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Original Article

Transient Renal Medullary Hyperechogenicity in the First Days of Life: Clinical and Ultrasonographic Follow Up

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Abstract ***Background:*** Renal medullary hyperechogenicity (RMH) has been reported in newborns with severe perinatal kidney damage, renal malformation, nephrocalcinosis, and in newborns with transient acute kidney injury. This study aims to determine clinical and laboratory features and potential risk factors of transient neonatal RMH. ***Methods:*** Twenty patients with RMH were included in the study. The demographic characteristics, biochemical parameters, blood pressure and urine output levels were recorded in all patients. Renal function parameters were calculated. ***Results:*** RMH was observed ultrasonographically (US) in all cases. Detailed nephrological work-up revealed a transient increase in blood urea nitrogen and creatinine levels in 1/3 and 2/3 of the patients, which returned to normal limits within 72-96 hours. A transient proteinuria had also subsided within two weeks. The control US examination were performed within the first week of life, and these revealed normal sized kidneys without increased echogenicity or dilated collecting system. ***Discussions:*** Although there was a transient increase in serum creatinine and urinary protein excretion in newborns with RMH, all patients became completely normal biochemically and sonographically within a few days. Renal medullary hyperechogenicity may be observed as a transient condition that can be subsided without any specific treatment under follow-up.

Key words *Hyperechogenicity; Kidney; Medullary; Newborn*

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Introduction

In the neonatal period, the renal cortex appears hyperechoic on ultrasound (US) examination, whereas the medullary pyramids are prominent and hypoechoic. Hyperechogenic appearance in the medulla so-called renal medullary hyperechogenicity (RMH) has been observed in some instances, including newborns with severe perinatal kidney damage, renal malformation, nephrocalcinosis, and those with transient renal failure.[1-4] It may also be transient in patients who have oligo/anuria and undergoes spontaneous recovery without any intervention.[5]

This paper has reported our experience on the spontaneous resolution of RMH in newborns who had mild renal impairment without accompanying fluid/electrolyte imbalance. We have reviewed the medical records of twenty cases with transient medullary hyperechogenicity in the renal US in the early neonatal period and evaluate potential risk factors for RMH.

Materials and Methods

This study was conducted with the approval of the Local Institutional Review Board (No: 25901600-604.01.01-16). Neonatal transient RMH was observed in renal US examination performed for various reasons during the first few days of life in 20 term and preterm neonates born at Gazi University Medical Faculty Neonatal Intensive Care Unit (NICU) between January 2016 - December 2020. Patients were followed up in the outpatient clinic or in the NICU.

Demographic characteristics, birth weight and week, mode of delivery, prenatal history, APGAR score, and reason for hospitalisation were evaluated. Blood pressure (BP) and urinary output were recorded in all patients. The oscillometric method is used to determine BP, and those between 3% and 97% percentile were considered normal according to the gestational weeks.[6] Serum levels of blood urea nitrogen (BUN) (N: 3-22 mg/dl), creatinine (Cr) (N: 0.3-0.9 mg/dl), uric acid (upper limit of normal (ULN): 12 mg/dl), alkaline phosphatase (ALP) (ULN: 281 U/L), calcium (Ca) (ULN: 10.6 mg/dl), phosphorus (P) (ULN: 10.4 mg/dl) and magnesium (Mg) (ULN: 3.8 mg/dl) were evaluated by using Canadian paediatric reference intervals (CALIPER).[7] Sodium (Na) (ULN: 150 mmol/l) potassium (K) (ULN: 7 mEq/L), and chloride (Cl) ULN: 111 mg/dl) levels were evaluated according to literature.[8]

Daily biochemical values were obtained in those with abnormal levels until normal levels were seen, and peak values were recorded. Urine was collected using adhesive urine collection bags for urinalyses. Urinary solute creatinine ratios (protein/creatinine, calcium/creatinine (ULN for the first 48 h:1.431 and for 72-120 h:1.205 mg/g[9] and (N: 0.02-0.52)[10]), fractional excretion of sodium (FENa) (ULN >2.5),[11] uric acid excretion per deciliter of glomerular filtration rate (GFR) (normal:1.69±0.84 mg/dl GFR),[12] the ratio of maximum reabsorption of phosphate to GFR (normal: 6.9±1.2 mg/dl) (TmP/GFR),[13] were calculated. Urine samples were obtained two weeks apart in patients having abnormal results. Acute kidney injury (AKI) stages were determined according to modified neonatal "Kidney Diseases: Improving Global Outcomes (KDIGO)" definition.[14] Since serum creatinine (sCr) on the first day of life reflects the maternal value, staging was performed by using the lowest value of the previous days as baseline sCr after the second day of life. GFR was calculated according to the Schwartz formula (normal: 10-20 ml/min/1.73 m^2).[15] Neonates who have not shown any clinical signs of dehydration and been initiated fluid or fed by daily need recommendations were included in the study. Patients diagnosed with RMH after the first week of life, who were hospitalised due to dehydration, and who received excessive intravenous hydration treatment were excluded.

