Clinical–Pathological Conference

Renovascular Hypertension
To Stent or Not to Stent?

Patrick B. Mark, Ernesto L. Schiffrin, Garry L. Jennings, Anna F. Dominiczak, Ji-Guang Wang, Marc De Buyzere, Jan A. Staessen

Presentation of Case
A 69-year-old women smoker was referred to the nephrology clinic for assessment of hypertension and declining kidney function. At the time of referral, serum creatinine was 241 μmol/L and office blood pressure was 191/100 mm Hg. Her general practitioner had already performed 24-hour ambulatory monitoring and found no evidence of a white coat component to the hypertension. The patient was taking 4 antihypertensive agents (nifedipine long acting 60 mg daily, candesartan 32 mg daily, bisoprolol 10 mg daily, and bendroflumethiazide 2.5 mg daily). Serum creatinine was 110 μmol/L when last recorded, 1 year before referral. Physical examination was unremarkable with negative urinalysis for blood and protein.

E.L. Schiffrin: On the history, you have a smoker with impaired renal function and no proteinuria. I think this should evoke suspicions and I thought you should comment on it.

P.B. Mark: Absolutely. The diagnosis is clear that this is probably atherosclerotic renal artery disease. I don’t think there is any debate on this. If there had been proteinuria, it would have opened the diagnosis to all kinds of glomerulonephritides. We happened to have access to 1 test that day, the ultrasound test. That wouldn’t have been the ideal test to seal the diagnosis.

G.L. Jennings: Was there an abdominal bruit? And would you like to comment on the usefulness of abdominal bruit?

P.B. Mark: I remember examining this lady and there was not an abdominal bruit. I also listened for femoral bruit. She did not have an abdominal or femoral bruit.¹

Renal ultrasound revealed asymmetrical kidneys with the left kidney measuring 8.1 cm with loss of cortical tissue. The right kidney measured 11 cm and appeared normal. The positive smoking history, renal impairment, resistant hypertension, and asymmetrical kidneys on ultrasound were highly suggestive of renovascular disease.

E.L. Schiffrin: You mentioned already renal artery stenosis, so I can ask whether you see atherosclerotic renal artery stenosis, in the absence of smoking or diabetes mellitus or other causes of severe disseminated atherosclerosis?

P.B. Mark: I would say no in general, but I have just had a similar case referred to me, which I have yet to see. I think they may need more lipid work-up. It is surprising to see a 40-year-old nonsmoker referred with atherosclerotic renal artery disease.

Her referring physician debated whether further imaging was likely to lead to alteration in management. The rapid decline in kidney function in the presence of a normal-sized right kidney with preserved cortical tissue gave rise to the possibility of remediable critical right renal artery stenosis. We considered magnetic resonance angiography, computed tomographic angiography, and formal invasive renal angiography as imaging modalities for assessment of renal artery stenosis. Impaired renal function with estimated glomerular filtration rate 18 mL/min/1.73 m² is a relative contraindication for magnetic resonance angiography, in light of the risk of nephrogenic systemic fibrosis.² Therefore, computed tomographic angiography with prehydration was performed as first choice noninvasive imaging. Computed tomographic angiography confirmed the presence of a tight calcific ostial stenosis of right renal artery (arrowed), as well as a moderately heavy aortic calcification and an atrophic left kidney (Figure 1)

Intervention With Renal Artery Stenting and Outcome
On the basis of declining kidney function, with resistant hypertension, in the presence of a critical stenosis to a single functioning kidney, we elected to proceed to renal artery intervention. The patient underwent renal artery CO₂ angiography with right renal artery angioplasty and stenting without complication (Figure 2).

For the subsequent days, there was a rapid normalization of renal function and substantial improvement in blood pressure, with creatinine falling to 92 μmol/L at 9 months post procedure. When most recently seen at clinic, 18 months post procedure, office blood pressure was well controlled (148/88 mm Hg) on 2 agents (bisoprolol and nifedipine) and serum creatinine was 122 μmol/L (estimated glomerular filtration rate 40 mL/min/1.73 m²).

