Case of Severe Hypertension and Nephrotic Range Proteinuria

Michael Bursztyn,[1] Rhian M. Touyz,[2] Cheryl L. Laffer, [3] Robert M. Carey,[4] Anna F. Dominiczak,[2]

[1] Hadassah-Hebrew University Medical Center, Mount Scopus, Jerusalem, Israel

[2] Institute of Cardiovascular and Medical Sciences, College of Medical, Veterinary and Life Sciences, University of Glasgow, UK

[3] Department of Medicine, Vanderbilt University School of Medicine, Nashville, Tennessee, USA

[4] Division of Endocrinology and Metabolism, University of Virginia Health Sciences Center, Charlottesville, Virginia, USA

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Case Introduction

The presentation of this patient is from the perspective of an Emergency Department. A 37-year-old man with severe hypertension was referred by a family physician. This is not uncommon where, sometimes, primary care physicians feel uncomfortable managing such patients and refer them to the Emergency Department for specialist care. Following assessment, these patients are usually quickly discharged.

The patient was generally healthy; however, for the last six months, he was treated with atenolol 50 mg for hypertension and had headaches, nausea, dizziness, and left flank pains. He had no diarrhea or constipation. He lost about five kilograms of weight during this period. He has no family history of hypertension or renal disease.

On examination, he was found to be fully conscious and relaxed, and his blood pressure was 190/126 mmHg with a pulse rate of 88 bpm and with no respiratory distress. His body mass index was 19.5 kg/m^2. There were no alarming findings on fundoscopy. He had clear lungs, normal heart sounds, no abdominal or flank tenderness, no organomegaly, no peripheral edema, and normal peripheral pulses. His calf blood pressure was 232/144 mmHg, ruling out coarctation of the aorta. He had no neurologic deficit and no other findings such as skin rash or purpura.

His electrocardiogram (EKG) was normal with some voltage criteria for left ventricle hypertrophy (LVH); however, at the age of 37 years, this is of questionable significance. His biochemistry showed severe hyponatremia, severe hypokalemia, normal creatinine level, low albumin level, high cholesterol levels, with normal liver function test (Table 1). This certainly changed the perception that he is just one of those patients referred because of his primary physician's uncertainty in management. He had normal complete blood count and red blood cell morphology. Urine sediment was normal but dipstick analysis was positive for protein. This was further confirmed using sulfasalicylic acid.

Medical History

After probing further into his medical history, it was discovered that twenty years earlier he had an appendectomy, but the appendix was not inflamed. There was some non-specific iliocecal inflammation. Crohn's disease was suspected, but not followed-up. Then, a decade later, he was admitted for surgery because of severe abdominal pain. Computerized tomography (CT) revealed some small bowel thickening and perhaps peritoneal fat stranding. He was treated with antibiotics, and he recovered.

Again, a few years later, he arrived in the emergency room with calf paon and deep vein thrombosis (DVT). A workup for hypercoagulopathy was recommended but not undertaken. He was treated with enoxaparin and later warfarin, which he discontinued after a year.

Discussion: Managing the Hypertension

> **Dr. Bursztyn:** I think that severe proteinuria and severe hypertension in the absence of evident glomerulonephritis are somewhat unusual.

Dr. Sica: There is significant proteinuria and significant hypertension. To determine the degree that they are linked, you probably need to ascertain what is going on at the level of the kidney. In our current practice, in Richmond, Virginia, biopsy is probably quickly forthcoming as soon as the blood pressure has some measure of control, so they do not bleed out when you biopsy the kidney. I would be attacking it diagnostically to see if there is any renal pathology that may be acting as an approximate cause of the hypertension.

Realizing that the hypertension has not been worked up from a neurohumoral point of view, it is highly likely that there is a renal element to this. I would go for the renal pathology earlier than later in the workup. Conventional serologic tests would be conducted to diagnose what might be going on at the level of the kidney as well. The rule-outs would be there, biopsy would be shortly forthcoming, unless some miracle happened, and sometimes these miracles happen, but not often.

Dr. Bursztyn: This happened in the land of miracles.

Dr. Laffer: I would also want a workup of his hyponatremia and hypokalemia, which are rather profound. Urine osmolarity and electrolytes might be useful.