In US examination, adhering to the four principles of Slovis et al, false impressions of the increased echogenicity of the kidney were avoided: (1) the intensity of the beam must not be increased to the point that the renal and hepatic parenchyma simulates the very bright specular echoes of the diaphragm; (2) the intensity must be set high enough to ensure penetration of the entire kidney; (3) scanning must be done in a manner that defines capsular echoes of the kidney in order to avoid partial imaging of the liver by superimposition; (4) the entire kidney must be in focus, with homogeneous parenchyma in both the near and far-fields.[3] Ultrasonographic studies were performed in all infants at the first week of life using the LOGIQ S7 Expert ultrasound system (General Electric Medical Systems, Wisconsin, USA) with a 2-8 MHz linear probe. (Figures 1 and 2). Second renal US examinations were performed one week later (Figure 3).

Results

Six patients (30%) were born with normal spontaneous vaginal delivery (NSVD), fourteen (70%) with cesarean section (C/S). Thirteen patients (65%) were male. Three (15%) cases were born prematurely. Mean gestational weight and age were 3094 g (min 650 - max 4220 g) and 37 weeks five days (min 28 - max 40 weeks five days), respectively. Blood pressures were within the normal range according to gestational age in all patients. Ten patients (50%) had maternal prenatal complications. Six patients (30%) were admitted to the hospital because of high bilirubin levels measured at the neonatal outpatient clinic. Two patients (10%) were hospitalised with the diagnosis of hypoxic-ischaemic encephalopathy (HIE), one required hypothermia treatment. Three patients (15%) were admitted to the NICU with the diagnosis of transient tachypnoea of the newborn (TTN), three (15%) with prematurity, two (10%) with premature rupture of membrane (PPROM), two (10%) with suspicion of early sepsis, and one (5%) with hypoglycaemia. One patient undergone US in the outpatient basis because of hypospadias evaluation. Table 1 summarises the clinical features of the patients.

Peak serum BUN levels ranged between 8 and

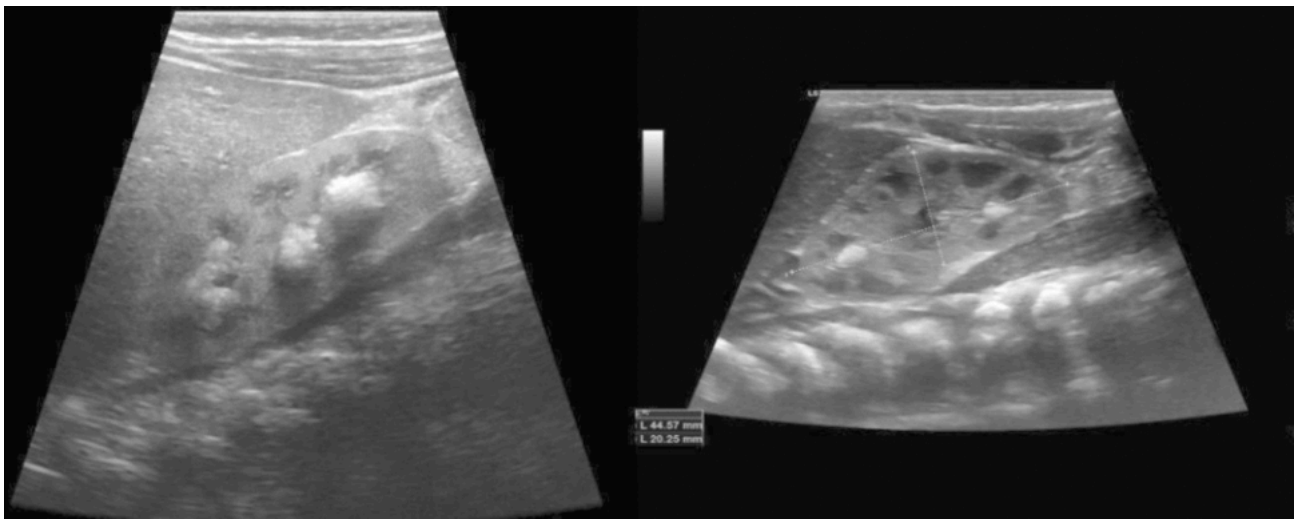


Figure 1. Medullary hyperechogenicity of the kidney.

