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# Reversing Chronic Kidney Disease with Niacin and Sodium Bicarbonate

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## **Review and Commentary by Stephen McConnell and W. Todd Penberthy**

OMNS (Oct. 14, 2021) This story began with initial discovery, motivated by necessity. It would lead a few years later to reproducible documented reversal of chronic kidney disease (CKD) stages 1 or 2. Success was achieved using 3 to 5 cents/day of 100-500mg niacin TID [three times a day] along with 1.0-1.8 grams of sodium bicarbonate (baking soda, 600mg at lunch and 1.2g before bed) with or without < 2g/day elemental calcium, as calcium carbonate.

Excellent results from the use of niacin to treat CKD have now been documented for more than 25 case-studies. This approach is well supported by continuous basic and clinical research, including dozens of clinical trials that provide substantial evidence for the use of niacin and sodium bicarbonate. These approaches directly address the needs of the typical CKD patient. Unfortunately, this approach is rarely implemented in the clinical setting.

CKD commonly progresses with age as it is observed in 68% of Americans  $\geq 60$ y of age. [1] Patients with CKD usually experience progressive loss of kidney function moving towards an increasing risk of end-stage renal disease (ESRD). CKD is the 9th leading cause of death in the US. [2] Fortunately, there are several simple approaches including the addition of modest doses of niacin (immediate release- or IR-Niacin) that can reverse CKD in many patients as described here.

Approximately 786,000 people per year, in the US progress to ESRD (stage 5 CKD), which is generally considered an irreversible condition. Most of these become completely dependent on regular trips to dialysis. Estimation of the stages of CKD is based on (GFR) glomerular filtration rates starting with  $\leq 60$ mL/1.7m<sup>2</sup> for 3mos as definitive of initial CKD diagnosis. Unfortunately, a creatinine derived GFR (crGFR) is only as reliable as the serum

creatinine measure. Use of this creatinine-based test has a "blind-area" in the earlier stages, and frequently leads to an under-estimation of the true risk.

## Stages of CKD

1. Mild kidney damage, eGFR 90 or higher
2. Mild loss of kidney function, eGFR 60-89
3. Moderate loss of kidney function
  - a. eGFR 45-59
  - b. eGFR 30-44
4. Severe loss of kidney function, eGFR 15-29
5. Kidney failure or close to failure, eGFR less than 15

## Niacin for CKD

Supplementation with daily low-dose niacin reliably reverses a large amount of the functional loss. This simple treatment is effective and critically important. Mortality rates with CKD are striking, as the 5-year survival rate for patients doing long-term dialysis is 35% compared to 25% in those with diabetes [T2DM] in the USA. [3]

Routinely, the first treatment approaches utilized for CKD patients, in the later stages, generally targets control of dysglycemia & reduction of hyperphosphatemia according to KDIGO guidelines. [4] Fortunately, there is an ever-increasing abundance of data revealing that simple niacin treatment is a profoundly effective treatment for reducing hyperphosphatemia -- and that is just the beginning. In basic research the evidence in favor of niacin for CKD has continuously accumulated. Clinical research proves that the niacin stimulated pathways involving increased NAD synthesis, PCSK9 inhibition, sodium transporter effects, PPAR gamma activation, and more, are exceptionally well-suited to addressing CKD, multimorbidity, and ultimately all-cause mortality. [5-41]

The clinical and financial impact of CKD when it progresses to end-stage renal disease (dialysis-dependence; ESRD) is profound. Clinically, CKD progression quickly leads to lifelong dialysis with co-morbid life-threatening cardiovascular disease. Financially, The out-of-pocket cost of CKD is greater than cancer and stroke with ESRD dialysis costing 30.9 billion per year 2013 or approximately 7.1% of total Medicare costs. [42] Medicare spends approximately \$250,000/y for every CKD patient, *prior to* the transition to ESRD and dialysis. Annual costs per dialysis patient can range from \$720k to \$2.2m per year. [43] These problems and their associated costs can be reduced by using 5 cents per day of niacin.

Originally, I (SM) was formally trained to operate a heart-lung machine, maintain full life-support & anesthesia, in the operating room monitoring patients undergoing open-heart

surgery. Much later, I transitioned to working as a field scientist, MSL (Medical Science Liaison) in the advanced laboratory diagnostics industry. My primary clinical focus since that time has mainly been lipidology. Because of my initial education/training, addressing cardiovascular disease, I now focus on prevention: lipidology. This training gave me an appreciation for nicotinic acid (niacin, vitamin B3).

I have now personally observed more than 25 documented cases of individuals having their CKD progression not only halted but reversed with the addition of 3 to 5 cents worth of niacin, per day (with 1.8-2.4g/day sodium-bicarbonate with/without 250-500mg/day calcium-carbonate).

