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STATE-OF-THE-ART REVIEW

Renal Compression in Heart Failure

The Renal Tamponade Hypothesis

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HIGHLIGHTS

- Renal dysfunction in heart failure remains highly prevalent and is associated with worse outcomes.
- The rigidness of the renal capsule is central in congestion-induced damage to the renal structures.
- Renal decapsulation has been shown to be beneficial in animals, making it an interesting, novel treatment to investigate in heart failure.

ABSTRACT

Renal dysfunction is one of the strongest predictors of outcome in heart failure. Several studies have revealed that both reduced perfusion and increased congestion (and central venous pressure) contribute to worsening renal function in heart failure. This paper proposes a novel factor in the link between cardiac and renal dysfunction: "renal tamponade" or compression of renal structures caused by the limited space for expansion. This space can be limited either by the rigid renal capsule that encloses the renal interstitial tissue or by the layer of fat around the kidneys or by the peritoneal space exerting pressure on the retroperitoneal kidneys. Renal decapsulation in animal models of heart failure and acute renal ischemia has been shown effective in alleviating pressure-related injury within the kidney itself, thus supporting this concept and making it a potentially interesting novel treatment in heart failure. (J Am Coll Cardiol HF 2022;10:175–183)

The kidney plays a central role in heart failure (HF), and several large studies have shown that renal dysfunction is one of the most powerful predictors of outcome in HF.[1,2] Already in 1990 it was demonstrated that the kidney is very sensitive to changes in perfusion. When cardiac index decreases by 25%, renal blood flow decreases by as much as 50%.[3] Later, several groups showed that reduced renal perfusion is among the strongest determinants of glomerular filtration rate (GFR) in HF.[4-6] Further studies in this field revealed that not only reduced renal perfusion but also, importantly, increased central venous pressure contribute to a decrease in renal function.[7,8] Ever since, several

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ABBREVIATIONS AND ACRONYMS

GFR = glomerular filtration rate

HF = heart failure

IAP = intra-abdominal pressure

KIM-1 = kidney injury marker 1

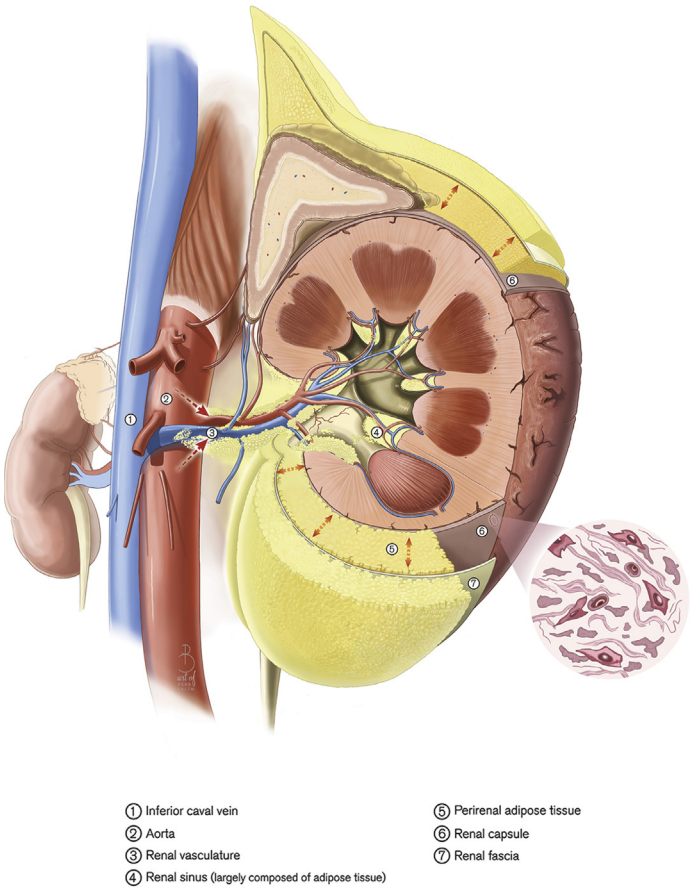
RAAS = renin-angiotensin-aldosterone system

TNF-α = tumor necrosis factor-α

studies have investigated the associations between clinical factors and renal congestion. Yet, the area of cardiorenal interplay remains complex.