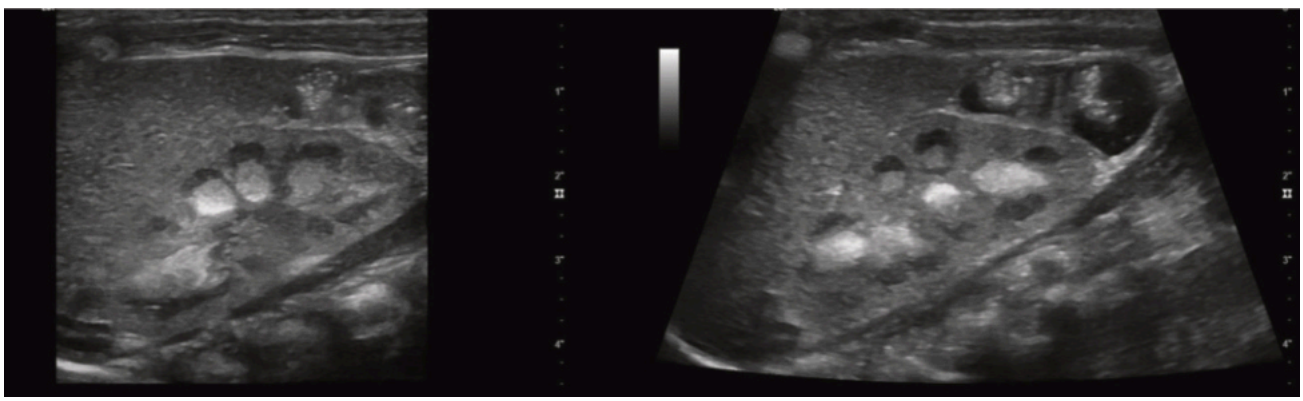


Figure 2. Medullary hyperechogenicity of the kidney (closer view).

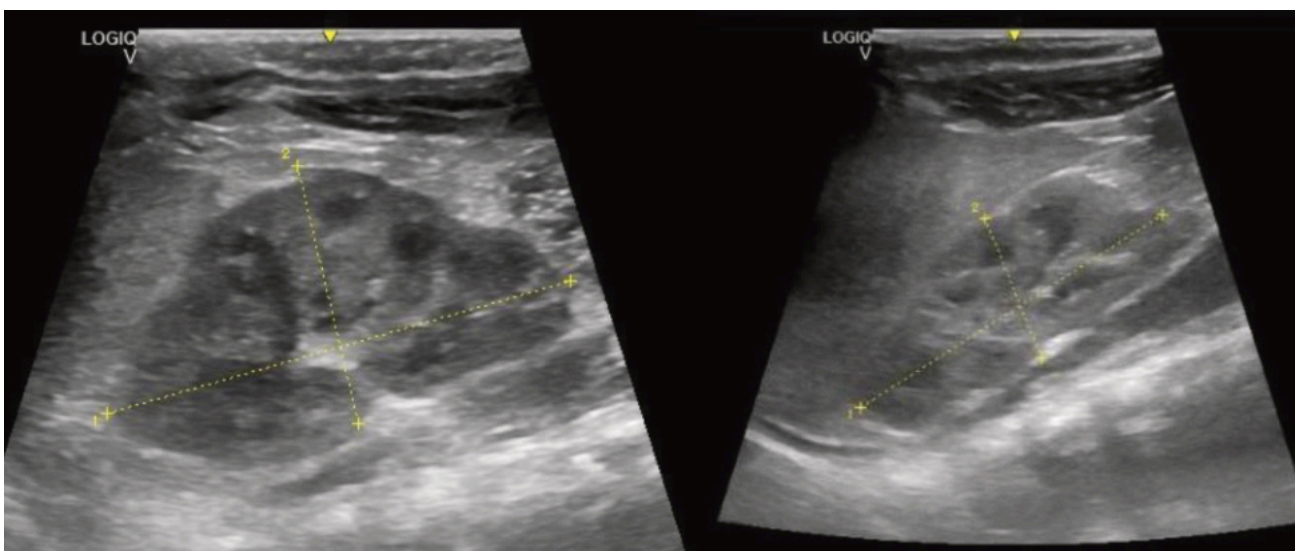


Figure 3. Normal appearance of the kidney.

