

PLASMAPHERESIS INDUCED RECOVERY FROM RENAL FAILURE IN MESANGIOCAPILLARY GLOMERULONEPHRITIS OF ACUTE ONSET

J Montoliu, E Bergadá, A Botey, A Torras, A Darnell, P Arrizabalaga, J López-Pedret, L Revert

Hospital Clinic, Barcelona, Spain

Summary

Plasmapheresis (PE) was used in the treatment of three patients with biopsy proven mesangiocapillary glomerulonephritis (MCGN) of acute onset and renal failure. The percent of glomeruli with extracapillary proliferation varied from 40 to 90 per cent.

Following PE, there were reductions in serum creatinine of 66 per cent, 61 per cent and 82 per cent in patients, one, two and three respectively. Plasmapheresis proved effective in a second episode of renal failure in patient three. After the completion of PE, renal function has remained stable in all patients for periods ranging from two to 12 months. However, haematuria and heavy proteinuria persist in every case. PE appears to facilitate recovery from renal failure in MCGN of acute onset, possibly by removing circulating mediators of acute inflammatory glomerular damage.

Introduction

A few cases of mesangiocapillary glomerulonephritis (MCGN) have been treated by plasmapheresis (PE), usually after several years of nephrotic syndrome and when renal function had already deteriorated significantly [1-3]. Overall, it has been difficult to substantiate any benefit derived. However, MCGN can also present as an acute nephritic syndrome with renal failure [4] and experience with the use of PE in the acute forms of the disease has been limited. Following our previously reported initial success with PE treatment in a case of dense deposit disease [5], we have seen two additional patients with MCGN of acute onset who apparently benefited from the use of PE. This article describes our therapeutic results in these three patients.

Patients and methods

The diagnosis of MCGN was established by renal biopsy in three patients in whom the known duration of renal disease was less than three months. Histological

diagnosis was based upon generally accepted criteria. Table I summarises the main clinical, laboratory and histological findings. Systemic lupus erythematosus was excluded in all patients.

Plasmapheresis 4L exchanges was performed through a subclavian catheter, using an IBM continuous flow separator, and replacing with plasma protein fraction (PPF). Prednisone 1mg/kg/day and cyclophosphamide 2.5mg/kg/day were also given for two months and thereafter prednisone was progressively tapered. Patient one underwent 10 PE over a period of 16 days, patient two had 12 PE in 24 days and patient three received 16 PE over a period of 42 days.

TABLE I. Summary of clinical, laboratory and histological data

	Patient 1	Patient 2	Patient 3
Age, sex	29, F	51, F	40, M
Known duration of renal disease	1 month	3 months	2 months
Major presenting syndrome	Acute nephritic syndrome	Nephrotic syndrome	ARF Hypertension
Peak serum creatinine	4.4mg/dl	5.6mg/dl	13.6mg/dl
Hypocomplementaemia	Yes (alternate pathway)	Yes (classic pathway)	No
C ₃ NeF	Positive	Negative	Negative
Proteinuria	9g/24 hr	7g/24 hr	18.8g/24 hr
Associated illness	Partial lipodystrophy	None	None
Renal biopsy	MCGN type II	MCGN type I	MCGN type I
(Percent of glomeruli with extra capillary proliferation)	(90%)	(40%)	(60%)

M = male; F = female; ARF = acute renal failure; C₃ NeF = C₃ nephritic factor; MCGN = mesangiocapillary glomerulonephritis

Results

Following PE, serum creatinine fell from a peak of 4.4 to 1.5mg/100ml in patient one (a 66 per cent reduction) from 5.6 to 2.2mg/100ml in patient two (a 61 per cent reduction) and from 13.6 to 2.4mg/100ml in patient three (an 82 per cent reduction). Figure 1 summarises these changes and Figure 2 depicts the duration of PE in each patient and the course of serum creatinine in relation to PE.

Patient three required dialysis temporarily but his lowest serum creatinine was obtained when he had been off dialysis for two weeks (Figure 2).

Patients one and two have been followed for 12 and 10 months and their present serum creatinines are 1.2 and 1.5mg/100ml respectively. The serum creatinine in patient three remained less than 3mg/100ml for six months. He then relapsed and serum creatinine increased abruptly to 5.6mg/100ml. He then underwent a second series of 12 PE over a period of 30 days.

