Focal segmental glomerulosclerosis in adults related to low birth weight – case series

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Aim

Lower nephron endowment due to low-birth weight (LBW), prematurity (PM) or perinatal complications like sepsis or asphyxia is well-known risk factor for development of chronic kidney disease (CKD) and arterial hypertension (AH) in later life.[1] We aimed to investigate clinical and pathohistological characteristics of adult patients with focal segmental glomerulosclerosis (FSGS) presumed to be related to LBW or PM.

Methods

Patients with diagnosis consistent with secondary FSGS were assessed for underlying condition related to FSGS. Patients were recruited from Registry of kidney biopsies (Department of Nephrology and Dialysis, Dubrava University Hospital, Zagreb) in the period from 2010 to 2021. The birth weight ≤ 2500 and ≤ 1500 g was considered as LBW and very LBV, respectively. PM was defined as delivery before 37th gestational week. Clinical, laboratory and pathohistological characteristics of patients were analyzed and results are shown descriptively.

Results

Of 180 patients with histologic findings consistent with secondary FSGS, we identified 5 patients with LBW and PM, two patients with very LBW (P1 and P7) and one patient (P3) born in 32nd week of gestation (birth weight 2800 g). Table 1 summarizes relevant clinical, laboratory and histopathological data.

All analyzed patients presented with arterial hypertension, hyperlipidemia and isolated proteinuria (in absence of other elements of nephrotic syndrome) without erythrocyturia. Seven patients had perihilar variant of FSGS (Figure 1) and one (P1) had classic (NOS) variant. All patients had at least nodular arteriolar hyalinosis and three of them (P1, P6, P7) hyalinosis in full arteriolar circumference. Majority of patients were overweight or obese. There were no immune deposits on electron microscopy, glomerular basement membrane had normal thickness and podocyte foot processes were almost preserved.

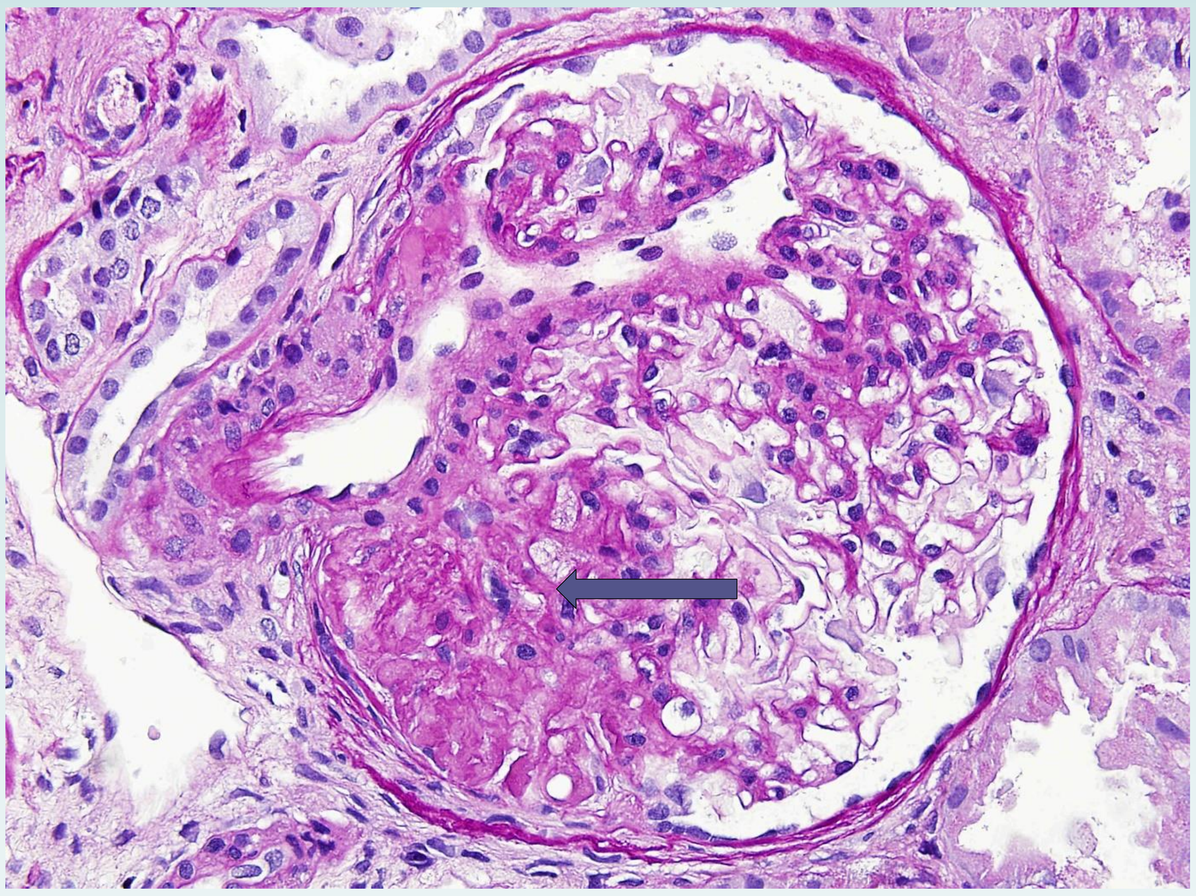

Figure 1 Kidney biopsy – light microscopy. Perihilar segmental sclerosis (arrow) in enlarged glomerulus (P2). PAS stain, original magnification x400.

Table 1 Relevant clinical and histological data of included patients.

Patient/sex	P1/M	P2/M	P3/F	P4/M	P5/M	P6/M	P7/F	P8/F
Age at Bx (years)	24.3	27.4	43.5	32.1	24.9	39.7	20.7	24
Age of first symptoms	18	26	40	32	21	39	16	17
BW (g)	1500	2500	2800	2200	2000	2000	900*	2400*
GW	n/a	n/a	32nd	n/a	36th	n/a	27th	n/a
BMI (kg/m^2)	31.8	28.7	32.3	27.5	32.7	26.5	22.0	26.4
24h proteinuria (g/day)	3.78	1.03	1.60	1.90	4.10	5.50	0.91	1.0
eGFR (ml/min/1.73m^2)	84	58	102	82	71	50	109	109
HU (yes/no)	no	yes	yes	yes	yes	yes	no	no
No of glomeruli	14	15	35	20	27	25	11	20
GSG (%)	7	8	6	0	30	80	0	20
SSG (%)	14	16	6	15	19	8	18	15
IF (glomeruli)	IgM1+	IgM1+	IgM+/-	neg	neg	IgM+/-	IgM1+	IgM+/-
IFTA (%)	15	5	3	5	15	35	0	15

** Additional perinatal asphyxia; M, male; F, female; Bx, kidney biopsy; BW, birth weight; GW, gestational weeks; BMI, body mass index; eGFR, estimated glomerular filtration rate; HU, hyperuricemia; GSG, globally sclerosed glomeruli; SSG, segmentally sclerosed glomeruli; IF, immunofluorescence; IFTA, interstitial fibrosis and tubular atrophy.*

Conclusion

FSGS related to LBW or PM usually presents with arterial hypertension and isolated proteinuria in absence of erythrocyturia. Perihilar variant of FSGS is the most common histological finding underlying kidney disease related to LBW or PM.

References

[1]Kanda T, et al. Hypertens Res. 2020;43:859-68.