35 mg/dl (mean:19.9±9.2 mg/dl) and Cr between 0.3 and 1.9 mg/dl (mean:1.13±0.50 mg/dl). Serum BUN and Cr levels were higher than the ULN by age and sex in one third and two thirds of the patients (65%), respectively. According to the modified neonatal KDIGO AKI classification, Stage 1 AKI was defined in six patients. GFR measurements were within the normal range for the first week of life and increased gradually in all patients. AKI was not determined in the remaining patients. Anuria or oliguria were not observed. Mean uric acid level was 7.4 mg/dl, which was within the age- and sex-specific normal limits in all individuals, despite seemingly higher levels compared to the ones beyond the first two weeks of the life; (14 (70%) patients had serum uric acid levels above >6.3 mg/dl). Serum sodium levels were slightly elevated (>150 mmol/l) in three patients (15%) without any signs of dehydration. Potassium and chloride levels as well as Mg, Ca, P and ALP levels were within the normal limits in all patients (Table 2).

Urinary protein/creatinine ratio was above the upper limit of normal in five patients (25%). Urinary calcium/creatinine ratio was elevated only in one patient with HIE who had oligohydramnios in her antenatal history and regressed to normal limits after two weeks. None of the patients had abnormal uric acid excretion rate, or TmP/GFR ratio. FENa was within the normal limits in all patients (Table 3).

The control USGs of all patients were repeated within the first week of life, and these examinations revealed the standard renal size and no dilatation of renal collecting systems. Mild bilateral parenchymal echogenicity in one patient and suspected nephrocalcinosis in the other one disappeared a week later. The hyperechogenic appearance was observed in renal medullary pyramids in all cases. Nineteen patients (95%) had bilateral RMH, and one had only left kidney involvement. All patients with the temporary increase in BUN and Cr levels returned to normal limits within 72-96 hours. Also, the increase in RMH disappeared within the first week in all patients.

Discussion

In this study, we evaluated the clinical and laboratory features of patients with medullary hyperechogenicity in renal USG performed for various reasons in the first days of life. High levels of BUN and creatinine returned to normal limits within 72-96 hours, and the increase in medullary echogenicity improved within a week. Acute kidney injury was not complicated by hypertension, hyperkalaemia, anuria, or Cr levels >2 mg/dl, and none of the patients required interventional treatment. Ali et al[16] reported three patients hospitalised with hypernatremic dehydration and bilateral RMH in US examination. In

Table 1 Demographic and clinical features of patients

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10
Birth weight (g)	3170	4220	3160	1500	3450	3180	3180	3145	3235	650
Birth week	38+4	40+3	40+5	28	37+3	38+1	39+5	38+4	39+3	27+3
Gender	M	M	F	M	F	F	F	F	M	M
APGAR Score (1/5 m)	5/5	9/10	9/10	6/8	9/10	9/10	9/10	9/10	2/7	3/7
Type of delivery	C/S	NSD	C/S	C/S	NSD	NSD	C/S	C/S	C/S	C/S
Antenatal history	Vasa Previa	–	Fetal distress	Fetal distress	Gestational DMP	Preeclampsia	–	–	PROM	–
Reason for hospitalisation	HIE	Jaundice	Jaundice	Prematurity	TTN	Hypoglycaemia	TTN	Jaundice	Neonatal sepsis	Prematurity
	Case 11	**Case 12**	**Case 13**	**Case 14**	**Case 15**	**Case 16**	**Case 17**	**Case 18**	**Case 19**	**Case 20**
Birth weight (g)	3465	3120	3700	3650	3640	1865	3700	2660	3095	4090
Birth week	40+3	38+4	39+5	40+1	40	33+2	38+2	36+3	39+5	38+1
Gender	M	M	M	M	M	M	F	M	F	M
APGAR Score (1/5 m)	9/10	5/7	9/10	9/10	8/10	8/9	9/10	8/9	9/10	8/9
Type of delivery	C/S	C/S	NSD	C/S	NSD	NSD	C/S	C/S	C/S	C/S
Antenatal history	–	Oligohydramnios	–	PROM	PROM	Fetal distress	–	PROM	–	Placenta Previa
Reason for hospitalisation	Jaundice	HIE	Hypospadias	PROM	PROM	Prematurity	TTN	Neonatal sepsis	Jaundice	Jaundice

