Section: Pathology

Original Research Article

IMMUNOHISTOCHEMICAL EXPRESSION OF WILMS' TUMOR 1 PROTEIN (WT1) IN SPORADIC NEPHROBLASTOMA 2 YEARS STUDY

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ABSTRACT

Background: The aim is to study WT-1 expression in different histological subtypes of sporadic Wilms tumour of the kidney. **Materials and Methods:** Total of 40 cases were collected and the study was done in the Department of Pathology at Paediatric tertiary care hospital during the period of 24 months from October 2019 to September 2021. Clinical details including age, sex, imaging were reviewed. Patients were staged according to NWTS and SIOP staging systems. Hematoxilin and eosin (H&E) and immunostaining with WT1 antibody was performed. Normal kidney glomeruli used as positive control. **Result:** The triphasic tumors were the commonest tumors accounting for 30cases (75%), biphasic tumors were seen in 5 cases (12.5%), monophasic (mesenchymal) tumors were seen in 3 cases (7.5%) and monophasic (blastemall) tumorsin 2 cases (5%). Favourable histology was seen in 32 cases (80%), unfavourable histology was seen in 8 cases (20%). In unfavourable histology-focal anaplasia seen in 6 cases (15%), diffuse anaplasia seen in 2 cases (5%). Lymphnode metastasis seen in 2 cases (5%). **Conclusion:** WT1 – Immunohistochemistry should be used for both early diagnosis and prognostication of sporadic Wilms tumor in routine practice.

INTRODUCTION

The kidneys are a pair of excretory organs situated on the posterior abdominal wall, one on each side of vertebral column, behind the peritoneum.[1]

Wilms tumor (WT), an embryonal malignancy of the kidney, occurs most frequently in children under the age of 5 years, affecting 1 in 10,000 individuals. The median age at diagnosis is 3.5 years. About 5-10% of children are affected with bilateral tumors, and these children are diagnosed somewhat earlier [25.5 months] than cases with unilateral, unicentric disease [36.1 months].[2]

Treatment of this malignant disorder includes surgery and chemotherapy for all patients and radiation therapy for those with advanced disease or specific adverse prognostic features. This approach has resulted in good to excellent long-term survival in most patients affected by this malignancy.[3]

WTs are associated with certain congenital defects, in particular sporadic aniridia (a malformation of the iris and surrounding tissue), hemi-hypertrophy, Beckwith-Wiedemannsyndrome (BWS, a congenital over growth syndrome characterized by growth abnormalities and a predisposition to several embryonal neoplasms, including WT), Denys-Drash syndrome (DDS, which consists of the triad WT, intersex disorders, and nephropathy), and various anomalies of the genitourinary tract. In male patients, aniridia and WT are often associated with genitourinary malformations and mental retardation, giving rise to the WAGR syndrome.

WT develops from remnants of immature kidney. Most WTs have a "triphasic" histology consisting of persistent blastemal cells, dysplastic tubules, and supporting stroma. The proportion of each of these components varies from infrequent to abundant within and among individual tumors. Occasionally, focal areas of mesodermal derivatives such as smooth muscle, striated muscle, adipose tissue, and more rarely, cartilage or bone, may be present. In approximately 25-40% of WT specimens, dysplastic lesions representing WT precursors are noticed.[4] These lesions are referred to as nephrogenic rests and the diffuse presence of nephrogenic rests as nephroblastomatosis. Two major categories of nephrogenic rests are recognized: PLNR and ILNR.

Staging criteria for Wilms' tumor are based exclusively on the anatomic extent of the tumor, without consideration of genetic, biologic, or molecular markers. Two major staging systems are currently used: a pre chemotherapy up-front, surgery-based system developed by the National Wilms' Tumor Study Group (NWTSG) and a post

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chemotherapy-based system developed by the International Society of Pediatric Oncology (SIOP). Both stagings systems have proven valuable in predicting outcomes.

WT1 is expressed during all stages of kidney development, while in the mature nephron, WT1 protein expression is restricted to the podocytes.[5] It has also been demonstrated in the mesothelial cells and in stem cells bearing the CD34+ phenotype. The WT1 protein was first classified as a tumor suppressor gene. An activator or oncogenic behavior may be acquired by mutations activator or oncogenic behavior may be acquired by mutations. It is now recognized that WT1 is mutated in about 10% of nephroblastoma.

Aim of the Study

1.To study WT-1 expression in different histological subtypes of Wilms tumour of the kidney

2.To investigate the diagnostic and prognostic value of WT1 N-terminal antibody in sporadic nephroblastomas.

3.To study in detail the immune histochemical (IHC) staining pattern and histopathology of all the cases of nephro-blastomas and to correlate with clinical presentation and radiology.

MATERIALS AND METHODS

The study was done in the Department of Pathology at Paediatric tertiary care hospital during the period of 24 months from October 2019 to September 2021. Clinical data was retrieved from HPE records. The specimens were fixed in 10% buffered formalin, grossed and sections were taken from representative sites. The sections were then processed in automated tissue processor and embedded in paraffin wax Formalin fixed paraffin embedded (FFPE) tissue sections of 5µ thickness were stained with H&E followed by immune histochemical analysis with WT1antibody and were analyzed with Clinical, radiological & histological features. NWTS staging & SIOP risk group stratification done.

Inclusion criterion

- Total nephrectomy specimens and Trucut biopsies of Nephroblastomas.
- Adequate tumor tissue for analysis.
- Complete Clinico-pathological data (age, sex, histopathological diagnosis)

Exclusion criterion

- Traumatic, congenital, inflammatory, conditions, tumors of kidney other than Nephroblastoma.