In the present paper, we discuss the 3 mechanisms that, individually or combined, may lead to renal congestion as a result of intrarenal or extrarenal compression. Another (classic) example of renal injury as a result of compression can be found in the Page kidney: a clinical syndrome of diminished renal function and/or hypertension as a result of external compression on the kidney, most commonly subcapsular hematoma.[9] The fact that the kidney is surrounded by a rigid and nonexpandable capsule plays a crucial role in the Page kidney and in renal compression in HF.[10,11] We will discuss these mechanisms and will coin a novel term for this: the "renal tamponade" hypothesis.

FIGURE 1 Anatomy of the Kidney and the Perirenal Space



The kidney is surrounded by the renal capsule. Histologically this capsule is made of dense irregular collagen tissue, making it particularly rigid. Around the renal capsule the perirenal space is bordered by the renal fascia and is made up of mainly adipose tissue. The renal sinus (4) is not covered by renal capsule and is therefore sensitive for local fat infiltration and compression of vasculature.

ANATOMY OF THE KIDNEY

The kidneys are located in the retroperitoneal space. The area surrounding the kidney is called the perirenal space (**Figure 1**, number 5). This space is enclosed by the renal fascia (**Figure 1**, number 7). The perirenal space consists mainly of adipose tissue; also, the renal vasculature, lymphatic system, and adrenal gland can be found within the perirenal space. The kidney itself is captured by a thick fibrous capsule that serves as protection to the soft renal tissue (**Figure 1**, number 6). Of note, the capsule does not cover the renal sinus, leaving an opening for the renal artery, renal vein, renal nerve. and renal pelvis to either enter or exit.[12] Within the kidney, the glomeruli are located in the cortex, and the tubules descend into the medulla.

Compression of the kidney and renal vasculature can therefore theoretically arise from increased pressures in 3 different compartments. An overview of the current data on increased pressure in the different compartments can be found in **Table 1**.

INCREASED INTRACAPSULAR PRESSURE

Increased pressure within the renal parenchyma can result from increased volume in the kidney caused by increased interstitial fluid in HF, in the context of an organ (the kidney) that cannot expand in volume (**Central Illustration**).

Histologically, the fibrous renal capsule consists of many collagen fibers in a dense, irregular structure, making it decidedly rigid.[10] Pressures up to 10,000 mm Hg are required to stretch the capsule to twice its size or even rupture it (**Figure 1**, no 6).[13] Pressures of such a magnitude are generally reached only during traumatic events or polycystic kidney disease.[14,15] In other instances, such as congestion from HF, pressures are likely deflected inward by the rigid capsule. To illustrate, in HF (with both reduced and preserved ejection fraction), as a result of maladaptive water and sodium homeostasis, intravascular pressures rise. As intravascular pressures reach a tipping point, fluid will exit the bloodstream into the interstitium.[16] In the skin, this gives pitting edema; in the lungs, it leads to alveolar edema. Interstitial edema is similarly present in the kidneys, albeit less visible. The kidneys, however, do not have the ability to expand as do the skin and subcutaneous tissue.[11] The reason

for this lack of expandability is the presence of the very rigid renal capsule (**Figure 1**, number 6).[13]

Several kidney congestion models in rats and dogs have demonstrated that when central or renal vascular pressures are increased, usually from clipping of the respective vein, renal interstitial pressures rise collinearly.[17,18] Moreover, GFR and urinary production almost instantly decrease. In addition, clipping of the renal vein induces proteinuria, reflecting (pressure-induced) damage the Bowman capsule.

Two studies independently examined renal perfusion in a congested kidney model and found diminished perfusion of the renal medulla but not the renal cortex.[18,19] Anatomically, this means the tubules are more at risk for damage from congestion than are the glomeruli. This is further supported by the notion that intrarenal expression of biomarkers of tubular damage, in particular KIM-1 and osteopontin, were increased in a murine model of renal congestion.[20] Even more interestingly, the expression of these biomarkers was attenuated by removing the capsule before inducing congestion.[20]

In addition to tubules and glomeruli, veins are also affected by intracapsular pressure overload, as has been demonstrated in several small ultrasound studies in humans.[21-23] In the healthy kidney, venous blood flow is minimally altered by hemodynamic changes. However, increases in pressure within the renal capsule will lead to a collapsing of renal veins because the capsule prohibits the kidney from expanding, and pressures are reflected inward. On ultrasonography, a discontinuous venous flow pattern can be recognized; this pattern is correlated with clinical signs and symptoms of congestion. This indicates that in the congested kidney, blood is solely being pulled through a compressed vein during diastole.

