used as a gold standard for the diagnosis of PA. The presence of A was confirmed in all of the 6 cases upon surgery; in the remaining 16 pts with PA, the diagnosis of IHA was based on evidence of bilateral adrenal hyperplasia on CT scan with enhanced radiotracer uptake at scintigraphy with 111In-cholersterol under dexamethasone suppression. All pts underwent 2 separate ALDO/PRA determinations in the seated position, at baseline and 90 min after captopril 50 mg per os. Diuretics, ACE-inhibitors and betablockers were withdrawn at least 4 weeks before the test, and substituted with doxazosin and/or calcium antagonists if necessary. On a different occasion, an intravenous saline load (2 l 0.9% NaCl in 4 hours) was carried out; lack of ALDO suppression was defined as ALDO > 7.5 ng/dl at the end of the saline load. Formal ROC curve analysis showed that the areas under the estimated curves for ALDO/PRA before (ALDO/PRAb) and after captopril (ALDO/PRAc) were significantly different (p=0.03). A value of 35 for ALDO/PRAb had 95.4% sensitivity but only 28.4% specificity for the identification of PA; the corresponding sensitivity and specificity values for ALDO/PRAc at the same cut-off were 100% and 68.0%, respectively. Specificity for the identification of A cases at the same cut-off point (100% sensitivity) was 28.3% for ALDO/PRAb and 68.0% for ALDO/PRAc. We conclude that a single determination of ALDO/PRA 90 min after 50 mg captopril is a better screening tool for PA compared with usual ALDO/PRA determinations.

Key Words: secondary hypertension, captopril test, primary aldosteronism

P-665
CSA OR FK-506 INDUCED POST-TRANSPLANT HYPERTENSION AND OXIDATIVE STRESS IN KIDNEY TRANSPLANT PATIENTS. EFFECT OF RAMIPRIL

Post-transplant hypertension and endothelial dysfunction have been attributed to effects of calcineurin’s inhibitors, CsA and FK-506 on factors controlling vasomotor tone. We showed in CsA induced hypertensive patients a NO system up regulation and O2− and ROS overproduction which increasing NO metabolism could induce hypertension, remodeling and chronic reaction (L.Cal’o et al, Transplant. Int. 13(Suppl.1):413-8, 2000). However it is still debated whether CsA and FK-506 have different effect. We now compare CsA and FK-506 effects on markers of oxidative stress and endothelial dysfunction in 16 kidney transplanted patients with post-transplant hypertension (10 treated with CsA (CsAp) and 6 with FK-506 (FkP), using a molecular biologic approach. To this end monocytic p22phox, a NADP/NADPH system subunit, TGFβ, major profibrotic cytokine, Heme oxygenase-1 (HO-1) (induced by and protective from oxidative stress, and endothelial NO synthase (ecNOS) gene expression (RT-PCR with specific primers and densitometric analysis) were measured at baseline and after 2 month treatment with the ACE inhibitor Ramipril (5mg/day).

At baseline, in CsA and FK-506 treated patients p22phox and TGFβ mRNA production were similarly and significantly increased in comparison to normotensive healthy controls (C) (0.9±0.05 d.u. and 0.83±0.05 respectively in CsAp and 0.87±0.1 and 0.82±0.05 respectively in FkP vs 0.53±0.05 and 0.91±0.02 in C, p<0.0001 and p<0.05). CsAp and FkP ecNOS mRNA was increased vs C confirming NO system up regulation (0.92±0.09 in CsAp and 0.99±0.02 in FkP vs 0.39±0.08 in C, p<0.0001). Ramipril reduced blood pressure (from 140±119/71±7 to 129±6/85±5 in CsAp and from 138±79/2±7 to 127±10/82±6 in FkP, p<0.02). Ramipril also reduced p22phox mRNA (0.83±0.05 in CsAp, p=0.03 and 0.87±0.1 in FkP, p=0.01) and TGFβ mRNA (0.74±0.01 in CsAp, p<0.01 and 0.81±0.1 in FkP, ps<0.02) but had no effect on HO-1 (0.85±0.03, n.s. in CsAp and 0.88±0.12 n.s. in FkP), and ecNOS mRNA (0.88±0.07 in CsAp and 0.92±0.06 in FkP, n.s.).

In conclusion CsA or FK-506 treatment induces comparable oxidative stress and post-transplant hypertension in kidney transplanted patients. Ramipril normalized blood pressure and reduced the effect of oxidative stress, thereby protecting from endothelial dysfunction, fibrogenesis and chronic rejection induced by oxidative stress.