Dr. Selvarajah: Putting it simply, he has nephrotic syndrome but is not edematous. He has two interesting things: he is hypertensive, which can be associated with secondary FSGS (focal segmental glomerulosclerosis), which is a significant glomerulopathy, and he has Crohn's disease, which is associated with kidney disease. You can get interstitial nephritis which would produce blood in the urine. There are other renal pathologies that are associated with Crohn's disease. It is not clear if these are due to the treatment for Crohn's disease like 5-ASA (5-Aminosalicylic acid) agents or Crohn's *per se*. Renal biopsy would certainly be important. His electrolytes are interesting in that he is hyponatremic and hypokalemic with normal excretory function. Certainly, as mentioned, checking osmolalities and checking the renin and aldosterone levels to see if there are any clear causes of hypokalemia or checking the magnesium level as well would be relevant.

Dr. Bursztyn: Of course, in our place, renin and aldosterone tests take time, and as you can see, we are still in the emergency room. The low albumin and high cholesterol levels suggest that it is nephrotic syndrome. Nephrotic syndrome is a hypercoagulable state; however, it is usually a state of intravascular volume depletion. Therefore, unless there is glomerulonephritis, most typically lupus nephritis, these things do not go with such severe hypertension. At this time, in the emergency room, someone thought that a renal vein thrombosis could somehow link the massive proteinuria with acute or subacute hypertension.

Prof. Touyz: What treatment was he on?

Dr. Bursztyn: Atenolol.

Dr. Hamrahian: I agree with the majority of the comments made, but I will take a different approach. The first question is, how do we stabilize the patient coming in with such elevated blood pressure and electrolyte abnormality with some missing data? Just by

looking at your EKG with mild LVH voltage criteria, it tells me that despite the diagnosis of hypertension and being on atenolol, this advanced or significantly elevated blood pressure is something, which most probably is acute exacerbation of a relatively stable hypertension from the past; otherwise, he would not have a relatively normal EKG.

The second question is, what is the bicarbonate level of the patient? What is the serum osmolarity of this patient? We need the urine protein:creatinine ratio and/or albumin: creatinine ratio (ACR) that would be the second phase of workup. A thorough workup is needed. We must see if the hypokalemia is related to hyponatremia versus two different etiologies. Does this patient have, by chance, a plasma cell dysplasia giving us a pseudohyponatremia. I think there are different things that we have to separate first, but the key thing is to stabilize the patient.

Dr. Certikova Chabova: I am a clinical nephrologist, and I would strongly disagree that this patient should first have renal biopsy because, first, you cannot do renal biopsy in someone who has such high blood pressure. It would take at least one week to safely lower it. He has no critical organ damage at this moment, so his blood pressure should be lowered gradually in stepwise fashion, first thing. The second is that the proteinuria might decrease after his blood pressure goes down, and I would say that it is not sure that he has no glomerulonephritis. There are glomerulopathies that have no changes in sediment, and they have only nephrotic syndrome. This can still be glomerulonephritis. However, I will ask whether he was taking atenolol when his heart rate was at 88 bpm with such high blood pressure. High blood pressure should decrease the heart rate, and when you give atenolol it should go down. The first thing is to decrease his blood pressure because this is really dangerous, and the workup should be first, but not renal biopsy.

Dr. Bursztyn: We also did not think, at that time, that a renal biopsy would be appropriate and I just want to remind you that he is still in the emergency room. However, given the thinking, that renal vein thrombosis may somehow link the nephrotic syndrome with severe hypertension, renal ultrasound with venous Doppler was done. It did not show any renal vein thrombosis; however, it showed a discrepancy between the sizes of the kidneys. The left kidney at 8.5 cm was hyperechogenic, and the right kidney was 12.5 cm. The resistance index on the left was 0.9, and on the right, it was 0.5.

Dr. Oliveras: Before the ultrasound results, I wondered about this low potassium level because I think that the patient has a nephrotic syndrome. However, we have no information about potassium in urine. You must know before other things if the potassium in the urine is low or is high because, this patient has had two previous confusing abdominal episodes. This comes first. Maybe, the patient has something related to this hypokalemia that has nothing to do with the nephrotic syndrome, and we must think about the possibility that these two things are linked. What was the potassium in the urine?

