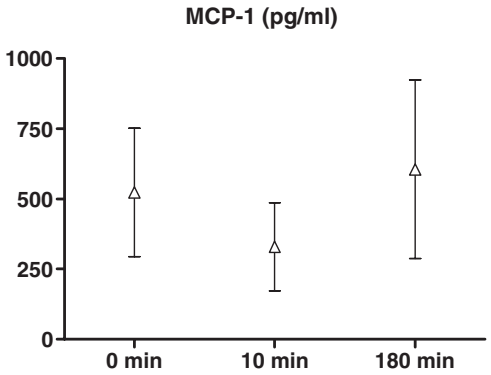

Fig. 1. MCP-1 profile during haemodialysis (before, after 10 min and 180 min) with enoxaparin anticoagulation.

concentration 8 h later (in that study heparin was used although the authors did not elaborate on that fact) [7].

The phenomenon that enoxaparin might reduce the circulating levels of MCP-1 may be very interesting and potentially useful. It suggests an anti-atherosclerotic effect of this LMWH and yet another use of it.

Acknowledgement. This work was funded by Medical University of Białystok, Poland (grant number 3-54723).

Conflict of interest statement. None declared.

Department of Nephrology and Transplantation with Dialysis Unit
Medical University of Białystok
Żurawia 14, 15-540 Białystok
Poland
E-mail: alunieczka1@wp.pl

Alicja Rydzewska-Rosołowska
Jacek Borawski
Michał Myśliwiec

1. Dragomir E, Simionescu M. Monocyte chemoattractant protein-1—a major contributor to the inflammatory process associated with diabetes. *Arch Physiol Biochem* 2006; 112: 239–244
2. Xia Y, Frangogiannis NG. MCP-1/CCL2 as a therapeutic target in myocardial infarction and ischemic cardiomyopathy. *Inflamm Allergy Drug Targets* 2007; 6: 101–107
3. Tesch GH. MCP-1/CCL2: a new diagnostic marker and therapeutic target for progressive renal injury in diabetic nephropathy. *Am J Physiol Renal Physiol* 2008; 294: F697–701
4. Bick RL, Frenkel EP, Walenga J *et al*. Unfractionated heparin, low molecular weight heparins, and pentasaccharide: basic mechanism of actions, pharmacology, and clinical use. *Hematol Oncol Clin North Am* 2005; 19: 1–51, v
5. Borawski J, Naumnik B, Mysliwiec M. Activation of hepatocyte growth factor/activin A/follistatin system during hemodialysis: role of heparin. *Kidney Int* 2003; 64: 2229–2237
6. Akahoshi T, Kobayashi N, Hosaka S *et al*. *In-vivo* induction of monocyte chemotactic and activating factor in patients with chronic renal failure. *Nephrol Dial Transplant* 1995; 10: 2244–2249
7. Jacobson SH, Thylen P, Lundahl J. Three monocyte-related determinants of atherosclerosis in haemodialysis. *Nephrol Dial Transplant* 2000; 15: 1414–1419
8. Pawlak K, Pawlak D, Mysliwiec M. Possible new role of monocyte chemoattractant protein-1 in hemodialysis patients with cardiovascular disease. *Am J Nephrol* 2004; 24: 635–640
9. Pawlak K, Pawlak D, Mysliwiec M. Impaired renal function and duration of dialysis therapy are associated with oxidative stress and proatherogenic cytokine levels in patients with end-stage renal disease. *Clin Biochem* 2007; 40: 81–85
10. Douglas MS, Ali S, Rix DA *et al*. Endothelial production of MCP-1: modulation by heparin and consequences for mononuclear cell activation. *Immunology* 1997; 92: 512–518

doi: 10.1093/ndtplus/sfp060

Advance Access publication 23 June 2009

Brain renin–angiotensin system: Is it important in dialysis patients?

