O-19

PRIMARY ALDOSTERONISM IN A REFERRAL HYPERTENSION CENTER
Giuseppe Regolisti, Franco Perazzoli, Aurelio Negro, Carlo Sani, Simona Davoli, Pietro Coghi, Ermanno Rossi. 1 II Division of Internal Medicine, Arcispedale S. Maria Nuova, Reggio Emilia, Italy

Primary aldosteronism (PA) is a potentially curable form of secondary hypertension, but is regarded as being relatively rare in unselected hypertensive populations. The prevalence and characteristics of PA were evaluated on 1046 subsequent hypertensive patients (pts) sent to a referral Hypertension Center between January 1st 1997 and December 31st 1999. Screening of PA was performed with the captopril test. Final diagnosis was based on lack of suppression of aldosterone upon acute volume expansion. Aldosteronoma (A) was defined as a unilateral adrenal nodule on CT scan with enhanced uptake at 111-I-cholesterol scintigraphy. The diagnosis of idiopathic hyperaldosteronism (IHA) was based on evidence of bilateral adrenal hyperplasia on CT scan and enhanced bilateral uptake at scintigraphy. Sixty-six (6.3%) pts were finally diagnosed as having PA. In 16 (24.2%) of these pts, A was demonstrated by adrenal CT and scintigraphy, and the diagnosis histologically confirmed in the 10 cases so far submitted to surgery. In the remaining 50 (75.8%) pts IHA was diagnosed. The pts with PA had slightly higher systolic blood pressure values than those with essential hypertension (EH) (171.8±23.3 vs 166.9±14.1 mmHg, P<0.05 by t-test). Known duration of hypertension was greater in the pts with PA, although statistical significance was reached only in those with IHA (median 25.5-7.5 percentile): EH 28 (6-60), IHA 60 (24-120), A 46 (5-87) mo; P=0.004 by ANOVA). At the time of the first visit, 30/66 (45.5%) pts with PA were treated with 2 or more drugs, compared with 152/931 (16.3%) pts with EH (x^2=33.117, P<0.0001); in the former group there were 23/50 (46%) cases of IHA and 9/16 (56.3%) cases of A (x^2=0.182, P=0.670). In the group of the untreated pts there were 336/931 (57.6%) cases of EH, 13/50 (26.0%) cases of IHA and 4/16 (25.0%) cases of A (x^2=25.260, P<0.0001). In these untreated hypertensives the classification was classified as mild to moderate (i.e., <180/104 mmHg) in 318/536 (59.5%) cases of EH, 8/13 (61.5%) cases of IHA and 3/4 (75.0%) cases of A (x^2=0.428, P=0.807). Serum potassium values were significantly lower in the pts with either IHA or A compared with those with EH (EH 4.0±0.3, IHA 3.6±0.3, A 3.3±0.5 mEq/l; P<0.0001 by ANOVA). However, 37/66 (56.1%) pts with PA had serum potassium values ≥3.6 mEq/l; in this group there were 33/50 (66.1%) cases of IHA and 7/16 (43.7%) cases of A (x^2=1.668, P=0.197). We conclude that: 1) PA is more frequent than traditionally thought; 2) it is not necessarily associated with severe and/or resistant hypertension; 3) IHA seems to be more prevalent than A; 4) hypokalemia is not a sensitive criterion for the screening of PA.

Key Words: secondary hypertension, primary aldosteronism, prevalence

O-20

THE ROLE OF SPIRONOLACTONE IN THE TREATMENT OF PATIENTS WITH REFRACTORY HYPERTENSION
James M. Ouzan, Catherine Perault, Evelyne Carre, Michel Mertes, Thierry Corcos. 1Medical, CLINIC, Reims, France, Metropolitan; 2Institute, Jean Godinot, Reims, France, Metropolitan; 3Department, Cardiology, Reims, France, Metropolitan

The aim of this study was to show how the adjunction of Spironolactone(Spi) to the treatment of refractory hypertensive patients (RH) might permit an optimal blood pressure (BP) control.

Methods: Among all the 520 patients who have been referred for treatment of hypertension in a medical clinic from 1997 to 1999, 25 patients had refractory hypertension. The inclusion criteria were 1) hypertension of at least six months without any apparent cause 2)clinical

BP above 140/90 mm Hg in spite of at least two antihypertensive drugs (AT) given at optimal dosage 3) no previous use of Spi 4) proper 24 hours ambulatory blood pressure monitoring (ABPM) showing an average BP above 140/90 mm Hg. Potassium and serum creatinine were checked before the introduction of Spi and one month after. In case of renal insufficiency (creatinine above 15 mg/l) patients were excluded from the treatment by Spi. If patients were on Angiotensin Converting Enzyme inhibitor (ACE), this drug was replaced by Spi at a dosage of 1 mg/kg. Otherwise, Spi was added to the previous treatment at the same dosage.

Results: After one month of treatment by Spi, 23 patients had a clinical BP below 140/90 mm Hg. On the whole population of RH patients, ABPM showed a significant decrease in both systolic (151.6±1.8 mm Hg to 128.6±2.2 mm Hg p<0.001) and diastolic pressure (85.7±1.8 mm Hg to 77.6±1.8 mm Hg p<0.013). Three months after introduction of Spi, the decrease in the number of AT, which was made possible by the introduction of Spi, was highly significant (p<0.001). No biological side effect was observed. Two patients experienced clinical side effects. In these two cases, Spi was replaced by ACE, which turned out to be more effective after treatment by Spi than previously .

Conclusion: Hypersecretion of Aldosterone may be underestimated in patients with RH. The use of Spi for these patients is safe, efficient and gives durable results, which leads to a simplification of the initial treatment.

Key Words: Spironolactone, Ambulatory blood pressure monitoring, Refractory hypertension
thelin-1 (2.8-fold), and a significant decrease in the gene expression of myosin heavy chain-α compared to VEH. In addition, upregulation of the inflammatory mediator COX-2 (4.3-fold, P<.05) and the cytokine osteopontin (15.5-fold, P<.01) were identified in ALDO-treated rats. Immunohistochemical analysis identified the presence of these two molecules in the media and adventitia of coronary arteries starting at day 7 of ALDO infusion. Thus, we identified vascular inflammation as a pivotal early event in the onset and progression of aldosterone/NaCl-induced myocardial disease with COX-2 and osteopontin as potential mediators of this process.

Other Financial or Material Support - Pharmacia Corp.

Key Words: aldosterone, vascular inflammation, COX-2