty and stenting was performed. The right renal artery demonstrated less than 50% stenosis with an insignificant hemodynamic gradient, thus was not stented. Following revascularization, the patient's blood pressure improved within 48 h, on dual oral antihypertensive therapy.

**Conclusions:** Preeclampsia that is refractory to multi-drug antihypertensive therapy should raise suspicion for renal artery stenosis. Suspected patients can be screened safely with Doppler ultrasonography which can be then followed by angiography. Even if renal artery stenosis does not seem severe, early renal revascularization may be considered in patients with severe preeclampsia who do not respond to antihypertensive management.

**Keywords:** Preeclampsia, Renal artery stenosis, Renovascular hypertension, Secondary hypertension

## Background

Renal artery stenosis is a notorious cause of secondary hypertension resulting from the activation of the renin-angiotensin system in response to reduced renal blood flow. Classic presentations include chronic refractory hypertension, recurrent flash pulmonary edema and renal insufficiency after initiation of an angiotensin converting enzyme inhibitor. Although rare, there have also been

reported cases of pregnant patients presenting with new onset or superimposed preeclampsia secondary to renovascular hypertension [1, 2]. In this subset of patients, renovascular hypertension carries significantly higher risks including obstetric, fetal and medical emergencies and death. Prompt treatment is required. However, the teratogenic risks of radiological investigations and antihypertensive medications such as angiotensin converting enzyme inhibitors or aldosterone antagonists limit management options and poses quite the dilemma. When possible, expedited delivery is beneficial; notwithstanding the fact that there has been success with interventional treatment prior

\* Correspondence: [michael.omar@jax.ufl.edu](mailto:michael.omar@jax.ufl.edu)

<sup>1</sup>Department of Medicine, University of Florida College of Medicine-Jacksonville, Jacksonville, USA

Full list of author information is available at the end of the article



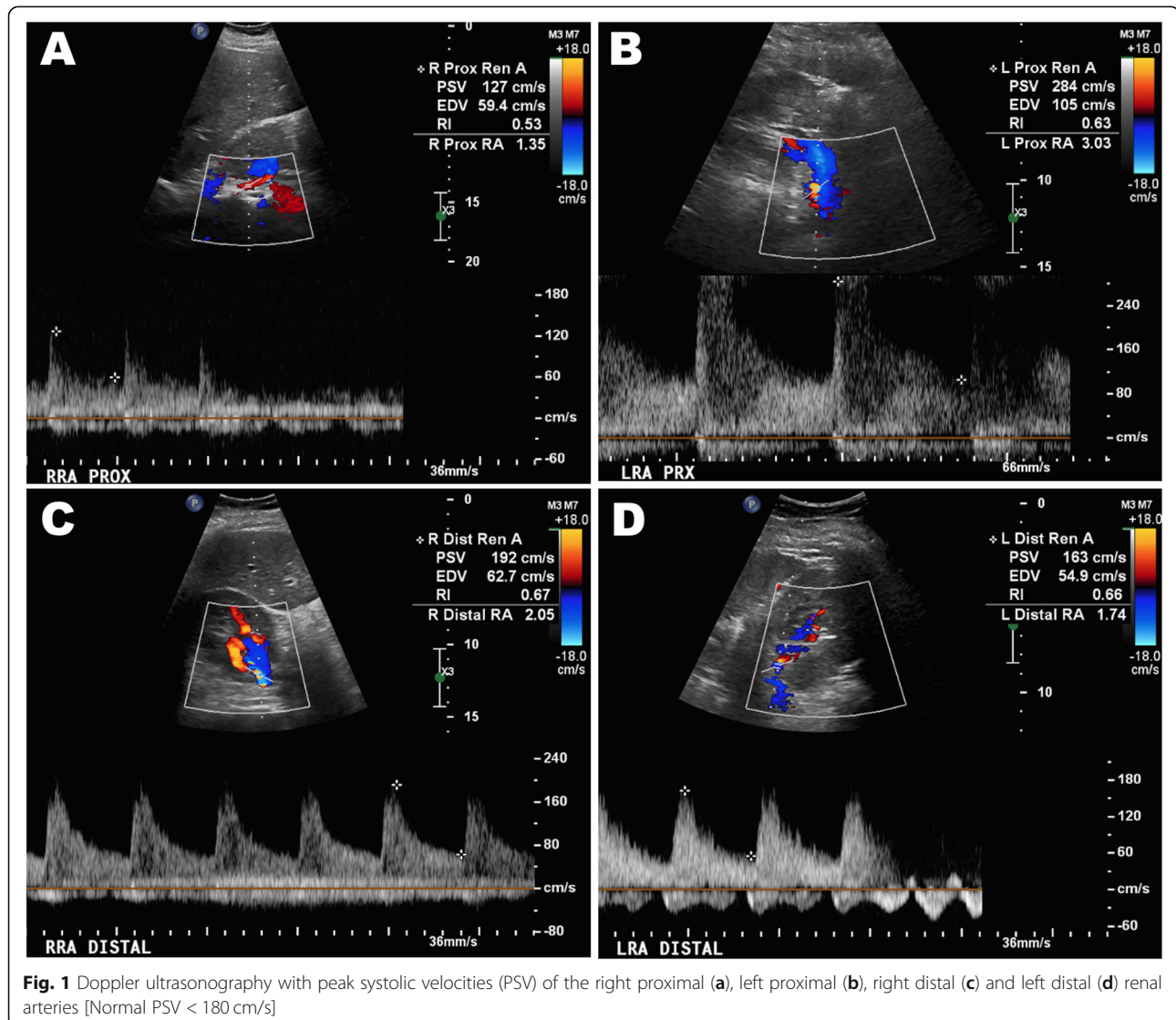
© The Author(s). 2020 **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>. The Creative Commons Public Domain Dedication waiver (<http://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article, unless otherwise stated in a credit line to the data.