In summary, interstitial congestion of the kidney, combined with the inability for the interstitium to expand because of the renal capsule, compresses intrarenal structures such as veins, glomeruli, and tubules, diminishing their function.

INCREASED PERIRENAL PRESSURE

Increased volume of adipose tissue within the perirenal fascia may lead to increased perirenal pressure (**Central Illustration**). Both the thickness of the perirenal adipose tissue surrounding the kidney and accumulation of fat in the renal sinus have been associated with chronic kidney disease, arteriosclerosis, hypertension, and the onset of diabetes.[24-27] This association can be explained by perirenal adipose tissue compressing the renal vasculature, leading to pathologic activation of the renin-angiotensin-aldosterone system (RAAS) and reduced renal perfusion (**Central Illustration**), as well as venous compression, (further) congesting the renal interstitium.[28,29] Alternatively, perirenal adipose tissue can cause RAAS activation through its inflammatory properties and increased local levels of TNF-α.[29,30] Renal sinus fat is of interest in relation to the renal capsule, inasmuch as the renal sinus is not protected from outside compression by the renal capsule (**Figure 1**, number 4). This means that increases in renal sinus fat volume directly increase pressures within the renal capsule.

Indeed, renal sinus volume has been correlated to both GFR and intrarenal perfusion in patients with type 2 diabetes.[31] Although this is not an acute mechanism, altered intrarenal hemodynamics resulting from perirenal fat may contribute to a decrease of renal function in the setting of interstitial congestion. Whether increases in perirenal fat and/or renal sinus fat contribute to renal congestion in HF is yet to be established.

INCREASED INTRA-ABDOMINAL PRESSURE

In patients with severe HF, intra-abdominal pressure (IAP) may increase because of ascites or increased fluid in the splanchnic system in the absence of ascites (**Central Illustration**).[32] The presence of ascites and its severity have been associated with impaired renal function in HF.[32] Reduction of IAP from decongestive therapies and mechanical removal of fluid restore renal function.[33] This indicates an indirect relationship between venous congestion and impaired renal function, a direct mass effect on the retroperitoneal kidneys from the weight of the fluid-filled peritoneum, or both (**Central Illustration**). In patients with (morbid) obesity, IAP is similarly increased and decreases after weight-reduction surgery.[34-36] Moreover, several studies indicate that weight-reduction surgery improves both renal and cardiovascular outcomes in morbidly obese patients.[34-38] Two studies from the same group indicate that renal venous compression, rather than parenchymal compression, is the main driver behind decreased GFR, increases in renin and aldosterone, and onset of proteinuria in patients with intra-abdominal hypertension.[39,40]

RENAL DECAPSULATION

Loss of renal function through compression of the kidney by the renal capsule suggests that renal decapsulation may improve renal function in patients

