HYPERTENSIVE CRISES AT THE EMERGENCY ROOM – WHO ARE THE PATIENTS? A TERTIARY CENTER EXPERIENCE.

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Objective: Hypertension pandemics is well recognized and with its low control rates can lead to hypertensive crisis and patients visiting excessively the Emergency Department (ED). The aim of the current study is to describe the profile of the patient presenting with a hypertensive crisis at the ED of a tertiary care emergency hospital.

Design and method: We retrospectively analysed all the charts of patients presenting at the ED of a tertiary care emergency hospital during 1 month (March 2018). We retrieved complete demographic, clinical, paraclinical and treatment data from the ED charts of patients presenting for symptomatic or asymptomatic hypertension (HTN) defined as blood pressure (BP) =>140/90 mmHg. We defined a HTN emergency (EMG) as BP =>180/120 mmHg with acute HTN-mediated organ damage (HMOD) and HTN urgency as BP =>180/120 mmHg without acute HMOD (URG). In all other cases BP =>140/90 mmHg was referred to as elevated BP (EBP).

Results: 5898 patients presented at the ED during 1 month, from which we studie 293 pts. evaluated for HTN (4.96% from all presentations). 48.2% were true HTN crises while the reminder presented for EBP/ Patients presenting for EMG (only 12.2%) were older, almost all had previous HTN and a higher number of comorbidities including higher prevalence of atrial fibrillation, had a longer stay in the ED while they were more likely to be admitted. See table for results.

Conclusions: Hypertensive disease remains a frequent cause of ED presentations with only half of cases representing true urgencies or emergencies, overburdening the emergency health care system. Patients with true hypertensive EMG represent a vulnerable category as they are older, with a higher number of comorbidities making them more likely to be admitted.

Characteristics	TOTAL	EBP	EMG	URG	*P value* (URG vs EMG)
No. (%)	293	152 (51.8)	36 (12.2)	105 (35.8)	-
Age (mean±SD) (years)	62.9 ± 14.5	59.6 ± 15.5	70.0±12.2	65.1±12.4	**0.02**
Male, (No, %)	131 (44.7)	74 (48.6)	17 (47.2)	40 (38.0)	0.19
Rural areas, (No, %)	229 (78.1)	115 (75.6)	28 (77.7)	86 (81.9)	0.58
Known HTN, (No, %)	205 (69.9)	96 (63.1)	33 (91.6)	76 (72.3)	**0.00**
sBP - arrival ED (mmHg) (mean±SD)	177.9 ± 22.2	160.8 ± 10.0	200.6 ± 21.0	195.5 ± 15.1	0.3
sBP - leaving ED (mmHg) (mean±SD)	141.0 ± 14.1	137.0 ± 10.0	153.1± 22.1	142.9 ± 13.1	**0.02**
Headache (No, %)	79 (26.9)	34 (22.3)	6 (16.6)	39 (37.1)	**0.02**
Chest pain (No, %)	40 (13.6)	20 (13.1)	12 (33.3)	8 (7.6)	**0.00**
Dispneea (No, %)	39 (13.3)	13 (8.5)	9 (25)	17 (16.1)	0.23
Epistaxis (No, %)	10 (3.4)	3 (1.9)	1 (2.7)	6 (5.7)	0.48
Dizziness (No, %)	58 (19.7)	21 (13.8)	6 (16.6)	31 (29.5)	0.13
Admitted (No, %)	62 (21.1)	31 (20.3)	23 (63.8)	8 (7.6)	**0.00**
Average no. of anti-HTN drugs (home)	1.25 ± 1.29	1.23 ± 1.32	0.86 ± 1.13	1.42 ± 1.27	**0.02**
Average no. co-morbidities	0.97 ± 1.15	0.8 ± 1.12	1.57 ± 1.14	1 ± 1.12	**0.01**
Atrial fibrillation (No, %)	27 (9.21)	10 (6.57)	9 (25)	8 (7.6)	**0.00**
Average time spent in ED (min) (mean±SD)	218.7±126.5	194.3±20.7	234.8±40.1	249.6±123.7	0.59

GENETICS OF HYPERTENSION ENDOPHENOTYPES AND METABOLIC TRAITS HIGHLIGHTS SHARED BIOLOGICAL PATHWAYS

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Objective: Hypertension frequently co-occurs with coronary artery disease (CAD), type 2 diabetes (T2D), obesity, and systemic inflammation. An overlap between the risk factors for hypertension, cardiovascular disease, and diabetes, as well as their overwhelming comorbidity suggests that these conditions share pathophysiological mechanisms. Large-scale genome-wide association studies (GWAS) have revealed hundreds of genetic loci associated with systolic, diastolic, and pulse pressure (SBP/DBP/PP), hypertension and other phenotypes indicative of metabolic health. We aimed to dissect shared genetic factors contributing to their comorbidity using multi-phenotype approach.