## **A family story**

While I (SM) was learning lipidology in the period between 2002-2007, my father suddenly went to the ER late one Sunday night and my mother called me hysterically, "I took your father to the ER and now they are scheduling him for placement of stents." I was concerned, as any son would be, but also as a scientist because I felt I may have 'failed him,' somehow: If only what I had already learned, I had only learned it, sooner.

My father was 81 at the time and he had been jumping rope for 30-minutes, twice daily. His body had a deceptively healthy look, and his triglycerides were low, but when we put it all together, he was "Pattern-B" -- insulin resistant. He had always been a 'stodgy,' stubborn, stoic World War II veteran. He was very introverted and typically had a limited range of emotions: rage, laughter, and silence. Later, I would find out he had Asperger's.

When I received the advanced laboratory data, it showed that he had low HDL2 and high ApoB. This is far more specific & confers much greater risk vs. an elevated LDL-C. Most importantly, this revealed he was insulin resistant a.k.a. pre-diabetic. At the time, I really didn't fully understand this. Even today, most clinicians really do not, due to continued reliance on using only tests for FBG & HbA1c. Ultimately, my father survived that, and we continued to institute aggressive medical management: A hard lesson learned.

My father and my mother traveled everywhere together. They commuted, seasonally to Florida each winter, to escape the cold weather in Northwest Pennsylvania. On New Year's Day, about 6 months after his MI & stent-placement procedure, I received a call from my mother, "Your father is in the hospital! They're going to have to do open heart surgery!"

They needed to do an aortic-annuloplasty (aortic heart valve repair), in addition to a quintuple CABG (5 bypass grafts). I thought to myself, "this is getting worse and worse." Having had previous personal experience working with thoracic surgeons during open-heart operations, I didn't want the procedure to begin until my brother and I were able to be present. Fortunately, the young thoracic surgeon and the techniques planned were excellent.

Later, in the spring, they returned home to Erie, Pennsylvania for the follow-up visit. Dr. Dave (the physician who asked me to set-up my 1st lipid clinic) said, "Hey I got some bad news for you. Your dad has renal insufficiency." I said, "Oh my God, he's in renal failure, what stage is he?" He did not know. That was a flag. Most clinicians don't know what stage their CKD patients are because the lab doesn't do calculations and the creatinine measure is not reliable or accurate. The creatinine measure has very little accuracy until after the CKD has 'hit' stage 3B, and beyond. [44,45] So, a lot of these patients along the CKD disease continuum, through each progressive stage, appearing to have less risk vs. the 'true' risk that is present. It's better to test a urine sample and see how much protein is recovered, and run a Cystatin-C and a crGFR to calculate a more accurate value. At that time, I only knew he was in failure, but when I did the crGFR calculation, I could see that he was well into the latter portion, of CKD-stage 4.

Recently, I had been putting together a new treatment algorithm with substantial literature support, data, on CKD. I was lucky to have been mentored by Dr. William F. Finn. [46]. Even if a patient has not already been scheduled for dialysis, he explained, and especially if they are currently on dialysis, you must **get the serum phosphorus down**. Excessive phosphorous is toxic to the kidneys as well as virtually every organ system and the entire body. [47,48] Phosphorus is a primary initiator of vascular calcification, among several other pathologies. If the kidneys start to lose a certain fraction of their normal function, the body can no longer efficiently clear phosphorous. When phosphorous serum levels reach abnormal levels, then you begin to saturate the tissues. Then phosphorous binds to calcium and it's the phosphorous, not the calcium that starts the pathology leading to calcium phosphate stones.

### **Niacin helps to get the phosphorous down.**

Even after you bring serum phosphate down you still have it in the tissues. The only biomarker available in a clinical setting, Fibroblast Growth Factor-23 (FGF-23), reflects the pathology behind long-term exposure to elevated phosphorus. FGF-23 can be decreased, simply by administering niacin. [14] However, the sodium phosphorous transporter works through a feedback mechanism to make more receptors to compensate.

So, calcium carbonate (from an antacid tablet) is commonly used first to bind the readily available intestinal phosphorous. This is among the cheapest and most effective phosphorus chelator approaches. Calcium carbonate should not be used above 2g/day elemental calcium, which is 40% of most of the formulations: Total 5g/day as calcium-carbonate. This should be administered at mealtime. The idea is to 'treat the meal', as there is generally very little phosphorous available to bind, outside of mealtime. When the kidney is in 'failure', after meals, excess phosphorous remains uncleared and leads to deposition in the tissues: valve leaflets; at the endothelial barrier; arterial subendothelial space (Mönckeberg's medial calcification: arteriosclerosis). [49] When sodium bicarbonate (baking

soda) is administered, based on the landmark study, [50,51] the transition from stages-3 & 4 to Stage-5/ESRD/Dialysis, can be reduced by ~80%, with just 1.8 grams sodium bicarbonate, alone. Mealtime dosing BID, (1X 600mg at lunch & 2X 600mg at dinner each day, i.e 1.8g total per day), optimizes the therapy.