Dr. Bursztyn: Unfortunately, I did not write it down, and I do not remember. It was low.

Dr. Phillips: He is hyponatremic and hypokalemic. Therefore, you have to think about a hyperreninemic state. He probably has a high renin level. He may have renal artery

stenosis and may have two things together. I know he has a related pathology that we will probably find on biopsy of his kidney that links together what was happening with his gastrointestinal tract for many years. However, this may be a case where you have two things: he may have renal artery stenosis and also some other glomerulopathy.

Dr. Bursztyn: Of course, these things are possible, but we were rigidly educated to follow Occam's razor and try to unify the diagnosis; as you will find out, it is not impossible.

Dr. De La Sierra: I do not have an explanation for some of the things that this patient has, but I was wondering why you were insisting on all this previous abdominal history. Considering that you are coming from Israel and the patient was probably Jew, you know one of the possibilities of all these undiagnosed abdominal events that the patient has, and now it seems that he has also a nephrotic syndrome. I would consider the possibility of familial Mediterranean fever with a complication of renal amyloidosis, promoting nephrotic syndrome.

Dr. Bursztyn: This is very important and good thinking and certainly relevant to the area. However, the patient was an Arab and missed two Fs from the familial Mediterranean fever. He had no fever or history of fever and no family history, so this is not supportive. The other thing was that, usually, when you have an amyloidotic nephrotic syndrome, you tend to be hypotensive and not hypertensive. I think severe hypertension in this context had been rarely described.

Hospital Admission

Following assessment, the physicians in the emergency department thought this patient deserves an admission. In the hospital, it was possible to see the CT results from a few years earlier with his abdominal pain, and on this one, both kidneys were of normal appearance and with normal perfusion.

The blood pressure, at the time of his DVT in the emergency room, was normal. In the hospital, again, he had a normal urine sediment, and the ACR was 11.1 g/L. For those who are not used to these units, it is very high.

Due to the history of thrombotic events and the evidence of vascular comprise of the left kidney, we performed a coagulation screen, which was normal, with the exception of prolonged partial thrombotic time (PTT) (Table 2). Prolonged PTT may suggest presence of the lupus anticoagulant, which was indeed found. Antinuclear and anti-neutrophil cytoplasmic antibodies were negative, so this was not supportive of lupus nephritis, and periarteritis nodosa is not usually associated with nephrotic syndrome.

As antiphospholipid antibodies were elevated, confirming the presence of the antiphospholipid syndrome, one might suspect that his past abdominal pain and evidence of colitis could have been related to thrombotic episodes.

His supine plasma renin activity (PRA) with the atenolol that he was supposedly taking was extremely elevated, and the aldosterone levels were also elevated. We can say that he had secondary hyperaldosteronism, which certainly explains the hypokalemia. CT confirmed that the

left kidney was small and had little nephrographic effect with a large, normally perfused right kidney (Figure 1). It seems that this patient has renovascular hypertension related to what appears to be occlusion of the left renal artery. You can see, I guess, the stump of the left renal artery next to the aorta (Figure 2). You can see, again, that the left kidney is small and has poor nephrographic filling. A dimercaptosuccinic acid (DMSA) scan revealed that the left kidney contributed less than 1% to the total renal function with a normally functioning right kidney.

Treatment with ramipril and spironolactone was started. His blood pressure decreased, and his headaches and dizziness dissipated. He was still hypereninemic, potassium level normalized, and creatinine level increased to 124 µL but decreased again to baseline, which is usual for such an immediate and rapid blood pressure reduction. Albumin:creatinine ratio decreased dramatically from 11 to 3. He was given warfarin with the diagnosis of antiphospholipid syndrome with both venous, at least one venous and one arterial, thrombosis, so he is doomed for lifelong anticoagulation.

He was discharged and followed up in the hypertension clinic; blood pressure was very high, and ACR was not performed though requested. INR was normal. He insisted on taking his medications. Since we have the habit of trusting our patients on one hand and suspecting them at the same time, he was invited to return. Again, he had the same findings. Fortunately, he was not taking his warfarin, which might have protected him from cerebral hemorrhage with this horrendous blood pressure level.

