Microscopic Examination of Urinary Sediments in Hematuria

Running Title:Microscopic examination of urinary sediments

Dr. Saveta Arora[1*], Dr. S V Jagtap[2]
[1]Post graduate, Department of Pathology, KIMS, Karad
[2] Associate Professor, Department of Pathology, KIMS, Karad
***Corresponding authors**
Dr. S V Jagtap, Associate Professor, Department of Pathology, KIMS, Karad.

Conflict of interest: No potential conflicts of interest relevant to this article were reported.
Authors contribution: All the authors have contributed equally in developing the manuscript
Acknowledgment: Nil

Abstract

Background and Aim: Hematuria is indicative of a wide range of etiologies of varying pathogenic significance. The microscopic examination of urinary sediments for the assessment of morphology of red blood cells (RBCs) is an economic, non-invasive screening to differentiate glomerular (GH) from non-glomerular hematuria (NGH). Hence, this study was performedto observe if 'dysmorphic RBCs' studied at early stages using Wright's stain (WS) and light microscopy (LM) can help in investigation of hematuria.

Methods: Thisprospective, observational study included 500 patients of either sex, aged 2 - 88 years, clinically diagnosed with significant hematuria (more than 3 RBCs/high power field). A detailed history of patient and data regarding possible causes of GH and NGH was collected.Urine sampleswere examined by WS. The slides were observed byLMfor crystals, squamous and transitional cells, bacteria, yeasts, or micro- organisms.All urine specimens were also examined by dipstick test to record urinary proteins and sugar. Statistical analysis was performed by using R software (Version. 3.6.0).

Results: Majority of patients were in the age group of >60years with male preponderance (Male: female=2.13:1). A significant correlation was observed betweenproteinuria andglomerular disease ($P < 0.05$). Casts were present in 72.62% of GH patients among which RBC casts were most common (54.38%). In 4.87%NGH patients the casts were present, among which WBC casts were most common (1.77%). The total sensitivity of WS and LM was 83.94% and 77.37%in GH, whereas 89.82% and 88.50% in all the subgroups of NGH, respectively.

Conclusion: It can be concluded that Wright stain can be used as a diagnostic tool in hematuria.

Keywords: Erthrocytes, Hematuria, Microscopy, Pathology, Urinalysis
DOINumber:10.14704/nq.2022.20.8.NQ44755 **NeuroQuantology2022;20(8):7324-7332**

INTRODUCTION

Hematuria is the condition which is indicative of the presence of red blood cells (RBCs) in the urine. It could be gross or microscopic. When the blood is visible in urine then it is gross hematuria while in microscopic hematuria the blood is detected on urinalysis or urine microscopy.[1] The microscopic hematuria is usually asymptomatic and in routine clinical practice it has a prevalence of 4–5%, which could be due to an underlying disease of the urogenital tract or the kidneys.[2] The presence of hematuria is indicative of a wide range of etiologies of varying pathogenic

significance. It is the most prevalent clinical finding of kidney and urinary tract disease. With respect to its origin, it could be divided to glomerular hematuria (GH) and non-glomerular hematuria (NGH).[3] Glomerular hematuria is a frequent manifestation of many renal diseases. The red blood cells (RBCs) coming through the glomerulus are dysmorphic and indicative of GH. Whereas, RBCs from the lower urinary tract are eumorphic and thus indicative of NGH.[4]

Urinary sediment (U-sed) examination is an integral part of urinalysis and is used worldwide as a diagnostic tool for the diagnosis of hematuria. Most clinicians still utilize U-sed examination directly or indirectly, in their diagnostic approach to renal diseases. The U-sed examination is usually performed microscopically. There are various techniques like phase contrast microscopy (PCM) and light microscopy (LM) which are available for diagnosis of hematuria.[5] For the detection of GH and NGH lesions, the examination of urine for dysmorphic and isomorphic RBCs by PCM is usually preferred in routine clinical practice but they can be highly expensive as compared to LM.[6] The traditional Wright stain is being used since 1890s; it is also a modified version of the Romanowsky method. It is primarily used to stain peripheral blood smears, urine samples, to be examined under a light microscope.[7] The examination of a dried smear of urinary sediment-stained with Wright's stain is also a very effective means of determining urinary erythrocyte morphology and marked hypochromia.[8]

There is limited literature-based evidence validating the utility and standardization of the LM of unstained urine sediment and of Wright's-stained urinary sediment, especially in Indian set up.[3] Hence, this study intended to observe that whether 'dysmorphic RBCs' if studied at early stages using Wright's stain and light microscopy, can help in investigation of hematuria.