In that study, the fraction of people of people that went to dialysis by the end of two years was roughly 35% on placebo, but the fraction that went to dialysis with the modest dose of sodium bicarbonate, was reduced roughly > 80%. [50] However, the concerns about sodium intake are frequently expressed. The literature is quite clear on this. The chloride salt of sodium is the issue, not the bicarbonate salt of sodium. This a key point. We just need to do a better job of identifying them early on. Do not assume the patient is stage 1 or 2 if the creatinine indicated that. We need better, more reliable biomarkers (EXAMP: Cystatin-C) and should insist the insurance companies reimburse for it.

This approach worked amazingly well for my father, because he reversed his CKD, by more than two stages! I calculated it incrementally based on where he was at each stage. He was nearing end-stage renal disease (stage 5) and he reverted back to stage 2, which was a virtual miracle at that time! I had never heard or seen of anything similar.

Niacin interested me when I came across a company that was working on a new chelator for phosphorus. I had already seen some literature on an extended-release niacin (ER-niacin) study showing a phosphorus-lowering effect and IR-Niacin having an antiproteinuric effect. Niacin was so effective that it moved the GFR up enough to reverse the baseline status by a full stage, even at very low doses. This seemed to be the plausible explanation for this net result.

Niacin (as well as no-flush niacinamide/nicotinamide) inhibits the sodium phosphate transporter. There are at least twenty peer-reviewed publications demonstrating this. [5-41,52-59] What was discovered was, if you want to control phosphorus, niacin is one the most effective methods and its efficacy is not affected by timing relative to meals. As little as 100mg of niacin will effectively reduce the serum phosphorus.

Some studies refer to this niacin-mediated effect as the "phosphorous fix". The additional CKD benefits of niacin include the antiproteinuric, as well. If you compare a blood test vs. urine test, then the urine is probably a much more reliable indicator, because when the basement membrane is damaged, filtration is impaired such that the basement membrane between the podocyte processes no longer conserves plasma proteins & the amount lost, 'leaked' is present in the urine. The appearance of albumin (protein) in the urine is a 'flag' that loss of serum protein due to impaired renal function. Often, this is one of the earliest markers. Blood biomarkers have some variables that could result in misclassification of CKD stages. Protein leaking from the kidneys, is a direct correlate to the podocyte/basement-membrane damage. This is the gold-standard measure of endothelial

function. I always like to use at least one blood marker (ideally CystatinC) in addition to the urine test, to facilitate extrapolating, "pinpointing" the true stage at baseline and where they are at follow-up.

I believe niacin is probably one of the best treatment options for a variety of chronic conditions/pathologies. CKD is a complex disease state. At its 'core', it is a vascular disease, but *if* you "hit all the right buttons" it is clearly possible to 'drive' CKD backwards.

With stage-5-CKD a.k.a. end-stage renal disease (ESRD), the scarcity of donor organs is a primary challenge. The reality is usually that dialysis will be required for the rest of the patient's life. That is a powerful motivator to the patient to consider niacin.

Ultimately my father's CKD, reversed from stage 4 to stage 2. When the sum of all the data, connecting-the-dots with all the biomarkers, he was close to end stage renal disease as he was scheduled to have a 1st encounter with a nephrologist. So, he was likely headed to dialysis, sooner vs. later.

## **The Current State of CKD Treatment & the Importance of Addressing Multimorbidity**

In regard to prevention, many physicians choose not to believe there is any way to prevent or reverse CKD. Unfortunately, most patients end up on dialysis, or at the very least their CKD continues to get worse.

Too often, a less than adequate job of correctly identifying pre-diabetes is implemented, early on in the CKD disease state. It is vitally important to have a method of measuring the glucose post-prandial (PPG) level at 1h and 2h post-glucose challenge (OGTT). Currently, this is the gold standard test for assessing pre-diabetes. There are blood biomarkers that have a VERY high level of precision determining the 1-hr PPG: 1,5-AG & AHB (*Alpha-HydroxyButyrate*).