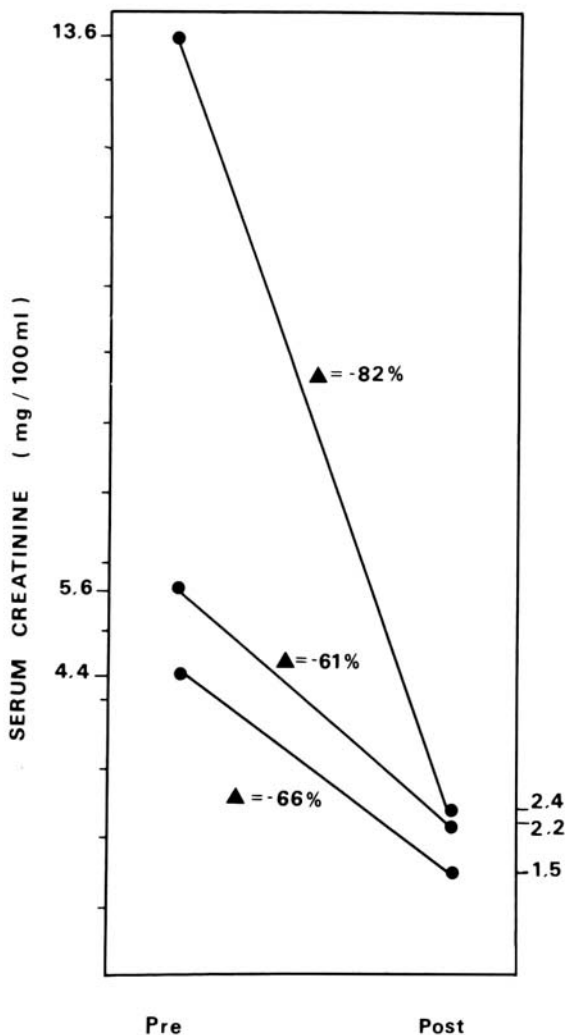
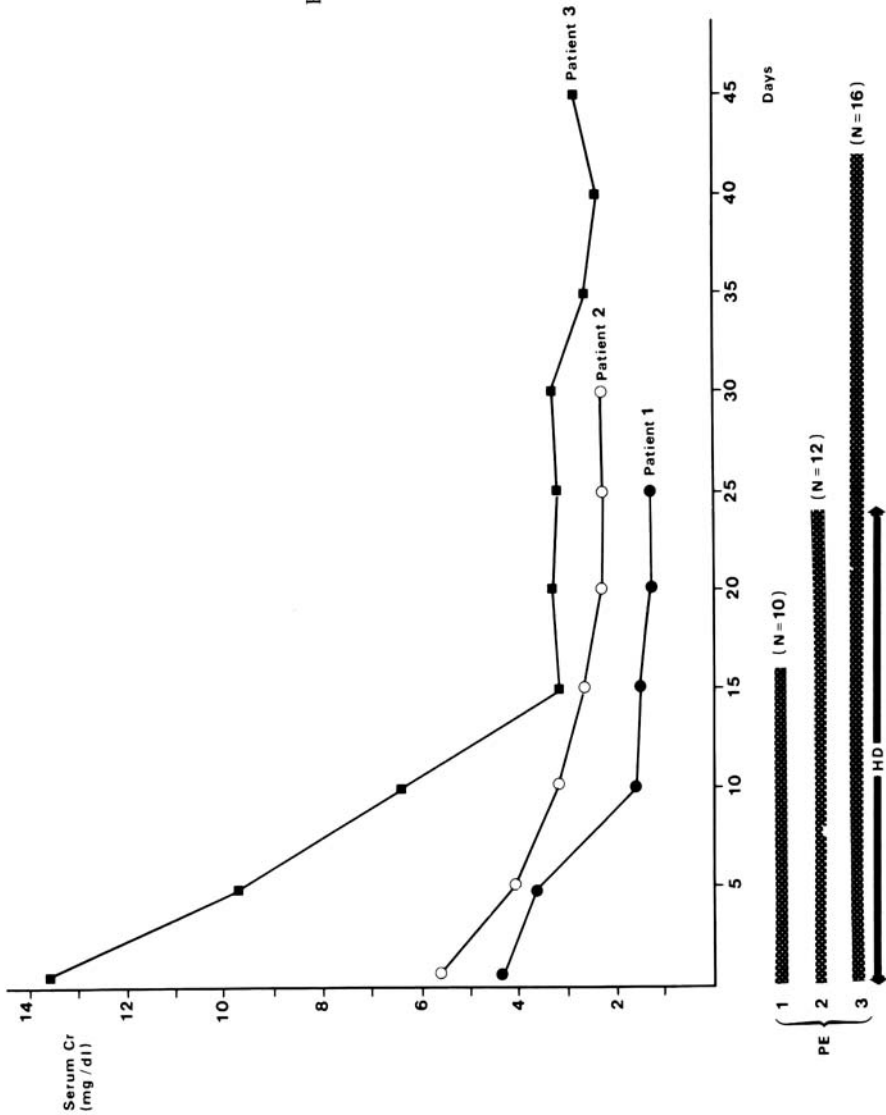


Figure 1. Summary of changes in serum creatinine following plasmapheresis. ▲: percent reduction in serum creatinine

Prednisone and cyclophosphamide were also added. Again, serum creatinine decreased rapidly to 2.5mg/100ml (Figure 3) and has been at this value for two months.