to successful delivery. Furthermore, even after delivery, the mortality risk of pre-eclampsia continues into the post-partum period thus urgent and aggressive treatment strategies should continue to be pursued for these patients including consideration of early revascularization.

### Case presentation

A 38-year-old female, gravida 3 para 2 at 33 weeks of gestation, was hospitalized for preeclampsia with severe features. A viable neonate had been expeditiously delivered yet the patient's post-partum blood pressures remained severely elevated ranging from 230/130 mmHg to 280/170 mmHg. She had no antenatal care but reported a history of uncomplicated hypertension during her prior pregnancies and tobacco abuse which was stopped 8 months prior. At the bedside, she complained of mild headaches but denied visual disturbances or upper abdominal pain.

She was alert and well oriented with a pulse of 80 bpm. There was no hyperreflexia, clonus, papilledema, peripheral edema or signs of pulmonary edema. Her examination was otherwise unremarkable including the absence of renal bruits. Apart from an elevated random urine protein to creatinine ratio of 0.7, the laboratory investigations were within normal limits including serum creatinine, electrolytes, platelet count, liver function and coagulation studies. There were no laboratory features of hemolysis. She was treated with multiple anti-hypertensives over the next 72 h including oral nifedipine, labetalol and clonidine as well as intravenous infusions of labetalol, nicardipine, hydralazine. Magnesium was used for eclampsia prophylaxis. Of note, a single dose of intravenous enalapril was given with a subsequent 60% increase in serum creatinine that returned to baseline within 24 h of discontinuation. Renal artery dopplers (Fig. 1) were performed which



revealed greater than 60% stenosis of the proximal left renal artery and at least 60% stenosis of the distal right renal artery. Computerized tomography angiography showed approximately 50% stenosis of the proximal left renal artery without stenosis of the right renal artery (Fig. 2). At this juncture, in the setting of recalcitrant severe preeclampsia and the mortality risk of impending eclampsia, an invasive strategy for better evaluation and possible intervention was deemed net beneficial. Renal angiography showed 50% stenosis of the left proximal renal artery for which balloon angioplasty and stenting was performed (Fig. 3). The right renal artery demonstrated less than 50% stenosis with an insignificant hemodynamic gradient, thus was not stented. Following revascularization, the patient's blood pressure improved, ranging from 180/100 mmHg to 160/90 mmHg within 48 h, on dual oral antihypertensive therapy. She was ultimately discharged to titrate further anti-hypertensive therapy as an outpatient.