The slight dip in renal function at 18 months post procedure suggests the possibility of late in-stent restenosis, although no repeat imaging has been performed to date.³ Even with this
minor dip, it is clear that this case represents successful short-to-medium-term outcome with renal artery angioplasty plus stenting for atherosclerotic renal artery stenosis. We will continue to work hard with the patient to address her other risk factors for atherosclerosis, including smoking cessation, treatment of dyslipidemia, and optimizing blood pressure control to try and protect the function of the single functioning kidney.

A.F. Dominiczak: Would you want to reimage and perhaps be ready to push the balloon across?

P.B. Mark: That is what we would like to do. However, the patient is extremely reluctant. And with the previous history of smoking and the several drugs, that is her choice. We would like to reimage. We had some debate with our radiologist because with the stent, we may have more difficulty imaging the stenosis and whether a straight angiogram might be better.

J.-G. Wang: You need to use ultrasound imaging to look at the change in the kidney and image the size of the kidney. That will tell us whether it is reversible or not reversible.

P.B. Mark: Yes, I think that is reasonable. If the kidney has become smaller or if the corticomedullary differentiation is less good, then it is possible to say that this may become less and less treatable.

E.L. Schiffrin: Have you succeeded in stopping her from smoking?

P.B. Mark: We have tried very, very hard. The answer is no. It makes you wonder about throwing all these treatments and exposing the patient to procedural risk as well.

**Successful Result in the Context of Recent Clinical Trials**

This successful result contrasts with recent well-conducted, high-profile randomized controlled trials of renal artery angioplasty and stenting compared with optimal medical therapy. The Angioplasty and Stenting for Renal Artery Lesions (ASTRAL), Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL), and Stent Placement and Blood Pressure and Lipid-Lowering for the Prevention of Progression of Renal Dysfunction Caused by Atherosclerotic Ostial Stenosis of the Renal Artery (STAR) trials have consistently failed to show benefit with renal artery stenting compared with medical treatment, in terms of either patient survival, cardiovascular events, renal function, or blood pressure. ASTRAL, which was a global study, including several patients in Glasgow, randomized 806 patients with unilater- or bilateral atherosclerotic renal artery stenosis to stenting or medical therapy and showed no difference in blood pressure, renal function, or progression to end-stage renal disease between the groups undergoing intervention compared with medical therapy. Renal artery stenting is not without risk, and in ASTRAL, serious adverse events directly related to renal revascularization were seen in 2.3% patients, including death and toe of limb amputation. The smaller STAR trial compared renal artery stenting (64 patients) to medical therapy (76 patients) and showed no overall difference in progression of renal dysfunction between the groups. More recently, CORAL in North America, randomized 947 patients with atherosclerotic renal artery stenosis and either hypertension or chronic kidney disease to renal artery stenting or medical therapy. For a median of 43 months follow-up, there was no difference in the composite end point of death from cardiovascular or renal causes, myocardial infarction, stroke, congestive heart failure, progressive chronic kidney disease, or end-stage renal disease between the stented group and those treated with medical therapy.

M. De Buyzere: Was she a good candidate to stent?

P.B. Mark: We had a good result. We have stented plenty over the years sometimes, with much poorer results. We would argue if ever there was a case for some benefit to be had, this was it. I do take your point. There was definitely established damage there, the renal function was poor, and the kidney was relatively small in size and there were other patient-related factors.

G.L. Jennings: As you just said, the results of the major trials in atherosclerotic renal artery stenosis have shown no benefit with the intervention over medical therapy. So that raises the question whether you went back a little bit further, would you really consider whether you should image her at all because medical therapy is what the guidelines are going to recommend.