TABLE 1 Historical Findings Indicating Pressure-Related Damage to the Kidney

Increased intracapsular pressure			
Preclinical studies[a]	Type of Animal (N)	Type of Intervention	Outcome(s)
Wegria et al (1955)[46,b]	Mongrel dogs	Clamping of the renal vein	• Proteinuria after renal venous pressure >250 mm H_2O
Stone and Fulenwider (1977)[47]	Monkeys (12)	Renal ischemia induced by clamping of the suprarenal aorta, unilateral renal decapsulation	• Preservation of creatinine and urea clearance in the decapsulated kidney
Burnett and Knox (1980)[17]	Mongrel dogs (10)	Clamping of the renal vein	• Increased renal interstitial pressure • Decreased FeNa during volume expansion
Khraibi et al (1977)[48]	Wistar rats (19)	Renal ischemia induced by clamping of the suprarenal aorta, unilateral renal decapsulation, acute volume expansion	• Attenuation of renal interstitial pressures after volume expansion in decapsulated kidney
García-Estañ and Roman (1990)[49]	Sprague-Dawley rats (27)	Renal decapsulation and infusion of ANP	• Increase in interstitial medullary pressure after ANP infusion • Normalization of cortical interstitial pressure after ANP infusion and decapsulation
Komuro et al (2017)[18]	Wistar rats (9)	Saline administration until CVPs of 10 and 15 mm Hg were attained	• Correlation between CVP and renal interstitial pressure ($r = 0.95$) • Reduced medullary perfusion, as assessed with contrast-enhanced ultrasonography
Shimada et al (2018)[20]	Sprague-Dawley rats (10)	Clamping of the renal vein, renal decapsulation	• Increased renal interstitial pressure • Proteinuria • Increased expression of markers of tubular damage • Decreased GFR and urine production • Attenuation of the above after decapsulation
Cruces et al (2018)[44]	Piglets (18)	Ischemia-reperfusion with or without renal decapsulation versus sham procedure	• Lower intrarenal pressure after renal decapsulation, as compared with ischemia without decapsulation and/or sham procedure • Lower renal lactate release after renal decapsulation, as compared with ischemia without decapsulation and/or sham procedure
Clinical studies	Study Population (N)	Type of Intervention	Outcome(s)
Stone and Fulenwider (1977)[47]	Hemorrhagic shock patients with acute tubular necrosis (21)	Unilateral renal capsular incision	• Greater renal plasma flow on the decapsulated side • Greater urine flow on the decapsulated side • Reduced incidence of anuria/oliguria as compared with anticipated incidence (7% vs 75%)
Nijst et al (2017)[23]	• Euvolemic HFrEF (40) • Euvolemic HFpEF (40)	• Infusion of 1 L hydroxyethyl starch 6% • Intravenous bolus of loop diuretic	• Blunting of intrarenal venous flow patterns after volume expansion • Return to baseline after loop diuretic administration
De la Espriella-Juan et al[50]	Acute HF (1)	Aggressive decongestive treatment	• Normalization of renal venous flow patterns
Ter Maaten et al (2021)[22]	Acute HF (15)	Standard of care decongestive treatment	• Normalization of renal venous flow patterns

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with congestive HF. This was confirmed in a rat model of renal congestion, where decapsulation reduced tubular damage.[20]

Data on decapsulation in humans is more than 100 years old.[41] In the late 19th and early 20th centuries, renal capsular incision or full decapsulation was a treatment often performed for various indications, ranging from renal abscesses to pre-eclampsia and oliguria, but not congestive HF.[41] Better alternatives such as dialysis and antibiotic treatment, as well as contradicting results on the benefit of decapsulation on renal outcomes, eventually rendered renal decapsulation obsolete in acute kidney injury.[42,43] However, recently decapsulation has been shown to alleviate ischemic acute kidney injury in piglets, potentially reviving the technique.[44] To date, no data on renal decapsulation in humans with HF exist.

RENAL TAMPONADE

We propose the renal tamponade hypothesis to explain the disproportionate impairment in renal function when central venous pressures increase in patients with HF. The renal capsule surrounding the kidney is very rigid and will not allow expansion when pressures rise. Increased central venous