Measures of fasting insulin, fasting glucose, and HbA1c can miss an unacceptably large number of pre-diabetics. The OGTT test, will reliably capture a pre-diabetes diagnosis. HOMA-IR (HOMA-IR; homeostasis model assessment as an index of insulin resistance) is an effective method to calculate and evaluate insulin resistance using conventional reference lab biomarkers: insulin levels, fasting glucose levels, and A1C [60,61]. If you have these 3, you can then calculate the HOMA-IR. This enables accurate documentation and validates spending the modest expense to do the proper tests.

As much as 70% of adults over the age of 30 do not have normal postprandial glucose (PPG). It's that bad! They say it's only 30% or 40%, but that's likely based on poor statistics. In fact, during every year in the last several decades, the percentage of individuals over the age of 30 with obesity has risen. The antiquated Frederickson classification was based on

cholesterol/triglyceride parameters, but we are presently in the 'particle age' of clinical lipidemia assessment. Like the Frederickson classification for lipid disorder sub-types (which was largely based on cholesterol measurements), current methods to assess the presence & severity of insulin-resistance a.k.a. pre-diabetes, are essentially obsolete.

Another aspect to consider is multiple comorbidities. Modern medicine currently generally takes the approach of treating one condition at a time, but there are nearly always multiple disease symptoms present that are tightly associated and anything that can ultimately address this is going to result in the most effective therapies, ideally prior to the fulminant disease.

The Academy of Medical Sciences declared in 2018 that multimorbidity is the number one top priority in healthcare research. [62] Estimates for a cure of cancer reveal that this would only increase lifespan by a mere 3 years on average because the associated co-morbidities were not addressed. [63] Niacin, however, addresses so many common denominators for disparate diseases that the impact of niacin treatment for CKD/ESRD is likely to benefit many more indications, especially the number one killer, cardiovascular disease.

At the end of the day, it is the effect on all-cause mortality that matters the most for any treatment. After the termination of the Coronary Drug Project-CDP trial, it was determined that all-cause mortality was reduced by 11%, 9 years after stopping niacin treatment (avg. dose 2.4 g/day). [64] This may be a feat unparalleled in proven clinical medicine. By contrast, statin all-cause mortality data has yielded mixed results.

## Conclusion

In over 25 documented individual cases of CKD stages 2 through 4, after initiation of a combination-therapy of supplements based on GFR, including 500mg TID IR-Niacin, over a 3-month period, it was possible to improve their disease by at least one stage.

In basic and clinical research the evidence in favor of niacin for CKD is strong. Clinical research proves that the niacin is exceptionally well-suited to treatment and prevention of CKD, multimorbidity, and ultimately all-cause mortality.

Sampathkumar explained the current CKD treatment with niacin situation best, *"Pharmaceutical industry driven large-scale studies are unlikely to be undertaken given the low-cost of niacin. David is up against the formidable Goliath of players promoting costly non-calcium containing phosphorus binders. It is time that international bodies like Kidney Disease, Improving Global Outcomes (KDIGO) take a call on usefulness of niacin as a low-cost, effective, and low pill burden agent for phosphorus reduction in CKD with multiple pleotropic benefits."* [29]

## Recommended Doses to address Chronic Kidney Disease

Low-dose immediate release-niacin, 100mg - 500 mg, 1 to 3x/day. No-flush niacin or niacinamide will have equal efficacy on lowering phosphorus levels, but negligible cardiovascular benefits compared with standard niacin.

Sodium Bicarbonate (baking soda) 1.8g/d (1/3 at lunch and 2/3 at dinner).

Calcium carbonate antacid pills (400-1000mg elemental calcium or 2-4gms antacid tablets) with food to bind phosphorous in food.

Low-Dose-Thyroid Supplementation| (25-50µg T4/Levothyroxine or 1/2grain of Desiccated Thyroid).

Methyl Folate (0.8g to 2mg L-MethylFolate).

## Recommended Additional Monitoring

A full panel of metabolic parameters [baseline & 90-day f/u] can also determine 'collateral' benefit[s], especially related to cardiovascular health:

Apo-B decreases

Apo-A1 increases (INTERHEART Study)

Lp(a) mass decreases

Lp-PLA2 decreases

MPO/myeloperoxidase| decreases

AST/ALT/GGT hepatic parameters improved

Symptomology/Signs-Symptoms: TIA; Chronic Angina; Claudication; Dyspnea upon Exertion.

***The views of the authors, who are not physicians, are presented here for educational purposes. All readers are reminded to be sure to work with their own health care provider(s) before commencing this or any nutrition-based approach.***

*(Stephen D. McConnell is a lipidemiologist and researcher with an MSc in Cardiovascular and Renal Pathophysiology. W. Todd Penberthy, PhD, specializes in writing about targeted pharmacotherapeutics, CME, and biomedical texts.)*

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