MATERIALS AND METHODS

Study design

This single-centered, hospital-based, prospective, observational study was conducted after obtaining approval from the institutional ethics committee, in the Department of Pathology at a private medical college in Karad (Maharashtra) over a period of two years (July 2007 to June 2009). Prior to study initiation, a written informed consent was obtained from all the patients and from parents in case of infants/children. The patients were selected based on convenient sampling technique.

Selection criteria

A total of 500 patients of either sex, aged 2 - 88 years, clinically diagnosed with significant hematuria cases [more than 3 red blood cells per high power field] [9] of the standard urinary sediment were considered for this study. Patients with less than 3 red blood cells per high power field, with any recent antibiotic therapy, any previous kidney or blood infection were excluded from the study.

Data collection

A detailed history including age, sex, and data regarding possible causes of glomerular and non-glomerular hematuria was collected.

Lab investigation

Urine examination

Clean catch midstream urine samples were collected in sterile containers and were processed within 60 minutes after collection. Urine sample (10 ml) was centrifuged at 2000 rpm for 10 minutes and 9.5 ml of the supernatant was removed. The remaining 0.5 ml supernatant was resuspended with sediments by repeated pipetting and kept aside for wet and dry mounting.[10] The color of supernatant was examined for gross hematuria and to distinguish between hematuria and haemoglobinuria.[10] In hemoglobinuria, the supernatant should be clear pink with minimal or no deposits. In hematuria, the supernatant should be cloudy red or dark brown with RBC deposit.[10]

Wright's staining [11]

A drop of the resuspended urine sediment was used for wet slide preparation and was examined with low power objective (lpo) and high-power objective (hpo). Another drop of resuspended urine sediment was used for preparing dried smear for Wright's staining. Dried smears were flooded with Wright's stain (Oxford Lab Fine Chem LLP, Maharashtra, India) for two minutes followed by flooding

with water for four minutes.[11] They were then dried and mounted on a glass slide with coverslip. Wright's stain gives color reaction to cellular components of urine. The urine sediments were examined by light microscopy of unstained sediments and Wright's-stained sediments.

The number of casts was counted in 10 low power field (lpf), averaged, and reported as the number of casts per lpf. High power objective was used to identify casts by type viz. hemoglobin cast for Wright's stain and wet preparations, waxy and granular casts in urine, hyaline cast on wet prep of urine sediment, etc. Erythrocytes, leukocytes and renal epithelial cells were identified and counted using the hpo. At least 10 high-power fields (hpf) were counted, averaged and reported as cells/hpf.[10]

The slides were also examined for crystals, squamous and transitional cells, bacteria, yeasts, or micro- organisms.

All urine specimens were also examined physically and tested with "Siemens Uristix [Protein & Glucose]" to record urinary proteins and sugar by following kit instruction manual.

Criteria for diagnosis [12, 13]

The RBCs variable in size, had distorted irregular outlines & cytoplasmic extrusions, and bizarre shape with irregular outlines, were diagnosed as dysmorphic RBCs and thus were indicative of glomerular hematuria. The criteria of more than 20% dysmorphism of urinary red cells was used to classify hematuria as glomerular.

While the samples in which the RBCs were uniform in size, resembled circulating red blood cells were diagnosed as eumorphic RBCs and thus were indicative of non-glomerular hematuria.[10]

Statistical analysis

Statistical analysis was performed by using R software (Version. 3.6.0). Data were recorded in Microsoft Excel and expressed as frequency and percentage. For testing relationships between categorical variables Chi-square test was performed. Data was considered statistically significant when $P \leq 0.05$ was observed.

RESULTS

The presented study included 500 patients. Table 1 presents the data on distribution of these patients based on age and gender.