Feeling helpless, we readmitted him. On the second admission, his blood pressure was 230/140 mmHg, and he was, again, hyponatremic (130 meq/L) and hypokalemic (3.3 meq/L). After ramipril was resumed, everything normalized: blood pressure was 128/86 mmHg, and sodium and potassium levels were 136 and 4.6 meq/L, respectively, including a marked reduction of the ACR to 0.83 g/g creatinine. Albumin level rose from 25 g/L to 45 g/L. If someone had doubts, it is the urine into where his albumin was leaking. INR was 3.4 as appropriate for a patient with antiphospholipid syndrome.

Discussion of the next steps

> **Dr. Laffer:** His kidney was already shrunken at the time of the first admission, meaning that that was probably not an acute event.
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> **Dr. Bursztyn:** The history of hypertension was there for six months. It might have been longer; I cannot tell.
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> **Dr. Carey:** This is a great case. We used to see this kind of thing more frequently a couple of decades ago when we were doing renovascular bypass grafts for unilateral renal artery stenosis. The patient has a small, shrunken, nonfunctional kidney that is putting out a huge amount of renin. Hyperreninemia causes a huge problem: both blood pressure elevation and electrolyte abnormalities that puts the patient at a higher risk.
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> We treated many such patients, after demonstration of noncompliance with antihypertensive drug therapy, with unilateral nephrectomy. Since the kidney was nonfunctional, we really did not think that we were losing very much. Unilateral

nephrectomy was highly successful in a number of cases. There is still a problem, however. That procedure does not take away the antiphospholipid syndrome and the need for prophylactic anticoagulation. I think that to manage this kind of recurrent hospitalization, I would recommend unilateral nephrectomy.

Case Resolution

He was transferred to the surgical department at the time of admission. His blood pressure was 145/80 mmHg. Due to the demonstrated noncompliance and lifelong need for anticoagulation, had we entertained the thought of revascularization for the kidney, he would have to take warfarin. If the renal artery would have been stented, additional double antiplatelet therapy would have been needed. That is quite tricky. Additionally, the contribution of the left kidney to the renal function was minimal; therefore, the decision was to perform unilateral nephrectomy, as Dr. Carey had foreseen. Interestingly, it was difficult to convince the surgeons. I have never run into such a case where I had difficulty convincing a surgeon to perform surgery, but that probably deserves a different discussion.

A successful laparoscopic nephrectomy was performed, and blood pressure was 126/86 mmHg off antihypertensive therapy. Electrolyte and albumin levels remained normal, and ACR decreased even further (0.45 g/g creatinine).

The pathology showed that medial hyperplastic changes could be seen in the intrarenal arteries; the glomeruli were reasonably preserved with some Bowman space enlargement. Substantial interstitial inflammation was seen, almost with lymphatic follicle formation in the lower part of panel A in Figure 3. Silver staining showed minimal periglomerular fibrosis but no capillary thickening (panel B Figure 3).

The pathology report revealed that the kidney was small (79 g, 9×4×3 cm), as we have known already. There was unremarkable vasculature and stump of the ureter. The capsule was easily removed, ruling out a variety of inflammatory diseases. Parenchymal slices revealed well demarcated and separable cortex and medulla. On microscopy, there was interstitial fibrosis and remarkable tubular atrophy. The glomeruli were not sclerotic. There was some thickening of capillary walls and mesangial abundance. There were no clots within renal blood vessels, and the pathologist concluded it is consistent with chronic ischemia, which is what we had clinically found.

The patient did not show up in the hypertension clinic. He claimed that because of his normal blood pressure, he could not get a referral.

The hypokalemia is readily explained by hyperaldosteronism. However, the hyponatremia is not. The hyponatremia was a recurrent finding. In medical textbooks, it is not a feature of renovascular hypertension. However, high angiotensin II levels generated presumably by the very high PRA promote tubular sodium reabsorption but, on the other hand, are dipsogenic and enhance water intake that probably have resulted in washing away the concentration ability of the kidneys. There is such a possibility that the urine osmolarity did not support an excess of antidiuretic hormone because he had diluted his urine very well. Possibly, the marked pressure natriuresis may also contribute to the final hyponatremic state.