TABLE 1 Continued

Increased perirenal pressure			
Preclinical studies	Type of Animal (N)	Type of Intervention	Outcome(s)
Hou et al (2014)[51]	Wistar rats (10)	High-fat versus normal diet	• PRAT was associated with infrarenal aortic endothelial dysfunction • PRAT was associated with albuminuria in obese rats
Ma et al (2016)[30]	Pigs (14)	High-fat/high-fructose versus standard diet	• PRAT in obese pigs with larger PRAT vasodilation of the renal artery was impaired. This was restored after TNF-α blockage.
Cops et al (2020)[52]	Sprague-Dawley rats (16)	• Surgical wire constriction on IVC • Randomization to sedentary versus moderately intense endurance exercise	• Endurance exercise lowered perirenal fat pad/tibia length ratio, whereas body weight remained similar to that of sedentary rats • Abdominal pressure was lowered in the endurance group • Cystatine C was lower in the endurance group • No differences between other indices of kidney function could be found
Clinical studies	Study Population (N)	Type of Intervention/ Observation	Outcome(s)
Lamacchia et al (2011)[53]	Type 2 diabetes (151)	Quantification of peri- and pararenal fat thickness on ultrasound	• PRAT was independently associated with eGFR ($\beta = -0.327$) after correction for WC and BMI.
Sun et al (2013)[54]	• Obese healthy volunteers (67) • Age- and sex-matched healthy lean volunteers (34)	Quantification of peri- and pararenal fat thickness on ultrasound	• PRAT was higher in obese patients with albuminuria than in obese patients without albuminuria
Geraci et al (2018)[24]	Hypertension (269)	Quantification of peri- and pararenal fat thickness on ultrasound	• Correlation between PRAT and eGFR ($r = -0.284$) • In multivariable regression, PRAT remained an independent predictor of eGFR after correction for BMI and WC
Ricci et al (2018)[55]	Morbid obesity (284)	Sleeve gastrectomy	• PRAT was an independent predictor of systolic blood pressure in obese subjects ($\beta = 0.160$) after rigorous correction for known risk factors • PRAT significantly reduced after sleeve gastrectomy
D'Marco et al (2019)[26]	Chronic kidney disease (classes I–V) (103)	Perirenal fat between renal cortex and hepatic/splenic border measurement on ultrasound	• PRAT was larger in patients with prediabetes, CKD stage 4 or 5, and higher triglyceride levels but not in those with a history of hypertension
Koo et al (2020)[25]	Community-based cohort (3,919)	Estimation of PRAT on CT	• PRAT was associated with renal (OR 2.05) and aortic (OR 1.11) atherosclerosis
Notohamiprodjo et al (2020)[56]	• Healthy normoglycemic (230) • Prediabetes (87) • Type 2 diabetes (49)	Quantification of renal sinus fat volume on CMR	• Diabetes ($\beta = 7.34$) and prediabetes ($\beta = 7.13$) were significantly associated with more renal sinus fat, compared with normoglycemia in control individuals.
Spit et al (2020)[31]	Type 2 diabetes (51)	Quantification of renal sinus fat volume on CMR	• Correlation between renal sinus fat and GFR ($r = -0.38$) • Correlation between renal sinus fat and effective renal plasma flow ($r = -0.38$)
Fang et al (2020)[57]	Type 2 diabetes (171)	Ultrasound assessment of PRAT diameter	• Correlation between PRAT and eGFR diameter ($r = -0.181$)

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pressures lead to increased renal interstitial pressures, compressing renal structures such as the tubules, intrarenal veins, and glomeruli in the encapsulated kidney. Future research is warranted to further elucidate the relationship between HF, congestion, obesity, and impaired renal function. Whereas decreased renal perfusion may be difficult to influence, and indeed, attempts to improve renal perfusion have shown not to be associated with improved outcome,[45] intrarenal congestion may possibly be a treatment target. Ultimately, renal decompression therapies may be a novel therapeutic field to explore in decreasing the incidence of worsening renal function and worsening HF.

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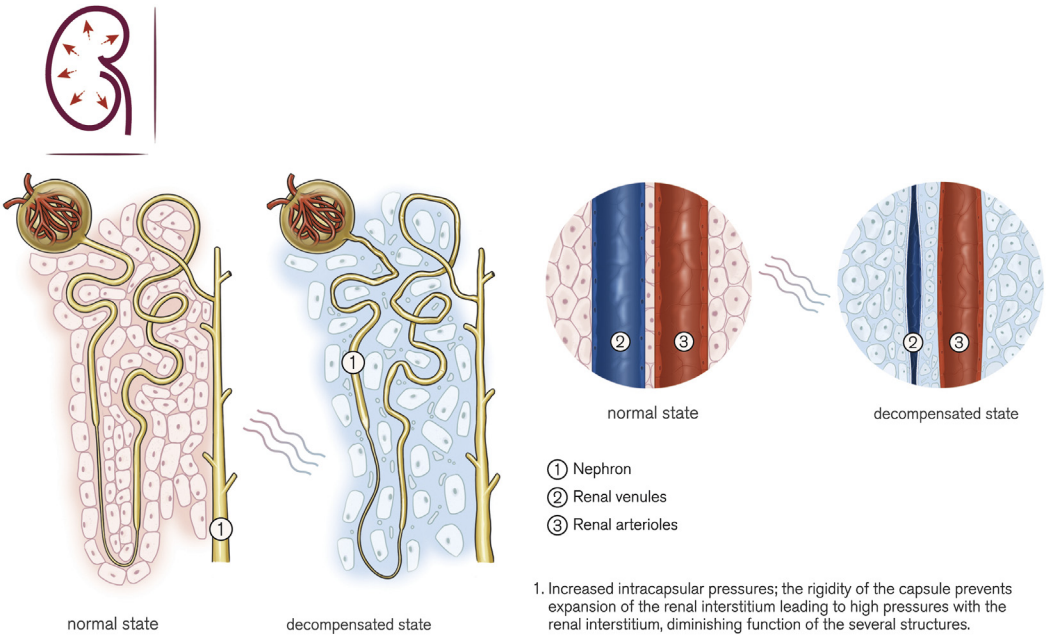
TABLE 1 Continued