## Discussion

Renal artery stenosis is a well-established cause of secondary hypertension resulting from the activation of the renin-angiotensin system in response to reduced renal blood flow. Atherosclerosis is the most common etiology and is usually suspected in patients over the age of 45, dyslipidemic patients, or smokers. However, other etiologies such as fibromuscular dysplasia in younger patients or Takayasu's arteritis should be considered. Atherosclerotic stenosis typically affects the proximal main renal artery near the ostium compared to fibromuscular dysplasia which typically affects the distal segments.

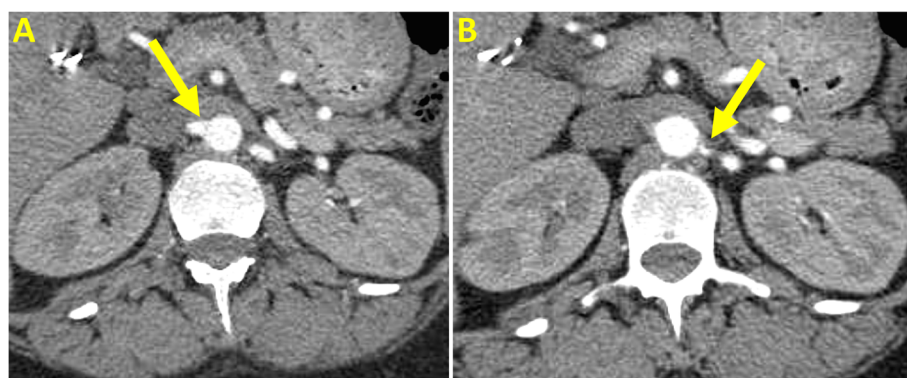
Classic presentations include chronic refractory hypertension, recurrent flash pulmonary edema and renal insufficiency- notably after initiating an angiotensin converting enzyme inhibitor (ACE-I) or angiotensin receptor blocker (ARB). Although rare, there have also

been reported cases of pregnant patients presenting with new onset or superimposed preeclampsia secondary to renovascular hypertension [1, 2].

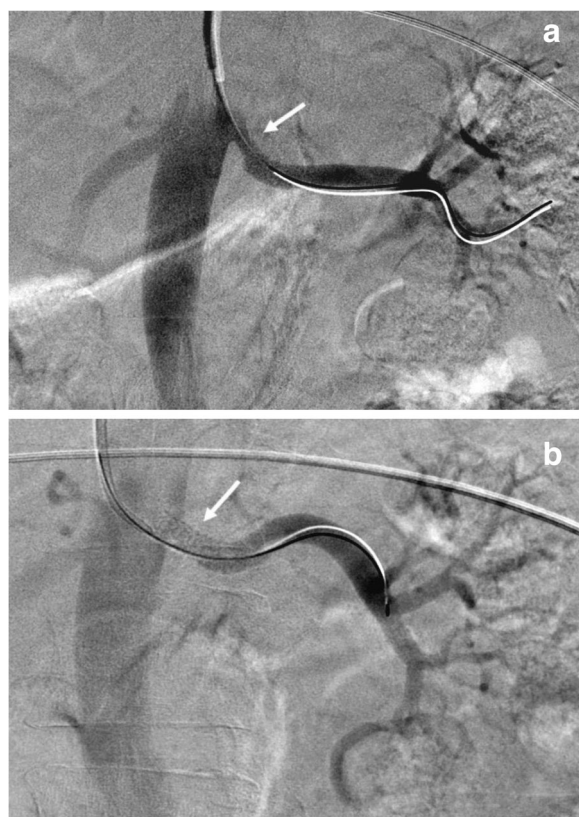
Of the different modalities used to investigate renal artery stenosis, doppler ultrasonography is the safest and has a sensitivity of at least 85%, though it frequently overestimates stenoses as in our case [3, 4]. Magnetic resonance or computerized tomography angiography have superior diagnostic accuracy with a sensitivity of 94% but the gold standard remains conventional catheter based angiography [5]. Supplemental studies such as direct renal vein renin, captopril renography or plasma renin activity to aldosterone ratios may be helpful in diagnostic dilemmas, though not currently routinely recommended [6].