Sir,

Excessive inter dialytic weight gain (IDWG) is an important clinical problem and portends a poor outcome in haemodialysis patients [1]. Treatment is based on the restriction of salt and water intake. However, the compliance is usually poor and one-third of patients treated with maintenance haemodialysis are chronically overhydrated.

The cardiovascular and haemodynamic effects of the peripheral renin–angiotensin system (RAS) are well documented. Conversely, the current knowledge regarding the effects of the brain RAS on blood pressure and thirst is mainly based on experimental studies. It was shown in experimental models that the stimulation of centrally located angiotensin 1 receptors (AT1-R) causes a rise in blood pressure, a release of vasopressin [2], thirst and thereby an increase in salt [3] and water [4] intake. In this respect, the effects of the RAS system on the brain could be important in haemodialysed patients. The brain RAS system upregulation is likely to increase the patients' drinking behaviour, along with an increase in salt and water intake causing excessive IDWG. Therefore, the brain RAS represents a potential target for RAS inhibitors to counteract these centrally related effects.

Dipsogenic activity is mediated only by AT-1 receptors. Brain tissue, with the exception of the circumventricular organs, is separated from the circulation by the blood–brain barrier. AT-1 receptors are widespread in the brain, and their stimulation in circumventricular structures causes drinking and pressor responses. In addition to circumventricular organs, AT1 receptors are also located in the inside compartment of the blood–brain barrier, and the activation of these AT1 receptors also causes drinking and pressor responses [5].

One is allowed to speculate that inhibiting centrally related effects of brain RAS with lipophilic angiotensin-converting enzyme and/or AT1-R blockers exerts a favourable effect on the IDWG. In a prospective, self-controlled and interventional study that included 30 anuric haemodialysed patients, we compared the IDWG in a period of 3 consecutive months without and with administration of telmisartan. We showed that the IDWG significantly decreased following telmisartan at a dosage of 40 mg/day [6]. These results suggest that the treatment of hypertension in haemodialysed patients with AT1-R inhibitors does not only aim at reducing the blood pressure—a short-term effect. In the long run, it also diminishes thirst, salt and

water intake and hence the extracellular fluid volume and tissue sodium concentrations that are essentially responsible for high blood pressure and left ventricular hypertrophy in these patients.

Conflict of interest statement. None declared.

[1]Division of Nephrology Murat Demir[1]
Department of Internal Medicine Ali Kutlucan[2]
[2]Department of Internal Medicine Mehmet Tugrul
[3]Department of Cardiology Sezer[1]
Suleyman Demirel University Yasin Turker[3]
School of Medicine, Isparta, Turkey
E-mail: drmuratdemir@yahoo.com

1. Saran R, Bragg-Gresham JL, Rayner HC *et al*. Nonadherence in hemodialysis: associations with mortality, hospitalization, and practice patterns in the DOPPS. *Kidney Int* 2003; 64: 254–262
2. Ferguson AV, Kasting NW. Electrical stimulation in subfornical organ increases plasma vasopressin concentrations in the conscious rat. *Amer J Physiol* 1986; 251: 425–428
3. Smith PM, Beninger RJ, Ferguson AV. Subfornical organ stimulation elicits drinking. *Brain Res Bull* 1995; 38: 209–213
4. Thunhorst RL, Fitts DA. Peripheral angiotensin causes salt appetite in rats. *Amer J Physiol* 1994; 267: 171–177
5. Fitzsimons JT. Angiotensin, thirst, and sodium appetite. *Physiol Rev* 1998; 78: 583–686
6. Demir M, Kutlucan A, Turker Y *et al*. Does the inhibition of rennin–angiotensin system effect on inter-dialytic weight gain in haemodialysis patients? *Nephrol Dial Transplant* 2007; 22(Suppl): 99 (FP239)

doi: 10.1093/ndtplus/sfp069

Advance Access publication 17 June 2009

The incidence of biopsy-proven glomerulonephritis in Cairo University, Egypt: a 5-year study

Sir,

The incidence of biopsy-proven glomerulonephritis (GN) varies in different geographical areas and is affected by socio-economic conditions, race, differences in genetic susceptibility and environmental exposure. Recent studies suggested a changing pattern of incidence of GN in different parts of the world [1,2]. For instance, the incidence of end-stage renal disease (ESRD) as a result of focal segmental glomerulosclerosis (FSGS) has increased 11-fold in the past two decades in a recent study [2].