Increased intra-abdominal pressure[a]			
Preclinical studies	Type of Animal (N)	Type of Intervention	Outcome(s)
Harman et al (1982)[58]	Mongrel dogs (7)	Insertion of inflatable bags in the peritoneum	• At 20 mm Hg, GFR decreased to <25% of baseline value • At 40 mm Hg, dogs became anuric and <7% of baseline GFR remained • CO also diminished to 37%, an effect that could be attenuated by infusion of fluids
Bishara et al (2011)[59]	• Sprague-Dawley rats, aortocaval fistula, compensated (86) • Sprague-Dawley rats, aortocaval fistula, decompensated (6) • Sprague-Dawley rats, aortocaval fistula, decompensated and pre-treated with NOS inhibitor (6)	Pneumoperitoneum induced by injection of air through a needle	• After IAP >14 mm Hg, GFR, urinary flow, and sodium excretion decreased • This effect was most pronounced in decompensated rats • Pretreatment with NOS inhibitor exacerbated the decline in renal function
Abu-Saleh et al (2019)[60]	• Sprague-Dawley rats, sham operated (23) • Sprague-Dawley rats, aortocaval fistula, compensated (8) • Sprague-Dawley rats, aortocaval fistula, decompensated (9), and pretreated with tadalafil (6) • Sprague-Dawley rats, acute myocardial infarction (7), and pretreated with tadalafil (11) • Sprague-Dawley rats, sham treated with tadalafil (6)	Pneumoperitoneum induced by injection of air through a needle	• After IAP of 10 mm Hg RPF, GFR, urinary flow and sodium excretion all diminish • This effect is most pronounced in decompensated ACF rats • This effect is ameliorated by pretreatment with tadalafil • GFR recovered after IAP normalizes in all groups • In sham and MI rats, natriuresis increases to above baseline level after IAP is normalized, in decompensated rats natriuresis remains low
Clinical studies	Study Population (N)	Type of Intervention/ Observation	Outcome(s)
Sugerman et al (1998)[61]	Morbidly obese (15)	• Measurement of abdominal pressure using urinary catheter manometer • Roux-en-Y gastric bypass	• Significant reduction of IAP 1 y after GBS
Nguyen et al (2001)[36]	Morbidly obese (64)	• Randomized to either open or laparoscopic gastric bypass	• IAP increased in both groups on day 1 postoperatively • IAP returned to baseline in the laparoscopic group on day 2 postoperatively • No data on long-term change in IAP
Lambert et al (2005)[62]	Morbidly obese (45)	• Measurement of abdominal pressure using urinary catheter manometer • Roux-en-Y gastric bypass	• Obese patients have a higher IAP compared with general population • IAP correlates to number of comorbidities • IAP is not reduced by incising the peritoneum • The first 2 days post operatively IAP increases • No long-term data on IAP after GBS
Mullens et al (2008)[63]	Acute decompensated HF refractory to treatment (9)	Paracentesis (5) or ultrafiltration (4)	• After mechanical fluid removal IAP was significantly reduced (mean reduction 5 mm Hg) • After mechanical fluid removal, renal function improved (mean serum creatinine from 3.4 ± 1.4 mg/dL to 2.4 ± 1.1 mg/dL)
McIsaac et al (2019)[37]	Morbidly obese (471)	Gastric bypass (sleeve gastrectomy or Roux-en-Y)	• In patients with microalbuminuria, GFR increased from 109 ± 10 mL/min to 120 ± 36 mL/min 24 months after GBS • In patients with microalbuminuria, albumin-creatinine-ratio normalized 24 months after GBS • No data on IAP either pre- or postoperatively
Rubio-Gracia et al (2020)[64]	Acute decompensated HF (43)	• Measurement of abdominal pressure using urinary catheter manometer • Standard of care decongestive treatment	• Higher baseline IAP was associated with poorer diuretic and natriuretic response • Higher baseline IAP was associated with higher serum creatinine • IAP >12 mm Hg after 72 h of decongestive treatment was associated with higher mortality and rehospitalization rates