Treatment may involve aggressive medical therapy with statins, antiplatelets and antihypertensives and/or renal artery revascularization. Historically, ACE-I or ARB therapy has been cautioned especially in bilateral renal artery stenosis because of the possibility of reduced post-stenotic renal perfusion pressures and subsequent ischemic nephropathy and renal failure. However, there have been observational studies suggesting a mortality benefit to closely monitored ACE-I or ARB treatment [7].

In terms of invasive treatment, percutaneous transluminal renal angioplasty with or without stenting has become the standard versus surgical revascularization. Although a recent systematic review showed only marginal benefit to this approach compared to medical therapy alone, there is evidence that select patient do have significant benefits in blood pressure control [2, 8, 9]. Furthermore, studies have shown that usually at least 80% stenosis is required to produce any significant hemodynamic stimulus to the renin-angiotensin system and thus may be a threshold for invasive treatment [10, 11]. However, as in our case, few patients have been shown to benefit from revascularization at stenoses of as



**Fig. 2** Computerized tomography of the right (a) and left (b) proximal renal arteries (arrows). Approximately 50% stenosis of the left renal artery is noted



**Fig. 3** Percutaneous renal angiography showing the proximal left renal artery (arrows) with 50% stenosis prior to stent placement (**a**) and improved flow post stent placement (**b**)

low as 50% [8, 11]. Additionally, these hemodynamic studies were performed in non-pregnant patients. Thus, whilst these data are important to avoid unnecessary procedures, clinical acumen remains necessary for select cases where revascularization of seemingly insignificant stenoses may yet produce a clinical response.

In women with preeclampsia due to renovascular hypertension, there is significant risk for obstetric and medical complications including death especially with severe preeclampsia (blood pressures more than or equal to 160/90 mmHg even without signs of end organ dysfunction or hemolysis). Prompt treatment is required. Yet, the teratogenic risks of radiological investigations and antihypertensive medications such as ACE-I/ARB's limit diagnostic and management options and pose quite a dilemma. When possible, expedited delivery is beneficial. However, there has been some success with interventional treatment prior to delivery [2]. The high mortality risk of eclampsia continues into the post-partum period and it is uncertain when blood pressures can be expected to normalize in preeclampsia even in the absence of renovascular hypertension [12]. Therefore, an urgent and aggressive management strategy

should be pursued for these patients with consideration for early revascularization if a rapid clinical response is not seen with medical management.

## Conclusions

Preeclampsia that is refractory to multi-drug antihypertensive therapy should raise suspicion for renal artery stenosis. Suspected patients can be screened safely with Doppler ultrasonography which can be then followed by angiography. Even if renal artery stenosis does not seem severe, early renal revascularization may be considered in patients with severe preeclampsia who do not respond to antihypertensive management.

## Abbreviations

ACE-I: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker

## Acknowledgements

None.

## Authors' contributions

MO was responsible for initial case writing and discussion. WK was responsible for image collection and preparation. SM was responsible for manuscript preparation/formatting and proof reading. WA was responsible for proof reading, corrections, case formatting and journal selection. The authors read and approved the final manuscript.

## Funding

None.

## Availability of data and materials

Not applicable.

## Ethics approval and consent to participate

Not applicable.

## Consent for publication

Written informed consent was obtained from the patient for publication of their individual details and accompanying images in this manuscript. The consent form is held by the authors and is available for review by the Editor-in-Chief.