Key Words: ACE inhibitors, Post-transplant hypertension, Oxidative stress

P-666
SEVERE HYPERTENSION DUE TO BAROREFLEX FAILURE - A CASE PRESENTATION
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We present a case of severe, episodic hypertension due to baroreflex failure from carotid artery fibromuscular dysplasia. A 45 year old female presented with severe right-sided migrainous symptoms and horner’s syndrome. She had presented five years previously with similar symptoms on the left side. Carotid angiography revealed acute internal carotid dissection with extensive background fibromuscular dysplasia (FMD). Her blood pressure was elevated at 220/140mmHg and this continued to be extremely labile; systolic pressures varying between 90 and 240mmHg. A diurnal pattern emerged with systolic peaks occurring invariably in waking hours and these were often associated with flushing, headache and intense anxiety.

Renal artery stenosis secondary to FMD was considered and excluded with dynamic isoeto renography and magnetic resonance angiography. Spot plasma adrenaline, noradrenaline and dopamine concentrations were raised but 24 hour urinary excretion values were within normal limits. Intravenous clonidine (0.1mg) produced a prompt fall in heart rate and systolic blood pressure and suppressed plasma catecholamine concentrations. Autonomic nervous system testing revealed exaggerated pressor responses to exercise, cold and mental arithmetic.

We diagnosed baroreflex failure secondary to carotid fibromuscular dysplasia and bilateral carotid dissection. She was treated with labetalol which smoothed initial blood pressure profiles. In the longer term, 24 hour ambulatory blood pressure profiles have steadily improved without escalation of therapy and this may suggest a degree of baroreceptor recovery in this case.

Baroreflex failure syndrome is characterised by a failure to “buffer” sympathetic activity in response to environmental stress resulting in paroxysmal and often severe hypertension which may be accompanied by flushing, headache, diaphoresis and anxiety. It has been previously described following traumatic or iatrogenic damage to the carotid vessels. In contrast to pure autonomic failure, orthostatic hypotension is unusual. It may be differentiated from phaeochromocytoma, the main differential diagnosis, by clonidine suppression testing and measurement of 24-hour urine catecholamines. Formal autonomic testing is necessary for a firm diagnosis. Clonidine and phenoxybenzamine have been used with therapeutic success in this syndrome and we would also advocate the use of labetalol.

Key Words: fibromuscular dysplasia, carotid dissection, Baroreflex failure

P-667
CIRCADIAN RHYTHM OF BLOOD PRESSURE IN PATIENTS WITH PRIMARY ALDOSTERONISM COMPARED WITH ESSENTIAL HYPERTENSION PATIENTS
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The circadian rhythm of blood pressure in patients with essential hypertension (EH) is characterized by a night decline but wether the same pattern occurs in primary aldosteronism (PA) patients is not well known.

The circadian rhythm of blood pressure in patients with essential hypertension (EH) is characterized by a night decline but whether the same pattern occurs in primary aldosteronism (PA) patients is not well known.
We evaluated the circadian rhythm of blood pressure (BP) (using Spacelab 90700®) during 24 hours of 94 consecutive patients with suprarenal unilateral adenoma followed in our department and compared it with 50 patients with essential hypertension. The 24-hour monitoring of blood pressure was divided in waking BP (7-22h) and sleeping BP (22-7h). The two populations are matched by age, sex, serum creatinine and serum glucose (Table). In both groups we excluded patients having other organic disorders. We found that although average values of BP in PA are slightly higher comparing with EH patients (15.7 mmHg higher for systolic BP (BPs), 7.0 mmHg for diastolic BP (BPd), respectively), they have an equivalent night dip (10.0% and 10.4% in BPs, 14.3% and 12.3% in BPd, respectively)(Figure). In conclusion, our results have shown a circadian BP of dipping type in PA, with a night BP decline.