Table 2 Serum measurements of the patients

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10
BUN (mg/dl)	12	31	20	8	11	10	8	33	17	16
Creatinine (mg/dl)	1.2	1.3	1.4	0.3	1.9	1.9	1.5	1.2	0.7	0.5
Uric acid (mg/dl)	10.1	8.7	7.9	3.4	9.2	8.4	9.4	9.4	5.8	4.1
Na (mmol/l)	140	151	138	131	136	138	140	149	136	141
K (mmol/l)	4.6	4.0	4.5	4.3	4.6	5.1	3.6	4.9	5.3	4.6
Cl (mmol/l)	106	104	103	105	104	102	110	105	104	100
Ca (mg/dl)	9.8	9.7	8.6	9.9	8.7	9.1	9.4	9.9	8.3	9.6
P (mg/dl)	10.4	8.5	7.0	4.3	4.7	5.5	7.0	5.3	7.7	6.0
Mg (mg/dl)	2.7	2.0	1.8	2.1	1.9	2.1	2.0	2.0	2.1	2.3
ALP	130	105	160	247	245	129	149	156	148	250
	Case 11	**Case 12**	**Case 13**	**Case 14**	**Case 15**	**Case 16**	**Case 17**	**Case 18**	**Case 19**	**Case 20**
BUN (mg/dl)	33	22	11	26	20	35	13	25.3	31.8	14
Creatinine (mg/dl)	1.2	1.1	0.3	1.3	1.4	1.8	0.6	1.4	0.9	0.6
Uric acid (mg/dl)	12	10.1	2.5	6	7.2	6.6	5.3	7.6	7.7	6.9
Na (mmol/l)	149	142	139	146	140	138	148	152	151	142
K (mmol/l)	5.4	4.8	4.2	5.3	4.6	4.9	5.3	4.7	4.6	4.8
Cl (mmol/l)	108	104	104	102	102	103	104	109	110	105
Ca (mg/dl)	10.0	8.8	8.9	10.1	8.4	8.2	10.6	8.3	9.9	10.2
P (mg/dl)	8.1	7.5	5.8	7.5	7.2	4.9	5.3	7.5	6.6	6.2
Mg (mg/dl)	2.2	2.0	2.1	2.1	1.7	1.8	2.1	2.4	2.4	2.0
ALP	160	187	142	223	208	143	163	188	174	173

Table 3 Urinary measurements of the patients

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10
Protein/Cr (mg/g)	547	863	**4525**	1081	582	1271	1043	511	790	**2600**
Ca/Cr	0.06	0.009	0.03	0.7	0.2	0.3	0.4	–	0.007	0.01
Uric acid exc rate (mg/dl) GFR	0.9	1.4	0.2	0.8	1.1	1.3	1.7	2.4	1.0	0.5
TmP/GFR (mg/dl)	7.5	5.7	6.8	6.1	5.2	6.3	5.9	7.1	5.2	6.8
FENa	1.1	0.3	0.5	3.3	0.7	0.9	0.2	0.9	0.1	0.4
KDIGO Stage	1	0	0	0	1	1	0	0	0	0
	Case 11	**Case 12**	**Case 13**	**Case 14**	**Case 15**	**Case 16**	**Case 17**	**Case 18**	**Case 19**	**Case 20**
Protein/Cr (mg/g)	946	**1761**	326	948	**2042**	585	**1756**	966	480	549
Ca/Cr	–	**1.9**	0.3	0.01	0.05	0.05	0.006	0.02	0.2	0.02
Uric acid exc rate (mg/dl) GFR	0.9	1.2	1.6	1.4	0.8	1.0	1.0	0.9	0.9	–
TmP/GFR (mg/dl)	6.8	5.8	–	6.7	5.8	5.7	–	5.1	5.8	5.3
FENa	0.2	1.3	1.1	0.4	0.9	0.5	0.3	0.7	0.8	0.4
KDIGO Stage	0	0	0	1	1	1	0	0	0	0

three patients, normal US findings were obtained after intravenous hydration treatment. Makhoul et al[5] found RMH in nine patients with oligoanuria and showed spontaneous recovery without specific treatment. In our study, it is noteworthy that none of our patients with RMH was oligoanuric. We did not give any specific treatment to our patients, including additional hydration therapy. The urinary output of our patients was sufficient. Although there was a transient increase in serum Cr measurements, accompanying dehydration symptoms were not existed and there was a trend of decrease in Cr levels during follow-up. Additionally, AKI stage was evaluated according to the neonatal KDIGO – AKI definition in all patients and only 30% had Stage 1 AKI.