## Competing interests

The authors declare that they have no competing interests.

## Author details

<sup>1</sup>Department of Medicine, University of Florida College of Medicine-Jacksonville, Jacksonville, USA. <sup>2</sup>Division of Medical Oncology and Hematology, University of Louisville, Louisville, USA.

Received: 13 September 2019 Accepted: 3 March 2020

Published online: 01 April 2020

## References

1. Heyborne KD, Schultz MF, Goodlin RC, Durham JD. Renal artery stenosis during pregnancy: a review. *Obstet Gynecol Surv.* 1991;46(8):509–14.
2. Marguerite F, Velasco S, Pourrat O, Pierre F. Successful angioplasty during pregnancy for renal artery stenosis. *J Obstet Gynaecol Res.* 2016;42:341–5.
3. Williams GJ, Macaskill P, Chan SF, Karplus TE, Yung W, Hodson EM, Craig JC. Comparative accuracy of renal duplex sonographic parameters in the diagnosis of renal artery stenosis: paired and unpaired analysis. *AJR Am J Roentgenol.* 2007;188:798–811.
4. Drieghe B, Madaric J, Sarno G, Manoharan G, Bartunek J, Heyndrickx GR, De Bruyne B. Assessment of renal artery stenosis: side-by-side comparison of angiography and duplex ultrasound with pressure gradient measurements. *Eur Heart J.* 2008;29:517–24.



5. Vasbinder GB, Nelemans PJ, Kessels AG, Kroon AA, Maki JH, Leiner T, Beek FJ, Korst MB, Flobbe K, de Haan MW, van Zwam WH, Postma CT, Hunink MG, de Leeuw PW, van Engelshoven JM, Renal Artery Diagnostic Imaging Study in Hypertension (RADISH) Study Group. Accuracy of computed tomographic angiography and magnetic resonance angiography for diagnosing renal artery stenosis. *Ann Intern Med.* 2004;141:674–82.
6. Kotliar C, Inserra F, Forcada P. Are plasma renin activity and aldosterone levels useful as a screening test to differentiate between unilateral and bilateral renal artery stenosis in hypertensive patients? *J Hypertens.* 2010;28:594–601.
7. Chrysochou C, Foley RN, Young JF, Khavandi K, Cheung CM, Kalra PA. Dispelling the myth: the use of renin-angiotensin blockade in atheromatous renovascular disease. *Nephrol Dial Transplant.* 2012;27:1403–9.
8. Raman G, Adam GP, Halladay CW, Langberg VN, Azodo IA, Balk EM. Comparative effectiveness of management strategies for renal artery stenosis: an updated systematic review. *Ann Intern Med.* 2016;165:635–49.
9. Ziakka S, Ursu M, Poulikakos D, Papadopoulos C, Karakasis F, Kaperonis N, Papagalanis N. Predictive factors and therapeutic approach of Renovascular disease: four years' follow-up. *Ren Fail.* 2008;30:965–70.
10. De Bruyne B, Manoharan G, Pijls NHJ, Verhamme K, Madaric J, Bartunek J, Vanderheyden M, Heyndrickx GR. Assessment of renal artery stenosis severity by pressure gradient measurements. *J Am Coll Cardiol.* 2006;48:1851–5.
11. Simon G. What is critical renal artery stenosis? Implications for treatment. *Am J Hypertens.* 2000;13:1189–93.
12. Podmow T, August P. Postpartum course of gestational hypertension and preeclampsia. *Hypertens Pregnancy.* 2010;29:294–300.

## Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

**Ready to submit your research? Choose BMC and benefit from:**

- fast, convenient online submission
- thorough peer review by experienced researchers in your field
- rapid publication on acceptance
- support for research data, including large and complex data types
- gold Open Access which fosters wider collaboration and increased citations
- maximum visibility for your research: over 100M website views per year

**At BMC, research is always in progress.**

Learn more [biomedcentral.com/submissions](https://biomedcentral.com/submissions)