Table 1: Distribution of subjects by Age and Gender

Age group (in years)	Gender (n=500)		Total f (%)
	Male f (%)	Female f (%)	
0-10	14 (58.33%)	10 (41.67%)	**24 (4.8%)**
11-20	8 (38.1%)	13 (61.9%)	**21 (4.2%)**
21-30	14 (35%)	26 (65%)	**40 (8%)**
31-40	35 (60.34%)	23 (39.66%)	**58 (11.6%)**
41-50	68 (68%)	32 (32%)	**100 (20%)**
51-60	84 (76.36%)	26 (23.64%)	**110 (22%)**
>60	117 (79.59%)	30 (20.41%)	**147 (29.4%)**
Total	**340 (68%)**	**160 (32%)**	**500 (100%)**

f (%), Frequency (Percentage)

Out of 500 patients included in the study, majority of them were in the age group of >60years with male preponderance (Male: female=2.13:1)(Table 1).

In presented study about 274 hematuria patients (54.80%) had glomerularcause, out of which diabetes was commonest [114 cases (41.61%)] followed byhypertension [70 cases (25.54%)] and acute glomerulonephritis [27 cases(9.85%)].

Further, 226 hematuria patients (45.20%) had non-glomerularcauses, out of which benign prostatic hyperplasia constituted themost common group [108 cases (47.79%)] followed by urinary tract infection [50cases (22.12%)].

Table 2: Association between Proteinuria and Glomerular disease type

Proteinuria levels (mg/dl)	**Glomerular Hematuria f (%)**	**Non-Glomerular Hematuria f (%)**	***P*value[c]**
Trace	0 (0)	108 (100)	0.0005*
1+ (30)	10 (12.82)	68 (87.18)	
2+ (100)	46 (52.27)	42 (47.73)	
3+ (300)	89 (91.75)	8 (8.25)	
4+ (> 2000)	129 (100%)	0 (0%)	

*, significant, C, Chi-square test, f (%), Frequency (Percentage)

A significant correlation was observed betweenproteinuria andglomerular disease ($P < 0.05$). This stipulates that the more the levels of proteinuria, the higher the chances of glomerular hematuria(Table 2)

Table 3: Distribution of Glomerular disease by proteinuria and Casts

Glomerular hematuria	**Total (n=274) f (%)**	**(n=274)**					**(n=274)**			
		Proteinuria f (%)					**Cast f (%)**			
		Trace	**1+**	**2+**	**3+**	**4+**	**RBC casts**	**WBC casts**	**Epithelial casts**	**granular & hyaline**
Diabetesf(%)	114 (41.61)	-	0	14 (12.28)	40 (35.09)	60 (52.63)	60 (52.63)	14 (12.28)	2 (1.75)	4 (3.51)
Hypertension f (%)	70 (25.55)	-	0	10 (14.29)	20 (28.57)	40 (57.14)	46 (40.35)	7 (6.14)	1 (0.88)	2 (1.75)
Acute Post streptococcal glomerulo-nephritisf (%)	27 (9.85)	-	10 (37.04)	5 (18.52)	6 (22.22)	6 (22.22)	8 (7.02)	2 (1.75)	0	0
Post infectious glomerulo-nephritis f (%)	20 (7.3)	-	0	8 (40)	8 (40)	4 (20)	8 (7.02)	8 (7.02)	1 (0.88)	1 (0.88)
Cirrhosis f (%)	15 (5.47)	-	0	5 (33.33)	6 (40)	4 (26.67)	10 (8.77)	2 (1.75)	0	0
Snake bite f (%)	10 (3.65)	-		4 (4)	3 (3)	3 (30)	6 (5.26)	1 (0.88)	1 (0.88)	0
Nephrotic syndrome f (%)	4 (1.46)	-	0	0	1 (25)	3 (75)	2 (1.75)	1 (0.88)	0	0
Multiple myeloma f (%)	4 (1.46)	-	0	0	2 (50)	2 (50)	2 (1.75)	1 (0.88)	0	0
Drugs f (%)	4 (1.46)	-	0	0	1 (25)	3 (75)	3 (2.63)	1 (0.88)	0	0
Primary renal	3 (1.09)	-	0	0	1	2	2	0	1 (0.88)	0

