Highlights of the Issue

- HLA incompatible kidney transplantation
- Cultural competency in the diabetes care pathway
- Aortic calcification as surrogate for valvular calcification and carotid plaque

Official Publication of the Indian Society of Nephrology
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Prevalence of diabetic nephropathy in an underserved rural community

Sir,

There is a paucity of data on the incidence and prevalence of chronic kidney disease (CKD) in India which is hampering the development of public policy and allocation of resources for the treatment of renal replacement therapy.

We report the results of our study that carried out a cross-sectional population survey in a village (Karakhadi, Gujarat) comprising 1889 adult persons of which 1681 participated (89% response rate); most of them are below the poverty line of less than US$ 2/day. Vital data was obtained on all participants, however, laboratory data was limited to persons who were known to be diabetic (n = 118). We collected samples for CBC, FBS, A1c, creatinine, urine albumin, and serum lipids. We also calculated the GFR by MDRD formula.

The crude prevalence of diabetes was 7.2% (all type 2); diagnosed hypertension in 4.3% and undiagnosed hypertension was 21.8%. The point prevalence of diabetic nephropathy defined as micro-albumin >30 was 13.6% (14 patients in CKD 1, and one each in CKD stages two and three respectively). Subjects who had micro-albumin <30 (86.4%) were 102. Further, in these diabetics, total cholesterol >200 mg/dl was found in 14%, triglyceride level >150 mg/dl was found in 25%, creatinine >1.1 mg/dl was found in 6%, and Hb A1c >6.5% was found in 45%. Patients with A1c >6.5% had statistically higher levels of micro-albumin (P = 0.001), total cholesterol (P = 0.053), triglycerides (P = 0.050), and LDL cholesterol (P = 0.045) suggesting metabolic complications of diabetes [Table 1].

Modi and Jha[1] found that 346 new end stage renal disease (ESRD) patients were diagnosed during the study period from a population base of approximately 0.5 million; diabetic nephropathy was the commonest (44%) cause of ESRD. There are significant differences in the study reported by Modi and Jha. [1,2] Their population included the large urban area of Bhopal with a relatively sophisticated level of tertiary medical services. Our cohort is a rural part of India with the majority of people below the poverty level without primary or tertiary medical services. Furthermore, they report on the entire population of approximately 0.5 million, whereas our study is limited to patients with known type 2 diabetes. Nonetheless, our findings are novel as we found that most of the patients with diabetic nephropathy were CKD 1, suggesting a relatively benign disease process.

It is noteworthy that despite the lack of manifestations of moderate or severe proteinuria, diabetics with A1c >6.5 had metabolic complications of diabetes. In this regards, our findings are in line with that of Raman et al.[3] group which carried out a population-based cross-sectional survey on 1414 patients having type 2 diabetes mellitus in South India. They found an incidence of metabolic syndrome to be 73.3%. In subjects with diabetes mellitus, without and with metabolic syndrome, the prevalence of nephropathy was 20.5% and 18.0% respectively.

These patients might benefit from ACE inhibitors, aggressive control of diabetes, and hypertension. We speculate that the low prevalence of diabetic nephropathy could be vegetarian diet; however, genetics or environmental factors may also be implicated. Another explanation could be that patients in CKD 5 are simply dying due to lack of renal replacement therapy and are not accounted for in this cohort. Longitudinal studies with larger sample size are indicated to study the incidence and prevalence of CKD in emerging countries such as India where there are vast disparities in access to health services between urban and rural areas.

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Table 1: Study of variables, means, and SD of the cohort

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>50.8</td>
<td>14.8</td>
</tr>
<tr>
<td>HbA1c</td>
<td>7.0</td>
<td>2.1</td>
</tr>
<tr>
<td>TChol</td>
<td>166.3</td>
<td>33.8</td>
</tr>
<tr>
<td>Trigly</td>
<td>126.6</td>
<td>80.4</td>
</tr>
<tr>
<td>HDL</td>
<td>44.9</td>
<td>10.6</td>
</tr>
<tr>
<td>LDL</td>
<td>96.8</td>
<td>28.7</td>
</tr>
<tr>
<td>VLDL</td>
<td>23.2</td>
<td>11.2</td>
</tr>
<tr>
<td>FBS</td>
<td>118.8</td>
<td>61.2</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.73</td>
<td>0.25</td>
</tr>
<tr>
<td>Micro-albumin</td>
<td>21.6</td>
<td>52.6</td>
</tr>
</tbody>
</table>
Plasma exchange for steroid unresponsive Devic’s disease

Sir,

A 15-year-old girl presented with weakness of both lower limbs, sensory loss below costal margin, and bladder disturbances for 3 days; loss of vision in the left eye for 3 months with past history of similar illness (3 months back) that showed partial improvement in motor power, sensory, and autonomic disturbances with steroid therapy without any improvement in vision. Physical examination showed optic atrophy in the left eye. Muscle power in lower limbs was 1/5 in proximal and distal groups. Pain sensation below D7 level was diminished. There was an exaggerated knee-jerk reaction and extensor plantar responses. Magnetic resonance imaging revealed demyelinating changes in right centrum semi-ovale of the brain and demyelination in spinal cord from C 4 level to D 9 level; visual evoked potential from left eye showed prolonged P100 latency with reduced amplitude. She was again treated with I.V. steroids for 5 days followed by oral steroids. As there was no improvement after 11 days of oral steroids, patient was treated with plasma exchange.

She received a total of five sessions of plasma exchange on alternate days. The amount of plasma removed was calculated based on the formula: 0.065 × body weight (1% hematocrit) and it was about 1.7 l, which was replaced with four packs of fresh frozen plasma (each containing 180‑220 ml of plasma), 100 ml of 20% albumin, and 1 l of normal saline. After third session, muscle power in the lower limbs improved to 3/5 in the proximal group of muscles and 2/5 in the distal group of muscles. Muscle power remained unchanged till discharge, i.e., after 10 days of completion of fifth session of plasma exchange, and the patient was able to walk with support. She maintains her muscle power at 10 months follow up.

Devic’s neuromyelitis optica (NMO) is an inflammatory disease of the central nervous system characterized by severe attacks of optic neuritis and myelitis. [1,2] The specific antibody called NMO-IgG was found in more than 70% of these patients. This antibody is targeted against aquaporin-4 (AQP-4) water channel,[2] widely expressed in the optic nerves, the spinal cord, and the peri-ventricular regions. Traditionally, the main stay of the treatment of the acute attack or the index event has been high-dose intravenous steroid. Plasma exchange found a significant role in patients who did not respond to corticosteroids in Devic’s disease in the recent literature.[3‑5] The proposed mechanism of benefit being removal of the antibodies and immune complexes and the reported recovery is to the extent of 40%.[4]

Plasma exchange has been shown to reduce IgG, IgM, and total complement levels by 63.4%, 68.9%, and 57.1%, respectively, after one exchange and 80.1%, 79.5%, and 59.7%, respectively, after five exchanges.[2] Here in, we report the importance of plasma exchange in the management of a patient of steroid unresponsive Devic’s disease.

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References