However, findings like ours have been described in several case reports, most of them pediatric cases (1-5). Hyponatremic hypertensive syndrome had been suggested thereof. An adult case series published in *Hypertension* almost two decades ago (6) evaluated 39 such patients as shown in Figure 4: PRA and plasma aldosterone are on a logarithmic, not a linear, scale; in the shaded area were the people who had normal sodium levels and in the open area were those who had hyponatremia, and they all had very high PRA. Plasma aldosterone was also high, but if we recollect that in primary hyperaldosteronism, if there is a tendency for hypernatremia, it must have been a secondary effect.

What does nephrotic syndrome have to do with renovascular hypertension? Proteinuria may happen when you have prolonged hypertension but is usually mild, certainly not in the nephrotic range and certainly not massive. Usually, when there is a heavy proteinuria, it originates from the non-ischemic kidney, maybe angiotensin II dependent, and is ameliorated by either revascularization or ACE inhibition. However, lowering blood pressure, per se, can also do it.

Dr. Halimi and colleagues (7) in France have observed such patients with renovascular disease and nephrotic syndrome. They have noted numerous previous case reports with these findings (7). They noted that most had thrombosed renal arteries. They had compared the patients with renovascular hypertension and nephrotic range proteinuria with those with nephrotic syndrome due to glomerular diseases. What they found is that after a captopril challenge, those with renovascular disease have a decrease in the GFR and renal plasma flow and a marked increase in PRA. It is not only renovascular hypertension but rather a more extreme variety of this entity. Others, also from France (8), perhaps prompted by the previous publication looked into their own database for the association of renovascular hypertension and proteinuria. What they found was that not only renal artery occlusion but also a big difference in size between both kidneys predicted massive proteinuria, in other words, an indication that the contralateral kidney was relatively healthy, allowing for massive proteinuria induced by the excessive PRA generated by the ipsilateral kidney. When the contralateral kidney is not “appropriately” hypertrophied, presumably reflecting also a reduced function, its ability to excrete protein may also be reduced.

Discussion: Case Resolution

Dr. Gaur: There have been case series published with antiphospholipid antibody syndrome associated with membranous nephropathy, so that should be kept in mind in the future if a patient presents with proteinuria of nephrotic range and somebody who has antiphospholipid with antibody syndrome.

Dr. Bursztyn: Absolutely. There are reviews which note a wide range of renal manifestations of antiphospholipid syndrome including both renal artery stenosis and thrombosis (9-11). Some of those could have been relevant, but I think there is no question what was the pathophysiology in our case.

Prof. Dominiczak: My comment is that, of course, renal artery stenosis is a progressive disease. You did nephrectomy, but we do not know what is going to happen with the other kidney. I have seen a similar case where everybody was very happy and everything came

through, but then things started to go wrong in the other kidney. Then, of course, the only treatment available is renal replacement therapy, and I think that is much more dramatic.

I spotted that he was 37 when he initially presented. There are many, many years for this other kidney to also become affected. I think that a patient like this, for me, needs to be observed and followed up in a specialist clinic because things might go wrong.

Dr. Bursztyn: Yes, but it did not work out. He did not show up in the clinic. As a matter of fact, at a ten-year follow-up through a phone call, he reported everything was fine, but who knows, especially with a noncompliant subject.

I would like to raise a question: given the rather benign appearance of the nephrectomized kidney, maybe with hindsight, would it have been better to try to revascularize or reimplant it?

Prof. Dominiczak: We will never know.

Dr. Carey: I do want to go back to the very beginning of this case. This patient presented with a history of left flank pain. That should have been a tip-off that something was going on in the left kidney.

Dr. Bursztyn: That is why renal vein thrombosis was entertained.

Dr. Carey: I think what was happening was a slowly progressive shutoff of intrarenal arculate and interlobular artery flow because of the thrombotic process that had been going on for a long time, I think, probably six months, you are left with a very small kidney that is nonfunctioning. The highest renin secretion comes from that kind of kidney. A small kidney, but it still must have some perfusion. A lot of times in a patient with renovascular disease from a main renal artery, that can occur by collateral vessels that come into play. You may have, actually, a total shutoff of flow by the main renal artery, but collateral circulation is still present, so you get these sky-high renin and aldosterone levels.