*e*ISSN1303-5150 www.neuroquantology.com

tumorsf (%)					(33.33)	(66.67)	(1.75)			
Systemic lupus erythematosus f (%)	2 (0.73)	-	0	0	1 (50)	1 (50)	1 (0.88)	0	0	0
Sickle cell disease f (%)	1 (0.36)	-	0	0	0	1 (100)	1 (0.88)	0	0	0
Total f (%)	274	-	10 (3.65)	46 (16.79)	89 (32.48)	129 (47.08)	149 (54.38)	37 (13.5)	6 (2.19)	7 (2.55)

f (%), Frequency (Percentage); RBC, Red blood cell; WBC, White blood cell

In majority of glomerular hematuria patients (79.56%) the proteinuria was in the range of (3+ to 4+) and in remaining 20.44% patients it wastrace to 2+. Casts were present in 72.62% cases among which RBC casts were most common (54.38%)(Table 3).

Table 4: Distribution of Non-Glomerular disease by proteinuria and Casts

Non-glomerular hematuria	**Total (n=226) f (%)**	**(n= 226)**					**(n= 226)**			
		Proteinuria f (%)					**Cast f (%)**			
		Trace	**1+**	**2+**	**3+**	**4+**	**RBC casts**	**WBC casts**	**Epithelial casts**	**granular & hyaline**
Benign Prostatic Hyperplasia (BPH)	108 (47.79)	90 (83.33)	12 (11.11)	6 (5.56)	0	-	0	0	0	2 (1.85)
Urinary tract infection(UTI)	50 (22.12)		20 (40)	24 (4)	6 (12)	-	1 (2)	2 (4)	2 (4)	0
Renal calculi	40 (17.7)	0	26 (65)	12 (30)	2 (5)	-	0	2 (5)	0	0
Trauma/Post-operative	18 (7.96)	12 (66.67)	6 (33.33)	0	0	-	0	0	0	0
Hypercalciuria	6 (2.65)	4 (66.67)	2 (33.33)	0	0	-	0	0	0	0
Carcinoma of bladder	4 (1.77)	2 (50)	2 (50)	0	0	-	0	0	1 (2)	1 (25)
Total	226	108 (47.79)	68 (30.09)	42 (18.58)	8 (3.54)	-	1 (0.44)	4 (1.77)	3 (1.33)	3 (1.33)

f (%), Frequency (Percentage); RBC, Red blood cell; WBC, White blood cell

In majority of non-glomerular hematuria patients (96.46%), the proteinuria was in the range of trace to 2+ and in remaining 3.54% patients it was 3+ to 4+. Casts were present in 4.87% patients among which WBC casts were most common (1.77%)(Table 4).

Table 5: Sensitivity of LM and WS in prediction of glomerular hematuria disease

Glomerular hematuria	**Total (n=274) f (%)**	**Dysmorphic RBCs by LM f (%)**	**Dysmorphic RBCs by WS f (%)**
Diabetes	114 (41.61)	72 (63.16)	82 (71.93)
Hypertension	70 (25.55)	66 (94.29)	68 (97.14)

Acute Post streptococcal glomerulonephritis	27 (9.85)	16 (59.26)	18 (66.67)
Post infectious glomerulonephritis	20 (7.3)	18 (90)	19 (95)
Cirrohsis	15 (5.47)	14 (93.33)	15 (100)
Snake bite	10 (3.65)	8 (80)	10 (100)
Nephrotic syndrome	4 (1.46)	4 (100)	4 (100)
Multiple myeloma	4 (1.46)	4 (100)	4 (100)
Drugs	4 (1.46)	4 (100)	4 (100)
Primary renal tumors	3 (1.09)	3 (100)	3 (100)
Systemic lupus erythematosus	2 (0.73)	2 (100)	2 (100)
Sickle cell disease	1 (0.36)	1 (100)	1 (100)
Total	274 (100)	212 (77.37)	230 (83.94)

f (%), Frequency (Percentage); RBC, Red blood cell; WBC, White blood cell; LM, Light microscopy; WS, Wright's staining

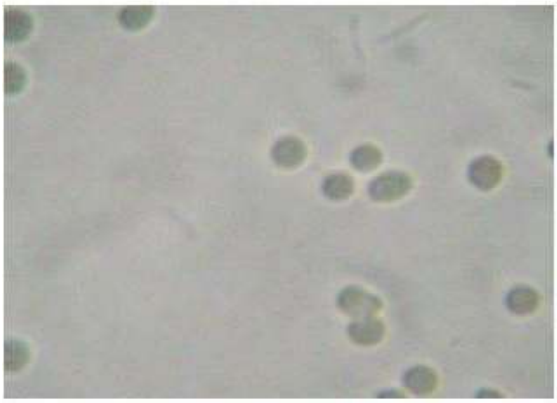
In this study the total sensitivity of Wright's stain (WS) was 83.94% as compared to the Light microscopy(LM) which gave sensitivity of 77.37% in hematuria caused by glomerular diseases.The sensitivity was better by Wright's stain (Color plate 3) than light microscope (Color plate 2) in all the subgroups of glomerular hematuria(Table 5).

