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The Importance & Power of Physicians Advocating for Themselves

By David Blitzer, MD, and Tomas Diaz, MD

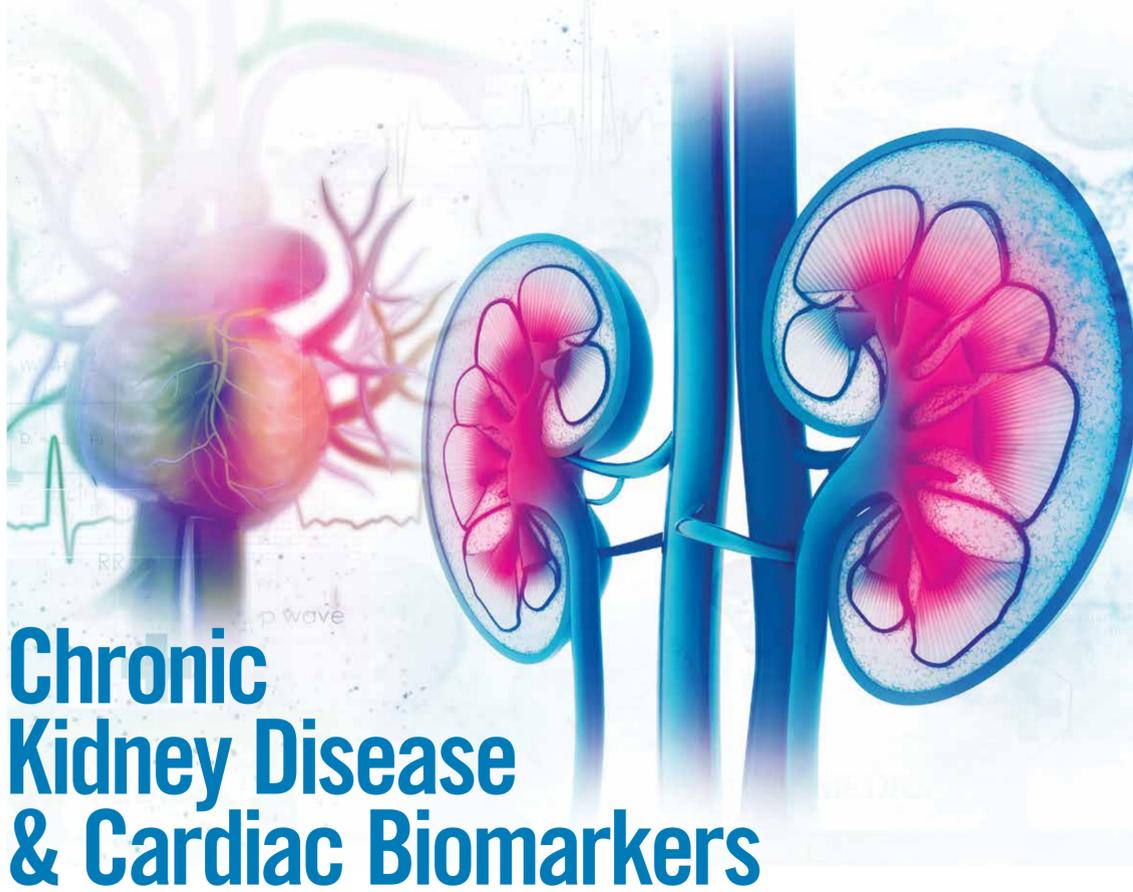
In the past, we as physicians have not done a great job of advocacy, and we have largely been removed from policy discussions. The emergence of physician advocacy is a relatively new phenomenon. During the AIDS crisis, a unified physician voice was largely missing from policy conversations. Since that time, physician advocacy for social change has grown. Physicians have led movements calling for sensible gun control only to be told to “stay in our lane.” Physicians have supported broader access to healthcare, defending the ACA against repeated repeal attempts by a government body with minimal healthcare experience.”

Despite bearing witness to the consequences of policy decisions, our expertise is dismissed, and our calls for action go unnoticed. With COVID-19, we have begun to find our voice but, as in the past, have lacked the power to push forward important structural changes to address current and future healthcare challenges.

If the current pandemic has taught us anything, it is the importance and power of physicians advocating for ourselves. While we are currently advocating for the supplies and support we need, this is also an opportunity—a call to action—to continue to represent our field, our patients, and our communities. While we enjoy the privilege of caring for others on a daily basis, we must not forget that our profession affords us a class privilege, which we should leverage to promote health equity. There is no doubt that there will always be a need for competent and dedicated clinicians to serve on the frontlines. But, this pandemic has shown that we will also always be in need of effective advocates for our patients and our profession.

