The Effect of Phlebotomy on Renal Function and Proteinuria in a Patient with Congenital Cyanotic Heart Disease

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Sir,

With interest we read the paper of Wilcox et al. [1] on renal function in patients with cor pulmonale and secondary polycythaemia. Effective renal plasma flow (ERPF) was found to be reduced and filtration fraction (FF) to be increased. A few days after haematocrit (Hct) reduction, FF fell in 5 out of 7 patients. Parallel with the decrease in FF, a fall in body weight was seen. The authors suggest that, at least temporarily, the increased postglomerular capillary resistance – which is present in these patients [2] – can be corrected by Hct reduction.

We now show another effect of phlebotomy in a patient with congenital cyanotic heart disease. It is known that glomerular lesions can occur in these patients [3, 4]. Spear and Vitsky [3, 4] described glomerular enlargement, congestion and capillary dilatation. There was mesangial hyper-cellularity and focal glomerular sclerosis on light microscopy. On electron microscopy, diffuse thickening of the glomerular basement membrane was found [5]. Besides the above-mentioned renal functional abnormalities also proteinuria is a common finding in these patients [4,5], probably as a consequence of the glomerular functional and morphological abnormalities. We recently observed a 36-year-old man, known with tetralogy of Fallot, who underwent a Blalock operation at the age of 6. He was admitted to our hospital because of nephrotic syndrome and hypertension. Hct was 67%. Phlebotomy was carried out and he was instituted on digoxin. Body weight decreased and oedema disappeared but proteinuria persisted. From this period no accurate renal function studies are available. The patient was transmitted to the department of nephrology. At this moment no clinical signs of fluid overload were present and blood pressure was 140/95 mm Hg. Further investigations did not reveal any other abnormality known to cause nephrotic syndrome. Hct was 55%, proteinuria amounted to 4–5 g/

Table I. The effects of phlebotomy in our patient

During After 8 weeks

observation phlebotomy3, after
discharge

Body weight, kg	56.7	55.3	59
Haematocrit, %	55	44	59
Serum creatinine, $\mu\text{mol/l}$	86	77	91
Serum urea, mmol/l	7.7	5.0	5.3
GFR, ml/min	73	101	78
ERPF, ml/min	132	223	155

ERBF, ml/min	293	398	378
FF	0.55	0.45	0.50
mmol/24h	8.7	8.5	8.4
Albumin excretion ¹ *, g/24 h	4.5	2.2	4.7
Fractional albumin excretion ¹³ , Caib/CCreat			
×10 ²	0.15	0.06	0.13

a During 4 days 250 ml of blood was removed every day. Plasma was reinfused. Measurements were performed the day after the last phlebotomy.

b The mean of 3 days is given.

24 h, and serum albumin was 31 g/l (table I). Glomerular filtration rate (GFR) and ERPF [6] were decreased with a striking increase in FF (normal value 0.25 ± 0.02). A kidney biopsy showed glomerular enlargement, hypercellularity and sclerosis in the glomerular mesangium, and hyalinization of afferent and efferent arterioles (fig. 1a). No tubular abnormalities were found. Immunofluorescence showed weak linear deposition of IgA and IgG and slight granular IgM and complement deposition. Electron microscopy revealed deposition of electron dense material (possibly red cell fragments, globulins and clotting factors), along the endothelial aspect of the glomerular capillary wall (fig. 1b).

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Fig.1.a Expanded glomerulus with mesangial hypercellularity and sclerosis. Hilar arteriole shows hyalinization. PAS stark. $\times 140$. b Electron micrograph of glomerular capillary wall shows accumulation of dense material (arrow heads) along the endothelial surface. $\times 11,000$.

The renal functional and morphological abnormalities in our patient are compatible with those cited above [1–5]. It could be argued that the renal morphological abnormalities are secondary to the polycythaemia, since high blood viscosity can induce increased postglomerular resistance and thereby glomerular enlargement and capillary dilatation [2]. This could then initiate the lesion of glomerular sclerosis [7]. Hct reduction in our patient was accompanied by a rise in GFR and ERPF together with a fall in FF and proteinuria. This observation, therefore, underlines the importance of a high transcapillary hydraulic pressure gradient in the development of glomerular proteinuria (and sclerosis) in patients with congenital cyanotic heart disease.

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