channel blocker (CCB) – Cilnidipine, has emerged as a promising molecule in India and most south-east Asian countries, for its significant benefits in terms of efficacy & side effect profile over conventional CCBs.

Cilnidipine has a novel mode of action of blocking both the L&N calcium channels and hence it not only provides optimal BP control, but also possesses reno-protective properties.

Several randomized clinical trials conclude that Cilnidipine has reno-protective benefits in hypertensive patients, with multiple modes of action on the kidneys, reducing proteinuria and retarding the decline in eGFR. However, there is a lack of real-world Indian data on the effectiveness & side effect profile of Cilnidipine.

**Methods:** This single-center, retrospective study was conducted with an objective to assess the effectiveness and safety of Cilnidipine in Indian hypertensive patients with albuminuria, from electronic medical records (EMR) of the Nephrology department of a multi-specialty hospital in India.

The EMR data of adult patients who were diagnosed with essential hypertension and were prescribed cilnidipine was retrospectively analyzed. The index date when the patient was initiated on Cilnidipine [10mg/20 mg BID] was considered as the baseline visit. Patients were classified based on the total number of antihypertensive (AHD) drugs prescribed along with cilnidipine. Data was collected and analyzed at the baseline visit and then at 6 months, 12 months and 24 months. Primary end point was the change in systolic blood pressure (SBP) and diastolic blood pressure (DBP) from baseline. Reduction of albuminuria and overall safety were secondary end points. The data was statistically analyzed using repeated measures ANOVA.

**Results:** Data of 151 patients was included in the study analysis. The mean age, height, weight and BMI were 55.8±1.0 years, 1.64 ± 0.003 meters, 66.8 ± 0.9 Kgs and 24.6 ± 0.3 kg/m2 respectively.

Out of the 151 patients, 114 patients were prescribed Cilnidipine + 1 AHD and 37 were prescribed Cilnidipine + 2 AHDs.

The mean ± SEM [95% confidence interval (CI)] change in the cilnidipine + 1 AHD group at 24months follow-up was SBP [14.4 ± 1.0, (12.0, 16.8) $P < 0.001$] mm Hg and DBP [8.5 ± 0.7 (6.8 to 10.2) $P < 0.001$] mm Hg, while the change in cilnidipine + 2 AHD group at 24-months follow-up was SBP [13.6 ± 1.7 (9.5 to 17.7), $P < 0.001$] mmHg and DBP [5.2 ± 1.3 (2.1 to 8.3), $P < 0.001$] mmHg.

The mean reduction in albuminuria was [36.2 ± 3.8 (27.2 to 45.2)] mg/dl and [73.6 ± 13.4 (40.7 to 106.4)] mg/dl in the Cilnidipine + 1 AHD and Cilnidipine + 2 AHD groups respectively ($p<0.001$).

The addition of Cilnidipine to the overall treatment regimen was well tolerated by the patients.

**Conclusions:** Addition of the novel, reno-protective CCB, Cilnidipine, to existing AHD regimens during routine clinical practice, significantly reduced the blood pressure and albuminuria and was well tolerated in Indian hypertensive patients.

I have no potential conflict of interest to disclose.

## WCN24-1739

### MALIGNANT ARTERIAL HYPERTENSION (MAH): THE OLDEST CAUSE OF THROMBOTIC MICROANGIOPATHY THAT IS A MEDICAL DIAGNOSIS CHALLENGE. A CASE REPORT

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**Introduction:** Thrombotic Microangiopathies (TMAs) result from various pathogenic mechanisms (immune, infectious, complement dysfunction, toxins, shear stress), sharing a common final phenotype, the damage of endothelial cells leading to microcirculation thrombosis. They are clinically characterized by microangiopathic hemolytic anemia (MAHA), thrombocytopenia, and ischemic damage to multiple organs.

Malignant Arterial Hypertension (MAH) is characterized by markedly elevated blood pressure (often exceeding 200/130 mmHg) associated with Grade III retinopathy, with or without papilledema, and multiorgan involvement due to diffuse arteriolar damage. Renal involvement varies and may manifest as severe renal failure associated with TMA. The pathogenic mechanism is linked to an increase in angiotensin II and a decrease in nitric oxide, resulting in vasoconstriction and platelet aggregation.

Treatment strategies vary depending on the etiology. The differential diagnosis is essential from the beginning, sometimes the diagnosis is made by rule out as we share in this case.

**Methods:** A 47-year-old male with a history of untreated hypertension presented at the ER with headache and blurred vision. His blood pressure was 180/100 mmHg, and he exhibited psychomotor excitement, with a brain CT scan showing no abnormalities. Laboratory results revealed anemia (Hb 10.5 g/dl), thrombocytopenia (86000/mm$^3$), and renal insufficiency (Urea 130 mg/dl, Cr 3.4 mg/dl). Urinalysis indicated findings compatible with ATN, and proteinuria 2.1 g/day. Peripheral blood smear showed schistocytes (4 per field), elevated LDH, and a negative direct Coombs test. The diagnosis of AKI KDIGO III due to TMA was made. Test fo rruling out rheumatological and infectious causes were negative. ADAMTS13 activity was requested, and treatment with plasma exchange and corticosteroids was initiated. This resulted in the disappearance of schistocytes on the peripheral blood smear and correction of thrombocytopenia, but no improvement in renal function.