This case demonstrates that there remains a group of patients who do benefit from stenting. Most clinicians accept that recurrent or flash pulmonary edema with preserved left ventricular systolic function in patients with renal artery stenosis...
is an indication for renal angioplasty ± stenting. Renal revascularization is unlikely to benefit patients with well-controlled blood pressure even on several agents and stable renal function. Small, shrunken kidneys have undergone irreversible ischemic damage, and functional improvement should not be anticipated with revascularization. Uncertainty remains in patients with renal artery disease and coexistent heart failure. The patients enrolled in these admirable clinical trials cannot represent every clinical scenario, and it is inevitable that high-risk patients, similar to the case presented, present particular diagnostics challenges and may not have been randomized in large numbers to these trials. Better characterization of the renal artery lesion using measures of fractional flow reserve may be helpful. Alternatively, functional assessment of the kidneys for hibernating renal tissue, which may benefit from revascularization, has been described. The results of these clinical trials should not deter clinicians from considering renal artery intervention, in carefully selected cases, where benefit is likely or the risks of stenting are outweighed by the likelihood of rapid progression to end-stage kidney disease in the absence of intervention.

Final Discussion of Hot Topics in Renal Artery Stenosis

A.F. Dominiczak: Can we go back to the picture with the narrowing and closing renal artery? There is a tiny, tiny flow there and it is about to close. What would happen next if nothing had been done? Well, I have had an identical patient. This was my first patient in the blood pressure unit many years ago, and we published this paper with Professor Chris Isles. What happened to the patient next was that she became anuric, the same age; everything was similar. She had malignant hypertension. She completely relied on the tiny bit of 1 closing renal artery. So this is a tightening stenosis to a sole kidney and next is dialysis. It is easy to criticize, but clearly 2 years later, this patient still does not need renal replacement therapy. So something has been achieved.

A. Brady (Glasgow): We had a lot of patients from our series in ASTRAL. For people who don’t know how we recruited it; if a patient had a stenosis like this, they never went in the trial. They got angioplasty. For all the patients who had 50% to 60% stenosis, where you weren’t sure, they sort of got put in the trial. I can’t speak for CORAL, but I bet for the CORAL centers, which are mostly North American; those patients with really severe stenosis were never included in the studies. So those trials actually tell us nothing about critical stenosis, and I think for this individual there is clear benefit.

J. Dawson (Glasgow): I would support you. I would have referred that patient for stenting. If we were to go back in time, even with the trial data I would still refer that patient for stenting. I think the more interesting question is what would I do now? Now that the renal function has declined. And the question I have to help me make my mind up is: How much of the benefit is likely or the risks of stenting are outweighed by the likelihood of rapid progression to end-stage kidney disease in the absence of intervention.

P.B. Mark: The therapy was not the same. I can’t answer the exact magnitude of each change, but the day post stenting, there was a drop in blood pressure. She had a torrential natriuresis and diuresis, blood pressure dropped, all drugs were stopped, then it was a labile evolving situation and it makes it extremely difficult to reinterpret what happened with the reintroduction of the drugs. But we didn’t reintroduce an angiotensin receptor blocker because there was no other compelling reason to do so. Although I think reintroducing any antihypertensive drug will probably lead to a relative drop in the renal perfusion again. We don’t know what her actual baseline is.

G.L. Jennings: Just a comment on the people with really tight stenosis didn’t go into these trials, so we don’t really know. CORAL did a retrospective subgroup analysis; those with a stenosis over 80% didn’t show any different from those with a stenosis <80%. You probably need 80% for it to be functionally significant.

M. De Buyzere: For functional renal reserve, do you have a proposal for a cut-off where you should do it? For pressure-wire for instance.

P.B. Mark: I don’t. For ASTRAL, I don’t recall the exact entry criteria, but it was ≈50% to 70%. It was a less severe stenosis. We have no experience pressure-wiring. We have some experimental experience of doing magnetic resonance-perfusion renography, which has been published by the Manchester group. It does look impressive for predicting response to renal revascularization, but we don’t have a big enough case series of those. I think it comes back to the ultrasound actually. If they have a decent-size cortex and a reasonable-size kidney, there is a reasonable chance it might be a good outcome. If it’s a 9 cm kidney or below, it’s unlikely to be a good result.