<table>
<thead>
<tr>
<th>P.A.</th>
<th>E.H.</th>
</tr>
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<tbody>
<tr>
<td>TOTAL</td>
<td>94</td>
</tr>
<tr>
<td>WOMEN %</td>
<td>50</td>
</tr>
<tr>
<td>AGE</td>
<td>50 ± 7</td>
</tr>
<tr>
<td>PLASMA GLUCOSE mg/dl</td>
<td>89 ± 16</td>
</tr>
<tr>
<td>SERUM POTASSIUM mg/dl</td>
<td>3.2 ± 0.3</td>
</tr>
<tr>
<td>SERUM CREATININE mg/dl</td>
<td>0.8 ± 0.2</td>
</tr>
<tr>
<td>PLASMA ALDOSTERONE pg/ml/24h</td>
<td>384 ± 109</td>
</tr>
<tr>
<td>PLASMA RENIN ACTIVITY ng/ml/24h</td>
<td>0.3 ± 0.1</td>
</tr>
</tbody>
</table>

Key Words: Ambulatory Monitoring, Secondary Hypertension, Primary Aldosteronism

P-668

PREVALENCE OF SECONDARY HYPERTENSION IN A SOUTHEASTERN US HYPERTENSION REFERRAL CENTER


Fifty million adults (43%) in the United States have hypertension. The prevalence of secondary hypertension is variably estimated at between 1 and 20% of adult hypertensives, depending on the clinical population assessed. Though relatively rare, the absolute number of persons affected is substantial. The diagnosis of secondary causes is critical, as these forms of hypertension are potentially curable with specific therapies based on underlying etiologies. Common etiologies of secondary hypertension are generally classified according to renal, endocrine or other miscellaneous origins. Renal parenchymal disease is often considered as most prevalent. Primary aldosteronism, parathyroid and thyroid dysfunctions are among endocrine causes. Other important causes include obstructive sleep apnea and medication or other chemical interactions. Obesity has recently been identified as a potential secondary etiology.

Recognizing the inadequacy of current prevalence data, we analyzed the rates of secondary hypertension among first time patients in a major southeastern hypertension referral clinic for one year. A preliminary evaluation of a random selection of 25% (N=40) of new patients seen between January and June 2000 revealed the following prevalence of secondary hypertension:

<table>
<thead>
<tr>
<th>Secondary Etiology</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal Parenchymal</td>
<td>10%</td>
</tr>
<tr>
<td>Endocrine</td>
<td></td>
</tr>
<tr>
<td>Aldosteronism</td>
<td>7.5%</td>
</tr>
<tr>
<td>Thyroid</td>
<td>7.5%</td>
</tr>
<tr>
<td>Other Miscellaneous</td>
<td></td>
</tr>
</tbody>
</table>

While prevalence rates are expected to be higher among referral populations, and may be unique to the particular population studied, an accurate analysis of likely rates of secondary hypertension provides important guidance for clinical evaluation of challenging cases. Implications for primary care and hypertension specialist providers will be addressed.

Key Words: Prevalence, Secondary Hypertension, Referral Center

P-669

SCREENING FOR PRIMARY ALDOSTERONE IN OLDER PATIENTS WITH HYPERTENSION


The prevalence of primary aldosteronism (PA) among hypertensive patients in referral centers is estimated to be 12%. The prevalence of PA among older subjects in referral populations has not been reported. Screening for PA with the plasma aldosterone concentration (PAC; in ng/ml) to plasma renin activity (PRA; in ng/ml/hr) has led to a dramatic increase in detection and treatment of the disorder. A positive screen for PA in our center is a PAC/PRA > 30.

Using an electronic data base we searched medical records for subjects ≥ 60 years old who had a positive PAC/PRA. Subjects were screened if they had resistant hypertension or hypokalemia. Blood pressure measures taken prior to and following initiation of a treatment regimen including a potassium-sparing diuretic are reported.

Fifty patients with a mean age of 65 had a positive screen for PA. Fifty-five percent were white; 75% female.

Key Words: Primary Aldosteronism, Elderly, Screening

P-670

NEURO-ADRENAL AND BLOOD PRESSURE RESPONSES TO SHORT-TERM HYPOTHYROIDISM


We investigated the relationships between blood pressure and neurohumoral parameters induced by short-term hypothyroidism. Twelve normotensive subjects with previous total thyroidectomy underwent ambulatory blood pressure monitoring and 24h, TSH, PRA, aldosterone, cortisol, adrenaline and noradrenaline determinations respectively six weeks after L-Thyroxine withdrawal (A) and two months after resumption of treatment (B).

A versus B means ± SD: day-time systolic BP 125.5 ± 9.69 vs 120.4 ± 10.79 mmHg (p<0.05); day-time diastolic BP 84.58 ± 7.91 vs 76.42 ± 6.84 mmHg (p<0.001); T3 0.86 ± 0.54 vs 2.96 ± 0.80 pg/ml (p<0.001); T4 2.54 ± 1.54 vs 16.28 ± 4.34 pg/ml (p<0.0001); TSH 0.08 ± 0.27 vs