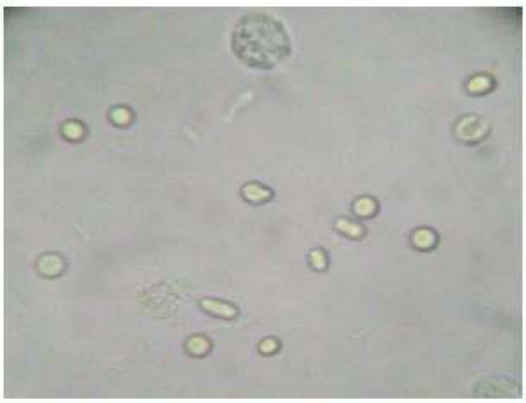
Table 6: Sensitivity of LM and WS in prediction of Non-Glomerular hematuria disease

Non-Glomerular Hematuria	**Total (n=226) f (%)**	**Eumorphic RBCs by LM f (%)**	**Eumorphic RBCs by WS f (%)**
Benign Prostatic Hyperplasia	108 (47.79)	101 (93.52)	101 (93.52)
Urinary tract infection	50 (22.12)	38 (76)	39 (78)
Renal calculi	40 (17.7)	35 (87.5)	36 (90)
Trauma/Post-operative	118 (52.21)	16 (13.56)	17 (14.41)
Hypercalciuria	6 (2.65)	6 (100)	6 (100)
Carcinoma of bladder	4 (1.77)	4 (100)	4 (100)
Total	226 (100)	200 (77.37)	203 (83.94)

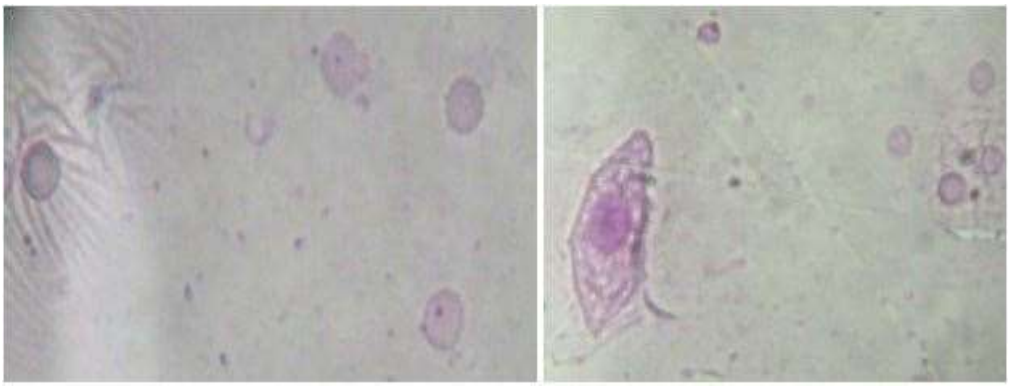
f (%), Frequency (Percentage); RBC, Red blood cell; WBC, White blood cell; LM, Light microscopy; WS, Wright's staining

Out of 226 cases of the urinary eumorphic RBCs, 203 cases were detected by WS, giving the total sensitivity of 89.82% as compared to LM which could detect 200 cases giving sensitivity of 88.50%. The sensitivity was better by Wright's stain (Color plate 4) than light microscope (Color plate 1) in all the subgroups of Non-glomerular hematuria(Table 6).