**Results:** The patient developed hypertensive encephalopathy (Grade IV retinopathy, brain MRI showingfindingsconsistentwithhypertension-relatedmicroangiopathy). He required titratable antihypertensive medications, leading to improved blood pressure and neurological status. Secondary causes of hypertension were ruled out. Renal biopsy revealed mesangial expansion, mesangiolysis, double contour formation in glomeruli, 35% interstitial atrophy and fibrosis, and arterioles with muscular hyperplasia resembling "onion skin." Immunofluorescence was negative, and electron microscopy showed no deposits. The ADAMTS13 activity was normal. Exome sequencing directed toward atypical hemolytic uremic syndrome was negative for clinically significant variants, leading to the interpretation of TMA secondary to MAH.

**Conclusions:** With the advent of antihypertensive therapies, the incidence of severe renal involvement in MAH has been decreasing in the last decades. The diagnosis of these patients is a real challenge, because TMAs from various causes can be associated with severe hypertension. It is essential to have complete diagnostic study from the beginning to adequate the management strategies. In our case, based on all the studies conducted, we can conclude that the TMAs were secondary to MAH because we could not find another factor associated.

I have no potential conflict of interest to disclose.

## WCN24-1762

### URGENT CARDIOVASCULAR CASES IN PATIENTS DIAGNOSED WITH DIABETES AND DE NOVO IN A HEALTHCARE PROVIDER IN MAGANGUÉ (BOL) BETWEEN JANUARY 2021 AND JULY 2023

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**Introduction:** The relationship between diabetes and cardiovascular risk has been extensively studied; the mortality associated with these two conditions remains a clinical and public health concern. The objetive was to assess cardiovascular emergencies in patients with pre-existing and newly diagnosed diabetes at a healthcare facility in Magangué, Bolívar, between January 2021 and July 2023.

**Methods:** An analytical study was conducted at a clinic in Magangué, Bolívar, spanning from 2021 to 2023. All patients admitted for cardiovascular emergencies were included, with 29% having diabetes mellitus. Descriptive summaries were made for quantitative and categorical variables. Statistical tests including Wilcoxon and Chi-square were used to evaluate differences between patient groups. A multivariate logistic regression model was applied to identify factors associated with cardiovascular mortality, calculating adjusted odds ratios with 95% confidence intervals. A p-value $<0.05$ was considered significant. R-CRAN software version 4.3.2 was used for statistical analysis.

**Results:** A total of 452 patients admitted for cardiovascular emergencies were studied. The majority were females with an average age of 63

years. The most prevalent comorbidities were dyslipidemia, acute myocardial infarction, obesity, and hypertension. Headache was the most common reason for consultation. Among patients with type 2 diabetes, pre-existing cases exhibited more comorbidities and renal issues, while new cases had poorer glycemic control. Survivors had higher systolic blood pressure compared to non-survivors. Obesity significantly increased the risk of cardiovascular mortality.

**Conclusions:** Traditional risk factors remain crucial for monitoring, emphasizing the need to strengthen primary care for early detection and reduction of catastrophic events in individuals at risk for type 2 diabetes.

I have no potential conflict of interest to disclose.

## WCN24-2027

### AN UNUSUAL CAUSE OF HYPOVOLEMIA HYPONATREMIA INDUCED BY INDAPAMIDE AND SUCCESSFULLY CORRECTED WITH FLUDROCORTISONE

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**Introduction:** Hyponatremia has been associated with confusion, lethargy, seizures, coma, and even death. Hyponatremia is related to many conditions. It has been reported that patients receiving diuretics like Indapamide can cause hyponatremia. To describe one patient with severe indapamide-induced hypovolemic hyponatremia and correcting with Fludrocortisone and 1-deamino-8-d-arginine vasopressin; DDAVP.

**Methods:** A 51-year-old man was admitted to the hospital with a seizure and severe hyponatremia (plasma sodium concentrations of 101 mmol/L) and hypokalemia (plasma potassium concentrations of 1.9 mmol/L) 10-12 weeks after they received indapamide 2.5 mg/d therapy for hypertension. An urgent CT scan of the brain was done, and we did not find any acute brain injury or focal seizure.

On examination, he had moist mucous membranes and normal skin turgor without postural hypotension and no signs of overload like raised jugular venous pressure (JVP), peripheral edema or shortness of breath. From the laboratorium, serum osmolality was 244 mmol/kgH2O (NR: 275295 mmol/kgH2O) correlating with the hypo-osmolar state, and urine osmolality was in the normal range 186 mosm/kgH2O (NR: 150-1150 mosm/kgH2O). Level sodium urine was 192 (NR:60-180) mmol/L, Calcium ion was 0,85 mmol/L and Urine volume was 9890 ml for 24 hours. The treatment was based on the correction of fluid and electrolyte disturbances using isotonic saline, and 3% hypertonic saline via a central line.