Dr. Bursztyn: No question about it.

Prof. Touyz: Do we know anything about his family history?

Dr. Bursztyn: Yes, there is no relevant family history. Like with any type of history, it depends on the historian.

Summary
A case of a 37-year-old man with severe hypertension and nephrotic syndrome is presented. He had a past history of hospitalization for abdominal pain, during which two symmetric kidneys with normal perfusion were revealed by an abdominal CT. He also had a calf DVT in the past, at which time he had a prolonged PTT which was overlooked. Kidney ultrasound with Doppler study showed a small hyperechogenic left kidney with an increased resistive index, suggesting unilateral renovascular hypertension confirmed by CT angiography, which showed occlusion of the left renal artery. The left kidney was found not contributing to the overall renal function (about 1%). Based

on his history of potential vascular abdominal events, documented DVT in the past, and renal artery occlusion, prolonged PTT, and presence of antiphospholipid antibodies and lupus anticoagulant, he was diagnosed as having antiphospholipid syndrome. Because of poor compliance with medical treatment, need of lifelong anticoagulation, and minor contribution of the left kidney to renal function, nephrectomy was performed. Both hypertension and proteinuria were resolved.

This case reaffirms the role of renovascular hypertension in massive proteinuria as previously described (2,3,5,12-15), as well as in many other case reports, and the resolution of nephrotic syndrome and the renin excess by medical interventions or by revascularization with surgery or angioplasty, and nephrectomy as in our case.

It also reaffirms the not-so-well-recognized hyponatremic hypertensive syndrome (1-6) and its similar resolution.

Our case also reaffirms the role of the antiphospholipid syndrome in renovascular hypertension as has been previously described (9-11,15,16).

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Figures Legends

Figure 1. Computed tomography with contrast medium, showing the small left kidney with poor nephrographic filling

Figure 2. Computed tomographic angiography of the kidneys showing normal right renal artery and kidney and a short stump on the left renal artery (white arrow)

Figure 3. Histology of needle kidney biopsy: panel A, hematoxylin and eosin stain, showing relatively normal glomeruli with marked tubular atrophy and intense interstitial inflammation; panel B, silver stain, showing minimal periglomerular fibrosis and some interstitial fibrosis but no capillary thickening

Figure 4. Relation between plasma sodium concentration and PRA (n=20) and plasma aldosterone (n=16) in patients with hyponatremic hypertensive syndrome. PRA and aldosterone are plotted on a logarithmic scale. Normal ranges for PRA and plasma aldosterone level are indicated by shaded areas (6). (reprinted from *Hypertension*)

Table 1. Emergency room laboratory findings

Parameter	Result
Plasma sodium (meq/L)	120
Plasma potassium (meq/L)	2.0
Serum creatinine (µmol/L)	89
Plasma glucose (mmol/L)	4.0
Serum albumin (g/l)	25
Total cholesterol (mmol/L)	7.1
Triglycerides (mmol/L)	3.7
Plasma osmolality (mosm/kgH_2O)	261
Urine osmolality (mosm/kgH_2O)	166
Urine sodium (meq/L)	14.0
Urine potassium (meq/L)	7.3

Table 2. Coagulation factors during admission

Parameter	Result
Prothrombin time (%)	64
Partial thromboplastin time (s)	87
International normalized ratio	1.1
Lupus anticoagulant	+++
Protein S (% activity)	94
Protein C (% activity)	153
Antithrombin III (% activity)	108
Homocysteine (µmol/L)	12.1
Anti-cardiolipin IgG (GPL units/mL); normal<18	135
Anti-cardiolipin IgM (MPL units/mL); normal<10	13.3
Factor V Leiden	heterozygote

Table 3. Serologic markers during hospitalization

Parameter	Result
Antinuclear antibodies	negative
Anti-neutrophil cytoplasmic antibodies	negative
Anti-hepatitis C virus antibodies	negative
Hepatitis B surface antigen	negative