Colour plate 1: Eumorphic RBCs by LM**Colour plate 2:**Dysmorphic RBCs by LM



Colour plate 3: Dysmorphic RBCs by WS**Colour plate 4:** Eumorphic RBCs by WS

DISCUSSION

The identification ofthe actual origin of hematuria, which could either be glomerular andnon-glomerular, is vital for its diagnosis. The currently available diagnostic methods for hematuria are expensive and require extensive training which makes it time consuming. Examination of urinary erythrocyte morphology by either light microscopy or Wright's staining could be an easy choice of diagnosis for it.[14] Simple method like Wright's staining is readily reproducible and gives permanent record for further analysis.[12]Thus, this prospective study was carried out to study morphological changes in appearance of free red blood cells inurine by light microscopy of unstained sediment and Wright's-stained sediment. The examination of unstained sediment, Wright's-stained sediment casts/slides formed the basis of the study.

In this prospective study, majority of the patients were old with male predominance (Male: female=2.13:1) indicating that older males were most affected by hematuria. This is in accordance with the study conducted by Alsudani (2007),[15] in which the male: female ratio was 1.1:0.5 indicative of male predominance, and the most common age group encountered was 50-60 years.[15] This could be attributed to the fact that males, especially older males, are more prone to and suffer more from infections, enlarged prostates, kidney diseases, cancers etc.

Most of the hematuria patients (54.80%) had glomerular cause, out of which diabetes was commonest followed by hypertension and acute glomerulonephritis. Further, 45.20% hematuria patients had non-glomerular causes, out of which benign prostatic hyperplasia constituted the most common group followed by urinary tract infection. This is in line with findings of Abolfathi et al (2007)[10] which showed that most of the hematuria patients (54.80%) had glomerular and 47% had non-glomerular causes.[10] Hematuria is a cardinal symptom of renal disease. Thus, majority of cases of hematuria ends up in getting diagnosed for symptoms of some renal disorders.[16]

Severe proteinuria (3+ to 4+) was seen in most of the glomerular hematuria patients as compared to mild proteinuria (traces to 1+) noted in non- glomerular hematuria patients, with a significant association among them. This resembles well with the study findings of Kovaˇcevi´c et al (2008)[17] which showed that patients suffering with glomerular hematuria had high levels of proteinuria (4+) as compared to non- glomerular hematuria patients (traces).[17] It usually signals moderate to severe kidney disease (glomerular cause) when hematuria presents with moderate to severe proteinuria.[1]

The diagnostic sensitivity was better by Wright's stain (83.94%) than light microscope (of 77.37%) in all the subgroups of glomerular hematuria. Similarly, in case of all the subgroups of non-glomerular hematuria, the sensitivity was better by Wright's stain (89.82%) than light microscope (88.50%) in the present study. Similar findings were noted by Fogazzi and Delanghe (2018)[18] in their study which showed that the sensitivity of

*e*ISSN1303-5150

Wright's stain and light Microscopy was 86.6% and 70.7% for detection of glomerular and non-glomerular hematuria, respectively.[18] As Wright's stain is a hematologic stain, it facilitates the blood cell types differentiation thus providing a clear image for diagnosis.[19]

In our study absence of casts along with mild proteinuria, severity of hematuria and secondary renal infection obscuring the morphology of urinary red blood cell constituted the false negative cases of glomerular hematuria among which acute glomerulonephritis and diabetes constituted the major group. In acute glomerulonephritis casts were present in only 37.04% of cases which correlates well with Yuste et al (2016)[20] who found 45% casts in cases of acute glomerulonephritis.[20] This observation emphasizes the importance of repeated examinations of urinary sediments, which may help to avoid hasty decisions and unnecessary urological investigations in less typical cases.

The presence of casts and proteinuria, along with minimally dysmorphic RBCs constituted the common cause for false positive cases of glomerular hematuria among which urinary tract infection and renal calculi constituted the most common group, which resembles well with study findings by Yuste et al (2016).[20]

The presented study has its own limitation. The present study had not included the phase contrast microscopy for comparison with light microscopy. Further, as Wright stain was effective in diagnosing glomerular hematuria, hematuria could be included as a surrogate marker of renal diseases in clinical trials and thus future studies are obligatory to validate this.

CONCLUSION

In the current study it can be concluded that Wright stain can be used as a diagnostic tool in hematuria. In country like India with limited resources and lack of facilities for advanced investigations it is imperative to have a simple, inexpensive, and repeatable test like urinalysis that permits more or less an accurate distinction between glomerular and non-glomerular hematuria. The results of urinalysis with clinical correlation and erythrocytes morphology examination should be considered as an important adjunct in the determination of hematuriawhich will be helpful for patients in their further management.