J. Walters (Glasgow): I am going to test Anna’s earlier assertion that there are no stupid questions. Now that the renal function is deteriorating 9 months after the stent was inserted, the question is whether it is instant restenosis or not. What is the role or is there a potential role for contrast-free imaging, using for instance time of flight magnetic resonance angiography, which would obviate any risk of contrast-induced injury to the patient but may be sufficient to answer the specific question about the presence or absence of in-stent restenosis?

P.B. Mark: I’m not sure that is a stupid question. That is way over my head. Seriously, I think that with magnetic resonance angiography, time of flight imaging is a beautiful concept. But the artifact with magnetic resonance imaging relating to the actual stent itself is going to make this difficult. I don’t have any experience with it.

J.A. Staessen: Why would you do the imaging again in this patient? Suppose you find out that the stent is thrombosed. What would you do?

P.B. Mark: I hope it is not thrombosed as the renal function would be considerably worse. I think that is an extremely difficult question to answer. If we find that there is a significant degree of in-stent restenosis, do we go back and subject the patient to another procedure and we will go round in the loop again?

E.L. Schiffrin: If we are spending so much money, or intend to, on this patient, why can’t we spend a lot of money on stopping her from smoking? Surely, this has contributed to any additional vascular damage that has occurred since the recent intervention.
J.-G. Wang: How often do you see this kind of patient? If you see rarely, 1 or 2 patients a year, I think that is not a problem. We also had a similar case as you had with a rapid renal function decline. The severity of stenosis is not a good indicator, but rapid renal decline is a good indicator.

P.B. Mark: We don’t look hard for renal artery stenosis beyond the clinical diagnosis. In the post-ASTRAL and post-CORAL age, we don’t pursue renal artery stenting as aggressively as we did in the late 1990s and 2000s. I don’t think we perform >=5 or 10 a year. We used to do many more than this.

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Disclosures
None.

References
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病例介绍

患者，女，69岁，有吸烟史，为评价其高血压和肾功能下降的程度被转诊至肾病门诊。转诊时，患者血清肌酐水平为241 μmol/L，诊室血压为191/100 mm Hg。她的全科医师之前对她进行过24小时动态血压监测，并没有发现有大型高血压的证据。该患者正服用6种抗高血压药物（硝苯地平长效制剂60 mg/d，坎地沙坦32 mg/d，比索洛尔10 mg/d，苯苄胺2.5 mg/d）。转诊前一年，最后一次记录的血清肌酐水平为110 μmol/L。体格检查无异常，尿检中血和蛋白结果为阴性。

E.L. Schiffrin；从病史上看，这是一位肾功能受损但无蛋白尿的吸烟患者。我认为这容易引起怀疑，我想你应该对此进行说明。

P.B. Mark；没错。诊断很清楚。这很可能是一例动脉粥样硬化性肾动脉疾病患者。我认为，对于这一点没有争议。如果患者有蛋白尿，那其诊断可能为某种肾小球性肾炎。那天我们刚好进行了超声检查。这并不是明确诊断的想象。

G.L. Jennings；有腹部杂音吗？你能对腹部杂音的用处发表评论吗？

P.B. Mark；我记得检查过这位女士，没有腹部杂音。我也听了股动脉杂音。她没有腹部杂音或股动脉杂音。

肾脏超声检查发现，肾脏不对称，左肾大小8.1 cm，无皮质组织。右肾大小11 cm，看似正常。有吸烟史，肾功能受损，难治性高血压，超声示肾脏不对称，以上表现高度提示肾血管疾病。

E.L. Schiffrin；你之前提到已有肾动脉狭窄，那么我想问，在没有吸烟或糖尿病，或严重弥漫性动脉硬化症其他病因的情况下，你是否看到了动脉粥样硬化性肾动脉狭窄？

P.B. Mark；通常我会说不，但是，我刚刚接诊过一位相似的病例，这个病例中我看到了肾动脉狭窄。我认为，他们可能需要更多的血脂检查。一位40岁的不吸烟者因动脉粥样硬化性肾动脉疾病转诊，这让人惊讶。