Our study aimed to obtain a comprehensive review of the incidence of biopsy-proven glomerulonephritis in Cairo University, Egypt, over the last 5 years. We analysed the clinical and pathological data of all renal biopsy samples that were obtained during the period from July 2003 to 2008. Age, gender, indication of renal biopsy and the pathological findings were recorded for analysis.

A total of 924 renal biopsy samples were referred for pathological assessment during the period of the study. The monthly incidence of biopsy-proven GN was 15.4 (range 13–19). Proliferative GN was reported in 497 cases (53.78%) and non-proliferative GN was reported in 427 cases (46.22%). Lupus nephritis was reported in 264 cases (28.57%). The female/male ratio was 221/41, 70% of lupus patients aged 18–45 years and 85% had renal impairment (mean serum creatinine was 3.21 $\pm$ 4.09 mg/dl). The common glomerular pathologies in patients with lupus nephritis were the proliferative classes II–IV (28.78, 30.30, 27.65%, respectively).

Morphologically, FSGS was the most frequent cause of GN (21.21%) followed by mesangial proliferative GN (18.93%), diffuse proliferative GN (13.96%), focal proliferative GN (12.77%) and membranous GN (10.93%) (Table 1). In females, mesangial proliferative, focal proliferative GN and diffuse proliferative GN were the predominant pathological findings, and in males FSGS, diffuse proliferative GN and mesangial proliferative GN were predominant. In those aged <18, mesangial proliferative GN, minimal change disease and FSGS were more prevalent compared to adults. Figure 1 shows the frequency of GN in patients with renal insufficiency.

Table 1. Incidence of biopsy proven GNs in the study group

Lupus nephritis (LN-GN): 264 Cases (28.57%)
Focal segmental glomerulosclerosis (FSGS): 185 Cases (20.02%)
Mesangial proliferative GN: 97 cases (10.49%)
Minimal change disease (MCD): 79 Cases (8.55%)
Membranoproliferative GN (MPGN): 68 cases (7.36%)
Membranous Nephropathy (MGN): 65 cases (7.03%)
Amyloid: 51 cases (5.52%)
Diffuse Proliferative GN: 48 cases (5.20%)
Focal Proliferative GN: 34 cases (3.68%)
Diabetic Glomerulosclerosis: 2 cases (0.22%)

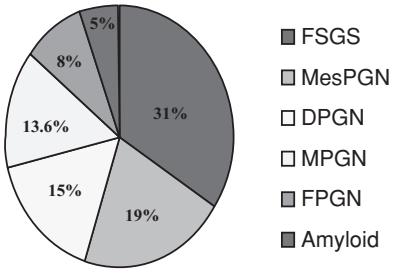

Fig. 1. Incidence of GNs in patients with renal insufficiency [352 cases (38.09%)]. We conclude that FSGS and proliferative GN (SLE and others) were the predominant forms of GN in the population of the study. Compared to other Arab countries, FSGS was the predominant GN in two studies coming from Saudi Arabia [3] and Kuwait [4]. MGN was the predominant GN in two reports from United Arab Emirates and Iran [5,6]. Our results could be explained by high incidence of lupus nephritis among the study subjects as well as the relatively young age of the study group. Further multicentre studies with larger sample size from different parts of the country would give more accurate information on the incidence and frequency of GNs in Egypt.

Conflict of interest statement. None declared.

Department of Internal Medicine Salwa Ibrahim
Cairo University, Egypt Ahmed Fayed
E-mail: Salwaibrahim@hotmail.com