Design and method: Using summary statistics from published GWAS available in public domain, including those from GWAS consortia ICBP (blood pressure, BP), CARDIOGRAM (cardiovascular), DIAGRAM/DIAMANTE (T2D), MAGIC/ENGAGE (glycaemic), GLGC/ENGAGE (lipids), GIANT (obesity), we analysed 3420 single nucleotide variants (SNVs) associated with BP traits, including SBP/DBP/PP, and 60 other cardiometabolic phenotypes (defined as related to cardiovascular and metabolic diseases).

Results: We identified 252 unique BP association signals, and found 301 BP SNVs overlapping with 540 SNVs reported for other phenotypes and located within 100 kb from one another. Further analysis revealed 22 SNVs associated with two and more cardiometabolic phenotypes. Specifically, rs13107325 in SLC39A8 gene was associated with mean arterial pressure, body mass index (BMI), and high density lipoprotein cholesterol; rs9368222 in CDKAL1 was associated with SBP, fasting glucose (FG), and T2D; rs3184504 in SH2B3 influenced DBP, risk of stroke, CAD, and ICAM1 levels; rs11556924 in ZC3HC1 affected DBP/SBP, and susceptibility to CAD; rs757081 in NUCB2 had an effect on PP/SBP, height and T2D; rs10830963 in MTNR1B altered PP, T2D risk, FG and corrected insulin response; rs653178 in ATXN2 influenced changes in DBP, ICAM1 level, and type 1 diabetes; and rs12454712 in BCL2 was related to higher SBP, waist-to-hip ratio adjusted for BMI, and T2D susceptibility.

Conclusions: Overall, we highlighted pathways associated with cardiometabolic traits, including circadian rhythm regulation (MTNR1B), pancreatic islet function (CDKAL1), appetite control and nutritional regulation (NUCB2 and ATXN2), immune and inflammatory signaling (SH2B3 and SLC39A8), cell cycle regulation and apoptosis (ZC3HC1 and BCL2), thus suggesting shared biological mechanisms between hypertension endophenotypes and cardiometabolic traits.

DIETARY SODIUM-INDUCED BLOOD PRESSURE RISE IS ASSOCIATED WITH A PRO-INFLAMMATORY PHENOTYPE OF CLASSICAL MONOCYTES – A RANDOMIZED CONTROLLED TRIAL IN HEALTHY HUMAN SUBJECTS

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Objective: High salt intake is associated with hypertension and cardiovascular disease. The mechanisms behind this are not fully elucidated, and may extend beyond the kidney. Proposedly, inflammation is involved. We investigated the effect of high salt intake on circulating monocytes and skin macrophages of healthy human subjects.

Design and method: We performed a randomized cross-over trial in healthy males. All subjects pursued a 2-week low salt diet (LSD: < 3 grams NaCl/day) and high salt diet (HSD: > 12 grams NaCl/day) in randomized order. After each diet, body weight and blood pressure were measured, and blood and urine samples were obtained. Flow cytometry was used for phenotypic characterization of monocytes into classical (CD14+CD16-), intermediate (CD14+CD16+) and non-classical (CD14-CD16+) subtypes. We examined the expression of pro-inflammatory chemokine receptors (CCR2, CCR5, CCR7, CXCR1), anti-inflammatory chemokine receptors (CD206, CD200R) and molecules associated with migration (CD62L, CD49d, CD29, CD11b, CD11c, CD18). In addition, LPS-induced cytokine secretion (IL-6, IL-8, IL-10, IL-12, TNF) in whole blood was investigated. Furthermore, after each diet, skin biopsies were taken, to investigate skin macrophage density (CD163) and macrophages expression of the M1 marker HLA-DR and the M2 marker CD206.

Results: Eleven subjects were included in this study. HSD increased body weight and systolic blood pressure, whereas CRP and monocyte number did not differ between LSD and HSD (Table 1). HSD increased CCR2 expression on classical monocytes (Figure 1A) and induced a trend towards decreased CD206 expression (Figure 1B). There was no effect on the other chemokine receptors or on the molecules associated with migration. Furthermore, HSD increased LPS-induced IL-6 secretion (Figure 1C) without any effect on the other cytokines. Macrophage density significantly increased after HSD (Figure 1D) and showed increased HLA-DR expression but decreased CD206 expression (Figure 1E+1F).