她的转诊医生辩称进一步的影像学检查是否能改变其治疗。在右肾大小正常，皮质保留的情况下肾功能快速下降，提出了关键性右肾动脉狭窄治疗的可能性。我们考虑采用磁共振血管成像、计算机断层扫描血管成像，以及侵人性肾血管造影等影像学方法来评价肾动脉狭窄。肾功能受损（估算的肾小球滤过率为18 mL/min/1.73 m²），是磁共振血管造影的相对禁忌症，因为有肾原性全身纤维化的危险。

因此，预水化和计算机断层扫描血管成像是非侵人性成像的首选。计算机断层扫描血管成像证实，右肾动脉开口存在严重的钙化性狭窄（箭头所示），以及主动脉中度钙化和左肾萎缩（图1）。

肾动脉支架介入治疗及预后

根据进行性肾功能下降，难治性高血压，单功能肾存对
在近期一些临床试验大大提高下临床的临床结果

这一临床结果与近期良好实施，引人注目的比较肾动脉血管成形与支架置入术与血管造影术的随机对照试验形成了对比。血管成形术与支架置入术治疗肾动脉病变试验（Angioplasty and Stenting for Renal Artery Lesions, ASTRAL）[4]，肾动脉粥样硬化性病变后血管预处理（Cardiovascular Outcomes in Renal Atherosclerotic LesionsCORAL）[5]，支架置入术治疗粥样硬化性病变后动脉粥样硬化性病变后解体试验（Stent Placement and Blood Pressure and Lipid-Lowering for the Prevention of Progression of Renal Dysfunction Caused by Atherosclerotic Ostial Stenosis of the Renal Artery, STAR）[6]，均未能证明肾动脉支架置入术对药物治疗更有益于，包括患者生存、心肌梗死，肾功能障碍进展试验（Stent Placement and Blood Pressure and Lipid-Lowering for the Prevention of Progression of Renal Dysfunction Caused by Atherosclerotic Ostial Stenosis of the Renal Artery, STAR）[6]，均未能证明肾动脉支架置入术对药物治疗更有益于，包括患者生存、心肌梗死，肾功能障碍进展试验（Stent Placement and Blood Pressure and Lipid-Lowering for the Prevention of Progression of Renal Dysfunction Caused by Atherosclerotic Ostial Stenosis of the Renal Artery, STAR）[6]。A.T. R. A. S. T. A. R. L. 是一项全球性研究，纳入了格拉斯哥的一些患者，研究将806例单侧或双侧动脉粥样硬化性动脉狭窄的患者随机分为支架置入组或药物治疗组。结果显示介入治疗或药物治疗的两组在血压，肾功能或终末期肾病改善方面均无差异。肾动脉支架置入术并不是没有风险，在ASTRAL研究中观察到，2.3%的患者出现与肾血运重建直接相关的严重不良事件，包括死亡和截肢手术[6]。比较肾动脉支架置入术（64例患者）与药物治疗（76例患者）结果（STAR）这一小型试验表明，两组的肾功能障碍进展无明显差异[6]。近期，美国CORAL试验将947例合并血流或慢性肾病的动脉粥样硬化性动脉狭窄患者随机分配接受支架置入或药物治疗，在中位43个月的随访中，对于心血管或肾脏原因的死亡，心肌梗死，卒中，血肌酐心力衰竭，进展性慢性肾病或终末期肾病组成的复合终点，支架置入组或药物治疗组间无明显差异[5]。

M. De Buyzere：这位患者适合支架置入术吗？

P.B. Mark：我们得到了较好的结果。这些年来我们作了大量的支架置入术，有时结果很差。我们会争辩，如果有可能会从中获益，那这例就是。我确实同意你的说法。该例患
J. Dawson (格拉斯哥)：我赞同你的观点。我本来应该推荐那些患者行支架置入术。如果我们能够回到过去，即使有目前这样的临床试验数据，我仍然会将患者转诊进行支架置入术。我想更有趣的问题是，既然患者的肾功能已经下降，现在我们能做什么？我更想确认的问题是：估计的肾小球滤过率能否恢复多少，是否能够改善其之后的变化是因