**Results:** On 5th day of hospitalization, treatment with fludrocortisone 200 mcg was initiated, with a marked improvement in hyponatremia. We give fludrocortisone for seven days. To control polyuria, oral DDAVP 2 mcg was administered, which decreased her urine output without causing hyponatremia. DDAVP was continued twice daily for three days with significant improvement in his urine output.

We followed up within 7th day of hospital discharge; the plasma sodium was 140 mmol/L. Fludrocortisone was administered to maintain a level of sodium. It exerts its effects by stimulating the reabsorption of sodium and water in the distal tubule, leading to the expansion of the ECFV.

**Conclusions:** Indapamide can cause both severe hypokalemia and hypovolemia hyponatremia. Rapid correction of natrium and volume status with the combination fludrocortisone and DDAVP can be considered to patient with polyuria and severe hyponatremia.

I have no potential conflict of interest to disclose.

## WCN24-2065

### THE EFFECT OF THE PRESENCE OF MULTIPLE (ACCESSORY) RENAL ARTERIES ON BIOCHEMICAL PARAMETERS OF RENAL FUNCTION AND HEMODYNAMIC MEASUREMENTS

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**Introduction:** The prevalence of accessory or multiple renal arteries in the general population varies widely based on the population examined, from 4% in Malaysians to 61.5% in Brazilians. Still, the average global prevalence among adults is estimated to be around 30%. The closest geographical region closest to the Republic of Croatia with available data on the prevalence of accessory renal arteries is Bosnia and Herzegovina, where a 25.8% prevalence in adults is reported. The effect of having accessory renal arteries on hemodynamics and renal function has been studied in different population, yielding variable results – from a significant correlation between the presence of accessory renal arteries and increased arterial blood pressure and/or serum creatinine and renin to no correlation whatsoever. The aim of this study was to evaluate the effects of the presence of accessory renal arteries on biochemical parameters related to renal function (renin, aldosterone, serum creatinine, serum electrolytes) and hemodynamics in a Croatian population managed at a tertiary care center.

**Methods:** This was a retrospective single-center study. Data was collected from the hospital informatics system on patients with accessory renal arteries managed in the arterial hypertension clinic of a tertiary care center. Patient data obtained between 2012 and 2023 was included. In order to be included into the study, patients needed to have available data on laboratory parameters, hemodynamics and an image of a multi-slice computed tomography intravenous contrast-enhanced scan of the aorta and renal arteries. Patients without all of the aforementioned data were excluded from the study. Data was analyzed using the JASP open-source statistical software. Statistical significance was set at a threshold of $p < 0.05$.

**Results:** 155 patients in total were included in the study – 80 without accessory renal arteries and 75 with one or more accessory renal arteries. The distribution of age and gender was equivalent between the two groups. The group with accessory renal arteries had significantly higher mean arterial pressures (calculated as 0.66*diastolic blood pressure + 0.33*systolic blood pressure) (100 mmHg vs 95.7 mmHg, $p = 0.016$), higher serum creatinine concentrations (84.5 umol/L vs 74 umol/L, $p < 0.001$) and had a significantly higher number of antihypertensive medications in their therapy (3 vs 1.5 different types of medications, $p < 0.001$). There were no significant differences between other measured parameters (serum electrolytes, renin, aldosterone, aldosterone/renin ratio, body mass index).

**Conclusions:** In this retrospective single-center study, patients with arterial hypertension who had accessory renal arteries had significantly higher mean arterial pressure and serum creatinine values and required more antihypertensive medications to maintain adequate arterial pressure values. These results indicate that the presence of accessory renal arteries might have an effect on the success of antihypertensive therapy and that these patients may represent a distinct phenotypic group that requires a personalized approach.

This abstract was also submitted for the 10th Conference of the Croatian Society of Nephrology, Dialysis and Transplantation in Zagreb, Croatia.

I have no potential conflict of interest to disclose.

## WCN24-2118

### HYPERKALAEMIA IN PATIENTS WITH CHRONIC KIDNEY DISEASE AND HEART FAILURE

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**Introduction:** The prevalence of heart failure (HF) with chronic kidney disease (CKD) is increasing. Both conditions are associated with poor outcomes including fluid overload causing hospitalisations and mortality. Patients with CKD are also less likely to have optimal pharmacological therapies for HF due to concerns over hyperkalaemia and declining renal function. This project aims to determine the prevalence of hyperkalaemia in CKD-HF patients and determine which factors are associated with hyperkalaemia.

**Methods:** Data on consecutive patients attending a CKD-HF clinic between 12th April 2019 and 11th September 2021 were retrospectively