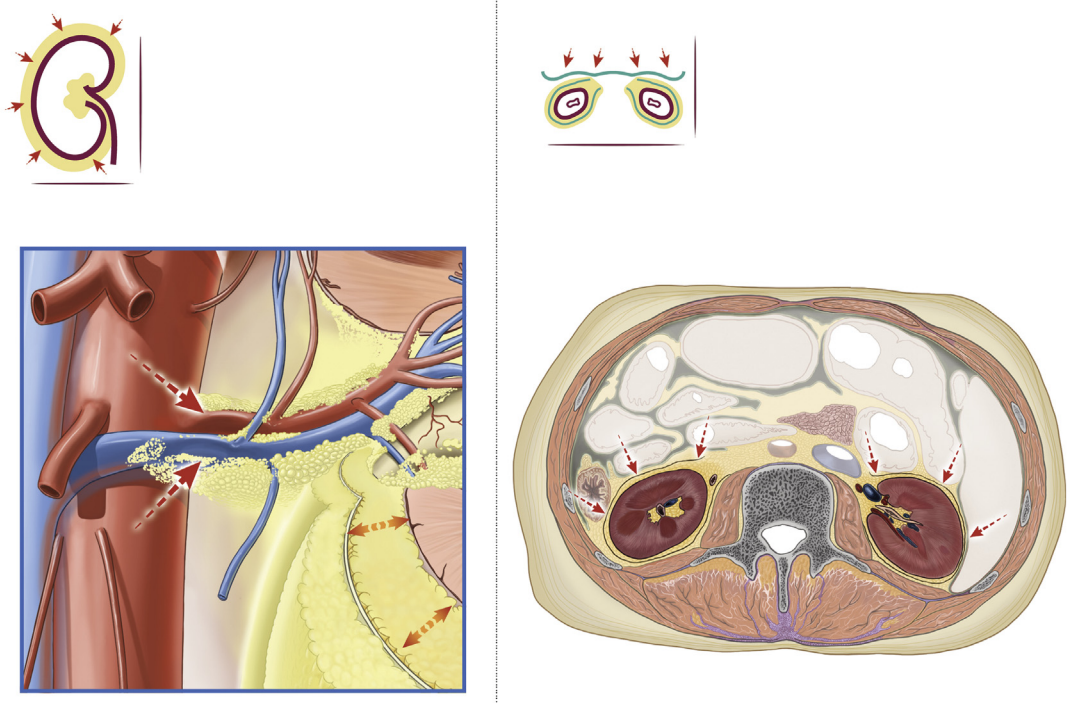
[a]Studies published in any language other than English, or with no available abstract, were omitted from inclusion in this table. [b]References as they appear in reference list.

ACF = aortocaval fistula; ANP = atrial natriuretic peptide; BMI = body mass index; CKD = chronic kidney disease; CMR = cardiac magnetic resonance; CT = computed tomography; CVP = central venous pressure; eGFR = estimated glomerular filtration rate; FeNa = fractional excretion of sodium; GBS = gastric bypass; HF = heart failure; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; IAP = intra-abdominal pressure; IVC = inferior vena cava; mm H_2O = millimeters water pressure; NOS-inhibitor = nitric oxide synthase - inhibitor; PRAT = perirenal adipose tissue; RPF = renal plasma flow; TNF-α = tumor necrosis factor-α; WC = waist circumference.

CENTRAL ILLUSTRATION Increased Intracapsular, Perirenal, and Intra-Abdominal Pressures



1. Increased intracapsular pressures; the rigidity of the capsule prevents expansion of the renal interstitium leading to high pressures with the renal interstitium, diminishing function of the several structures.



2. Increased perirenal pressure; increases in perirenal adipose tissue compress renal vasculature, exacerbating intrarenal congestion.

3. Increased peritoneal pressure; the weight of the peritoneal space, from either fat or fluid, compresses the renal vasculature, exacerbating renal congestion.

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Above, effect of intrarenal congestion. As fluid exits the bloodstream, pressures rise within the rigid renal capsule, compressing the tubules and the intrarenal venules. Below left, consequences to the perirenal adipose tissue, leading mainly to compression of renal vasculature. Below right, kidneys in the retroperitoneal space. The weight of the fat or fluid in this compartment compresses the renal vasculature.

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