REFERENCES

1. Saleem MO, Hamawy K. Hematuria. StatPearls [Internet]. 2020. [https://www.ncbi.nlm.nih.gov/books/NBK534213/]
2. Bolenz C, Schröppel B, Eisenhardt A, Schmitz-Dräger BJ, Grimm MO. The investigation of hematuria. Dtsch Arztebl Int. 2018;115(48):801-807.
3. Mehta K, Tirthani D, Ali U. Urinary red cell morphology to detect site of hematuria. Indian Pediatr. 1994; 31(9):1039-45.
4. Rhee RL, Davis JC, Ding L, Fervenza FC, Hoffman GS, Kallenberg CGM, et al. The Utility of Urinalysis in Determining the Risk of Renal Relapse in ANCA-Associated Vasculitis. Clin J Am Soc Nephrol. 2018; 13: 251-257.
5. Barraca D, Moreno JA, Aragoncillo I, Praga M, Gutiérrez E, Vega A, et al. Haematuria Increases Progression of Advanced Proteinuric Kidney Disease. PLoS one 2015; 10: e0128575.
6. Hamadah AM, Gharaibeh K, Mara KC, Thompson KA, Lieske JC, Said S, et al. Urinalysis for the diagnosis of glomerulonephritis: role of dysmorphic red blood cells. Nephrol Dial Transplant. 2018; 33(8):1397-1403.
7. Chang BS. Red cell morphology as a diagnostic aid in hematuria. JAMA. 1984; 252(13):1747-9.
8. Chadban SJ, Briganti EM, Kerr PG, Dunstan DW, Welborn TA, Zimmet PZ, et al. Prevalence of kidney damage in Australian adults: The AusDiab kidney study. J Am Soc Nephrol. 2003; 14: S131–S138.
9. Chou D. Henry's clinical diagnosis and management by laboratory methods. JAMA. 2007; 297(16):1827-33.
10. Abolfathi A, Hos aininasab A, Argani H. Differentiation of glomerular from non-glomerular Hematuria by three different methods of microscopic examinations of

erythrocytes in urine. Int J Mech Sci 2007; 32(3): 163-168.
11. Fletcher AL. Wright Staining Urine Sediment. Lab Med. 1978; 9(12):41-41.
12. Fairley KF, Birch DF. Hematuria. A simple method for identification of glomerular bleeding. Kidney Int. 1982; 21: 105-8.
13. Chu YD, Kitamoto Y, Tomita M, et al. Differentiation of haematuria by differential interference microscopy with a simple criterion. Kumamoto Med J. 1990; 42: 63-71.
14. Gutiérrez E, González E, Hernández E, Morales E, Martínez MA, Usera G, et al. Factors that determine an incomplete recovery of renal function in macrohematuria-induced acute renal failure of IgA nephropathy. Clin J Am Soc Nephrol. 2007; 2: 51-57.
15. Alsudani NM. Role of cytological examination of urine in the diagnosis of urinary tract infections. Indian J Urol. 2007; 5(1): 1-5.
16. Sevillano AM, Gutiérrez E, Yuste C, Cavero T, Mérida E, Rodríguez P, et al. Remission of Hematuria Improves Renal Survival in IgA Nephropathy. J Am Soc Nephrol. 2017; 28: 3089-3099.
17. Kovaˇcevi´c Z, Jovanovi´c D, Rabrenovi´c V, Dimitrijevi´c J, Djukanovi´c J. Asymptomatic microscopic haematuria in young males. Int J Clin Pract. 2008; 62: 406- 412.
18. Fogazzi GB, Delanghe J. Microscopic examination of urine sediment: Phase contrast versus bright field. Clinica Chimica Acta. 2018; 487:168-73.
19. Mcdonald MM, Swagerty Jr DL, Wetzel L. Assessment of microscopic hematuria in adults. Am Fam Physician. 2006;73(10):1748-54.
20. Yuste C, Rivera F, Moreno JA, López-Gómez JM. Haematuria on the Spanish Registry of Glomerulonephritis. Sci Rep. 2016; 6:19732.

*e*ISSN1303-5150