Renal medullary hyperechogenicity detected in the first days of life has a limited clinical significance in the lack of accompanying findings in otherwise healthy newborns. There were only a few studies on this subject. It has even been claimed that RMH may be a normal finding detected in the first days of life.[17] Therefore, it is our belief that we might contribute to the literature by making a discussion ranging from physiopathology to clinical findings.

The kidney structure mechanisms are not well understood during renal development, but it is known that the cortical arteries have large branching, especially afferent arterioles surrounding the glomeruli, while there is little branching in the medullary part of the arterial tree. Vasa recta acts on the medulla by providing oxygen and nutrients and plays a role in the return of electrolytes and dissolved substances reabsorbed by the medullary tubules.[18] Immature salt reabsorption in the thick ascending limb of the loop of Henle (TAL) and urea recycling mechanisms lead to low medullary tonicity, which limits water movement across collecting ducts. Dilution of urine depends on Na delivery to the distal nephron diluting site; concentration of urine, modulated by arginine vasopressin (AVP), requires the presence of a hypertonic renal medullary interstitium. Shortly before birth, long loops of Henle begin to be formed that will eventually contribute to medullary hypertonicity. Also, the urinary concentrating capacity of newborn infants is limited to generate and maintain a deep corticopapillary osmotic gradient due to the relatively shorter loops of Henle, low expression of aquaporin 2 (AQP2) (a marker of AVP activation in the collecting duct), and low sensitivity of the collecting duct to AVP in the antenatal period.[19] Indeed, temporarily limited urine concentration capability is somehow necessary in the postnatal period for better adaptation to extrauterine life, because it enhances clearance of excess extracellular pulmonary fluid. Conceivably, this physiologic impairness may lead to an echogenic appearance at the medulla in the first days of life.

Previous studies have suggested that precipitation of Tamm-Horsfall protein (THP) may be responsible for this transient condition.[20-22] In the newborn, THP, a macroglobulin that is normally found in renal tubules, serum and amniotic fluid, is produced by the cells of TAL and the distal convoluted tubule. Combined with hypoxia-induced renal tubular cell damage in the early life, the overload of THP is likely to result in "tubular stasis". Following first few days of life, increase in GFR and diuresis lead to a large amount of THP excretion in urine. Avni et al[23] demonstrated THP in the urine of two of the five newborns with transient renal failure. However, Riebel et al[17] could not detect an increase in urine THP levels in newborns, but they found a US finding of medullary hyperechogenicity.

Increased medullary echogenicity is transient and usually resolves in the first seven to ten days of life, parallel to the physiological increase in the GFR. The prevalence of RMH in postnatal renal US was found between 3.9-37% in different studies[17,24-26] and reported to be as a transient condition which improves within the first 7-10 days.[2,5]

In our study, we could not measure urinary levels of Tamm-Horsfall protein. Also, our study group had not contained a control group consisting of completely healthy newborns. It was an amorphous group with diagnoses that could and could not be associated with kidney involvement. The possibility of RMH, which we mentioned in the above of the discussion, to be a temporary, normal finding made it difficult for us to make a clear decision at this point.

Conclusions

Considering the findings in our results and relevant literature, we concluded that RMH may be benign and transient condition, spontaneously disappearing with close clinical observation and treatment of the underlying disease without any specific treatment.

However, all healthy newborns should have US, biochemistry, and urine examinations performed and followed-up to ensure. We know that RMH is not observed in all newborns and transient AKI is not detected in all of them. Another question that maintains its importance is "Is

there a difference in prognosis between the cases that RMH is observed and not?" or "What are the long-term results of RMH patients with AKI?". Therefore, in order to better understanding of the potential impact of RMH on renal outcome, a prospective, controlled and well-designed study on a larger patient group may be valuable.

Declarations

Funding: No financial or nonfinancial benefits have been received or will be received from any party related directly or indirectly to the subject of this article.

Conflicts of Interest: The authors certify that there is no conflict of interest with any financial organisation regarding the material discussed in the manuscript.

Availability of Data and Material: Not applicable

Code Availability: Not applicable

Authors' Contributions: Dr. Tas provided substantial acquisition of the data, drafted sections of the manuscript, and revised the manuscript; Dr. Hirfanoglu and Ezgu conceptualised and designed the outline of the manuscript; Dr. Akdulum performed radiological studies; Drs Onal, Turkyilmaz, Ergenekon and Koc critically revised the manuscript; and all authors approved the final manuscript as submitted.

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