If there is a silver lining in all of this, it comes from the affirmation that when we unite and advocate for ourselves and our patients, we can do great things. As the curtain of isolation lifts, we will continue to draw upon this newfound strength, and we hope you, dear reader, will join us.

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Chronic Kidney Disease & Cardiac Biomarkers



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Previous studies have established a direct association between chronic kidney disease (CKD) and cardiovascular (CV) mortality and morbidity. Evidence suggests that patients with CKD have an increased risk for myocardial infarction, heart failure, stroke, and mortality. “However, there is a paucity of data that helps identify those patients with kidney disease who are at an increased CV risk,” explains Horng H. Chen, MD.

To better understand the connection between CKD and cardiovascular comorbidities, as well as the impact of high sensitivity troponin (hs-TnT) and brain natriuretic peptide (NT-proBNP) on this association, Dr. Chen and colleagues conducted a retrospective analysis of the Olmsted County Heart Function Study cohort and published their results in *Mayo Clinic Proceedings*. Both hs-TnT and NT-proBNP have been sus-

pected to be telling biomarkers because both are cleared by the kidneys. The study authors assessed blood test data on hs-TnT and NT-proBNP to determine if they could be used to help identify patients with CKD who are at increased risk for adverse CV events.

Patients were classified based on their kidney function, and cardiac biomarker activation trends were characterized. Participants were organized into three groups dependent on estimated glomerular filtration rate (eGFR): normal renal function (eGFR ≥ 90 mL/min/1.73 m²; Group I), mild renal insufficiency (eGFR 60-89 mL/min/1.73 m²; Group II), and CKD (eGFR < 60 mL/min/1.73 m²; Group III). Dr. Chen and colleagues followed participants for a median of 10.2 years (interquartile range, 5.1-11.4 years). Compared with Group I, Group III participants were more likely to be older (75 \pm 9 years vs 58 \pm 9 years) and male (65.0% [82 of 126] vs 48.7% [399 of 819]).

Over 10.2 years follow-up, patients with CKD had an increased risk of heart attack (hazard ratio [HR], 1.95) and a composite of congestive heart

failure, stroke, and all-cause mortality (HR, 1.38) when compared with patients without CKD. “Patients with NT-proBNP or hs-TnT in Group III were at greater risk of CV events without significant interactions between eGFR and levels of NT-proBNP and hs-TnT,” adds Dr. Chen.

The findings suggest that patients with high levels of hs-TnT or NT-proBNP are at a higher risk for adverse CV events (Table) regardless of their renal function. “Patients with CKD are at an increased risk for adverse CV events, including heart attack, heart failure, stroke, and death,” Dr. Chen emphasizes. “Cardiac biomarkers like hs-TnT or NT-proBNP play a prognostic role in identifying individuals at high risk for adverse cardiac outcomes.” Overall, the evidence suggests that patients with CKD had a 38% higher risk of stroke than did patients in Group I (HR, 1.38).

“In this study, we demonstrated that NT-proBNP and hs-TnT have prognostic value regardless of patients’ kidney function,” says Dr. Chen. “Hence, these two biomarkers can be used to help clinicians identify patients with CKD who are at increased risk for adverse CV events.” Upon analyzing the optimal cutpoint for both biomarkers, both were at the third tertile. The optimal cutpoint for was 97.1 pg/mL for NT-proBNP and 3.8 ng/L for hs-TnT (Table).

Further research confirming that NT-proBNP and hs-TnT have prognostic value regardless of kidney function would help clinicians provide more precise care to patients with CKD who might be or are at an increased risk for adverse CV events and are candidates for aggressive CV risk strategies. “The next step in the research will involve clinical trials of patients with CKD who are identified to be at an increased CV risk using the NT-proBNP and hs-TnT biomarkers and randomizing them to aggressive risk modification strategies, to assess for improvement in long-term and short-term outcomes,” notes Dr. Chen. ■

Table Interactions of Biomarkers With Outcomes in CKD

Group	Biomarker		
	NT-proBNP, pg/mL		
	4.5-37.3	37.4-113.6	113.7-7899
I	43%	35%	22%
II	28%	35%	37%
III	7%	15%	78%
	Hs-TnT, ng/L		
	0.1-2.2	2.2-4.2	4.3-523.5
I	44%	31%	25%
II	30%	34%	36%
III	11%	22%	67%

Abbreviations: CKD, chronic kidney disease; Hs-TnT, high-sensitivity troponin T; NT-proBNP, N-terminal pro B-type natriuretic peptide.
Source: Adapted from: Vinnakota S, et al. *Mayo Clin Proc.* 2019;94(11):2189-2198.