All patients continue with heavy proteinuria and microscopic haematuria. Patients one and two are asymptomatic, whereas patient three has severe hypertension and steroid-induced hyperglycaemia.

No complications related to the plasmapheresis procedure were encountered in any patient.



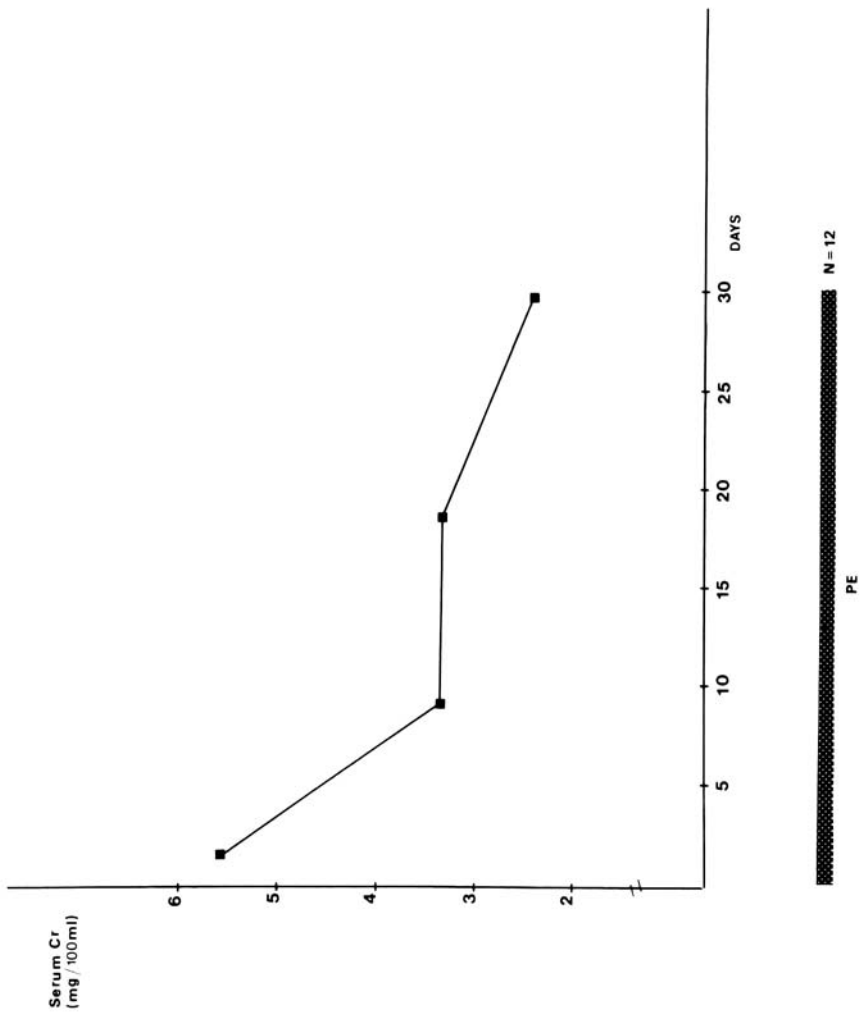


Figure 3. Second episode of renal failure in patient three and response to PE. Legend as in Figure 2

Discussion

Our results clearly demonstrate that PE may be useful in acute forms of MCGN accompanied by extracapillary proliferation. The possibility of spontaneous improvement seems unlikely in view of the close temporal relationship between use of PE and renal functional improvement. Moreover, PE proved equally effective when patient three developed his second bout of severe renal failure.

The mechanism by which PE improved renal function is uncertain. One possibility is that it reduced acute inflammatory glomerular damage by removing complement, clotting factors or other mediators of tissue injury.

Our experience, and that of others, is that PE is effective in the treatment of idiopathic rapidly progressive glomerulonephritis [3,6]. Since all our patients had significant extracapillary proliferation it would be reasonable to postulate that PE acted on this component of the disease. This cannot be proven in the absence of follow-up renal biopsies.

Circulating immune complexes were not routinely measured in our cases because we have found them of limited clinical value in the management of acute forms of glomerular disease.

However, PE does not appear to affect the basic underlying pathogenesis of the disease since all patients continued to have haematuria and heavy proteinuria in spite of the improvement in serum creatinine. Furthermore, patient three suffered a further episode of renal failure after completion of the first series of PE.

Thus PE appears to facilitate recovery from renal failure in MCGN of acute onset, possibly by removing circulating mediators of acute inflammatory glomerular damage. Further trials with PE in this situation seem warranted.

Acknowledgments

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Address for correspondence: J Montoliu, Hospital Clinic, Barcelona, Spain