为在围术期停用血管紧张素受体拮抗剂，接着能重新开始，或者在整个过程中保持治疗不变？

P.B. Mark：治疗并不一样。我不知道患者每一个变化的确切幅度，但是，在置入支架后的那天，患者血压下降。由于大量的尿钠排泄和多尿，血压下降，患者停用了所有降压药物，之后出现不稳定的演变情况。这使得我们极难解释恢复和用药发生了什么。但是，我们没有重新使用血管紧张素受体拮抗剂，因为没有其他强制性的原因需要这样做。只是我认为，重新使用任何抗高血压药物可能导致肾脏灌注再次出现相对下降。我们不知道她确切的基线情况。

G.L. Jennings：只评论一点，我们确实不知道真正的严重狭窄患者是否参加这些试验。CORAL研究所作的回顾性亚组分析表明，这些狭窄超过80%的患者并没有显示出与狭窄＜80%的患者有任何不同。要能使功能有显著差异，可能需要狭窄达到80%。

M. De Buyzere：对于功能肾的保留，其切点值的处理你有什么建议吗？例如压力导丝。

P.B. Mark：我不知道。对于ASTRAL试验，我记不清确切的入组标准，但是，狭窄程度约在50%~70%。这并不是非常严重的狭窄。我们没有压力导丝方面的经验。我们有一些试验性经验，开展过磁共振灌注肾灌注扫描，结果由Manchester研究小组发表[12]。在预测肾脏血运重建的反应方面，其实质令人印象深刻，但是，我们没有足够的病例样本。实际上我认为，应该再次回到超声检查。如果他们的皮质大小合适，肾脏大小合适，那么可以认为，他们的结果较好。如果肾脏9 cm或更小，则不太可能有好的结果。这位患者的肾脏大小11 cm，如果肾脏大小10 cm呢？我们认为，这很难预测，还需要更多的数据。

M. Walters（格拉斯哥）：我准备检查安娜大夫前提到的那个观点，这个观点没有愚蠢的问题。既然患者的肾功能在支架置入术后9个月开始恶化，那么问题是否会有即时再狭窄。无对比剂成像发挥什么作用，或者有潜在的作用吗？例如，使用时间飞跃法磁共振血管成像（TOFMR），可以避免对比剂诱发的对患者的损害危险，但是，这能够回答有关无支架再狭窄的具体问题吗？

P.B. Mark：我肯定那不是个愚蠢的问题。只是我无法理解。严肃地说，我认为，磁共振血管造影，时间飞跃法成像是一个非常好的概念。但是，与狭窄本身相关的磁共振成像的伪影使这变得困难。我没有任何这方面的经验。
J.A. Staessen: 为什么你要为这些患者再次做成像检查呢？推测你发现支架内有血栓形成，你会怎么做？

P.B. Mark: 我希望不是血栓形成，因为那样的话，肾功能将会变得相当差。我想，这是一个非常难以回答的问题。

如果我们发现有非常明显的支架内再狭窄，那么我们要让患者再做一次手术，这不是又回到在原地了吗？

E.L. Schiffirin: 如果我们正在花费或准备花费这么多的钱在这位患者身上，那么，我们为什么不能花更多的钱使患者戒烟呢？毫无疑问，自从近期的干预之后，吸烟的危害已经发生，并且导致了更多血管的损害。

J.-G. Wang：你看到这类患者的频率是多少？如果你很少看到这类患者，一年一例或两例，我认为，这不是个问题。我们也看到过一例相似的病例，患者的肾功能快速下降，狭窄的严重程度不是一个很好的提示指标，但是，肾功能快速下降是一个很好的提示指标。

P.B. Mark: 除非临床诊断外，我们没有着重关注肾动脉狭窄，在ASTRAL和CORAL时代，我们并不像20世纪90年代后期和21世纪初那样积极开展肾动脉支架置入术。我认为，我们一年完成不了5–10例。我们之前做得比这多得多。

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声明
无。

参考文献

文献