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Telemedicine Fatigue & the Stress of Remote Care

The first thing I heard from my team after starting fulltime telehealth was the exhaustion that seemed to set in at the end of the day. I have noticed this myself. After 8 hours of back-to-back virtual engagement with parents, I found myself with a kind of telemedicine fatigue that’s hard to describe.

There are a few potential explanations for this almost consistent report among my colleagues who had transitioned to a full telehealth practice. I’m going to continue to dig deeper into this, but I suspect that it represents a couple of things.

Often, there are inconsistencies in connection, lighting, front-facing lens hygiene, and video quality that require a kind of on-the-fly compensation. And there are the parents who want to hold their phone at arm’s length during a 30-minute consult, creating a simulated earthquake experience.

The most obvious potential contributor to this fatigue is the simple stress of transition. Adjusting to a completely different workflow is impossibly challenging, especially for health professionals who have been conducting analog care for most of their career. And on both ends of the encounter is the new “literacy” of engagement by live video connection. For example, the basic error of watching the screen display rather than the seeing-eye camera leads to a classic disjunctive virtual gaze that is subtly jarring and strangely distracting.

But the source of telemedicine fatigue goes beyond professional adjustment and correction of technical glitches.



I’ve identified that the emotional stress of subtly strained connection is a huge contributor to the exhaustion I feel. A video connection negates the subtleties of connection that are critical to my assessment of a parent and child. Identifying and exploring these subtleties is central to the care of chronically ill children and their families. It feels like I’m working hard to pick up on non-verbal cues that may be difficult to identify or simply out of the frame of view. The simultaneous observation of a mother and child in the same frame presents its own challenges in a home environment. I call this the “drive by” telemedicine assessment, as the child zips in and out of the field of view grabbing toys, running for snacks, etc. ■

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In Case You Missed It

Benzodiazepines, Co-dispensed Opioids Common in Hemodialysis

Patients initiating hemodialysis often are prescribed opioids and short-acting benzodiazepines, which are associated with increased mortality risk, according to a study published in the *Clinical Journal of the American Society of Nephrology*. Researchers examined the mortality risk associated with short- and long-acting benzodiazepines and their interaction with opioids in a cohort of 69,368 adults initiating hemodialysis. Of patients, 16% and 5% were dispensed a short- and long-acting benzodiazepine, respectively, within 1 year of hemodialysis initiation. Co-dispensing of opioids and short- and long-acting benzodiazepines occurred among 26% and 8%, respectively, of those dispensed a benzodiazepine during follow-up. Patients with an opioid prescription were more likely to be subsequently dispensed a short- and long-acting benzodiazepine (adjusted hazard ratios, 1.66 and 1.11, respectively). Compared with those without a short-acting benzodiazepine, those dispensed a short-acting benzodiazepine had a 1.45-fold increased mortality risk; this risk was increased 1.90-fold among those with opioid co-dispensing. There was an inverse association for long-acting benzodiazepines with mortality (adjusted hazard ratio, 0.84); no differential risk was seen by opioid dispensing. “Providers caring for patients undergoing hemodialysis should be given the tools needed to implement a collaborative, team-based approach for de-prescribing of short-acting benzodiazepines, particularly for patients who are likely to use opioids,” the authors write.

Environmental Chemical Exposure Tied to Kidney Disease

Increased exposure to heavy metal lead, cadmium, or volatile organic compounds may be associated with an increased prevalence of chronic kidney disease (CKD), according to a study published in the *Clinical Journal of the American Society of Nephrology*. Study investigators used data obtained from 46,748 adult participants in the U.S. National Health and Nutrition Examination Survey (1999 to 2016) to assess whether exposure to environmental chemicals is associated with CKD. Among 262 environmental chemicals, seven were significantly associated with an increased risk for albuminuria, reduced estimated glomerular filtration rate (eGFR), or the composite outcome of both. These chemicals included metals and other chemicals that have not previously been associated with CKD. Albuminuria was associated with serum and urine cotinine, blood 2,5-dimethylfuran (a volatile organic compound), and blood cadmium. Reduced eGFR was associated with blood lead and cadmium. The composite outcome was associated with blood cadmium and lead and three volatile compounds (blood 2,5-dimethylfuran, blood furan, and urinary phenylglyoxylic acid). “We need to make sure that workers have appropriate protective equipment when exposed to toxic chemicals and that we all have access to clean, safe water,” writes the author of an accompanying editorial. ■