Two micro sections of 4-5 micron thickness were prepared from the corresponding paraffin blocks, one on albumin coated slide for H&E staining and the other on poly-L-lysine coated slide for immune-histo chemical staining.

Routine Haematoxylin And Eosin (H&E) Staining Procedure.

- Deparaffinize and hydrate slides to distilled water.
- Stain in Harris" haematoxylin for 4 min.
- Rinse in tap water for 1min.
- Clarify in 1% hydrochloric acid solution for 1min.
- Rinse in tap water for 1 min.
- Blue in 0.5% ammonium hydroxide solution for 1 min.
- Rinse in tap water for 1 min.
- Dip in 95% alcohol three times.
- Dip in Eosin four times. Dehydrate in graded alcohols.
- Dip in 95% alcohol three times.
- Dip in Eosin four times.
- Dehydrate in graded alcohols.
- Clear in xylene two changes mount with DPX

Interpitation:

- Nuclei-Blue
- Cytoplasm-Varying shades of Pink
- Muscle fibers-Deep pink/ red
- Red blood cells- Orange/red
- Fibrin-Deep pink

The kits for WT1 immunohistochemical staining were obtained from DAKO Company. Staining was done according to the manufacturers protocol with monoclonal antihuman WT1, Clone 6F-H2, DAKO.

Method of Immuno histochemical staining

Immuno histochemical staining of WT1 was done using peroxidase - antiperoxidase method according to the protocol described by DAKO.

- 4 microns thin sections are taken on poly – lysine coated slides.
- Deparaffinization is done by dipping the slides in 3 changes of xylene
  - 10 min each, followed by 3 changes of absolute alcohol for 5 min each.
  - The slides are washed under running tap water for 15 min.
- Endogenous peroxidase activity is quenched by covering the slides with 3% H2O2 for 30 min.
- Wash under running tap water for 15 min.
- Antigen retrieval done by microwave oven (HIER, heat induced epitoperetrieval) with Tris buffer (1.21g of Tris-Hydroxymethyl methylamine and 3.75 mg of EDTA in 1000 ml distilled water).
- Slides are washed with TBS buffer (9.6g of Tris Hydroxymethyl methylamine and 8.6g of Na Cl in 1000 ml distilled water) pH 7.4-7.6.
- Incubated with Primary antibody (WT1) which is ready to use, at room temperature in a humidifier chamber for 30 minutes.
- The sections were washed again with TBS buffer (9.6 g of Tris Hydroxymethyl methylamine and 8.6 g of Na Cl in 1000 ml distilled water) pH 7.4-7.6.
- Incubated with secondary antibody in a humidifier chamber for 30 minutes.
- The sections were again washed with TBS buffer.
- Chromogen DAB for 20 minutes used for detection of enzymatic activity.

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- Counter staining was done with Haematoxylin.
- Dehydrate in alcohol and xylene.
- Mount with DPX

Immunohistochemical results were evaluated in a semi-quantitative manner and scored according to the percentages of positively staining cells. Cases were divided into the following groups:[6]

(-) No staining and only few scattered positive cells (<10%) was considered to be negative

1 +10 25% of cells stained.

2 +25 50% of cells stained.

3 +>50% of cells stained.

Grading of WT1 Antibody (6F-H2) Stain.[7]

Grading	Pattern
0	No staining
+1	Weak (focal or multifocal
+2	Strong (multifocal or diffuse)

Figure 1: Distension of abdomen

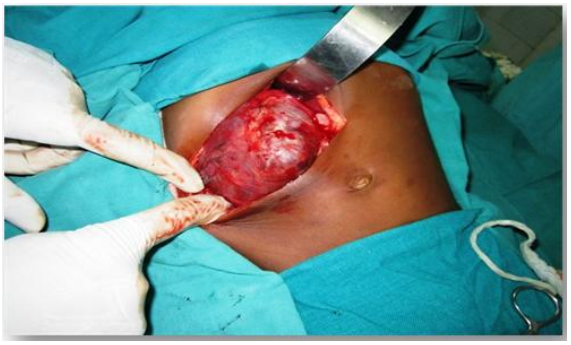

Figure 2: At surgery

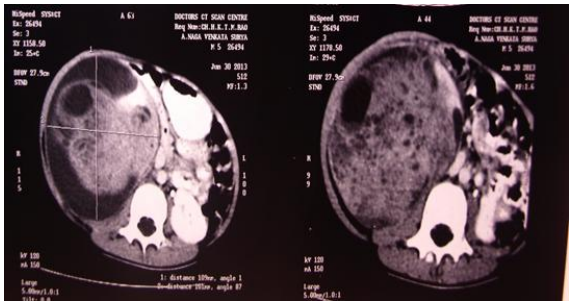

Figure 3: CT Scan – Wilms tumor

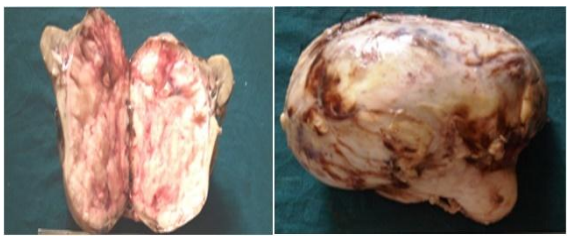

Figure 4: Wilms tumor – gross appearance

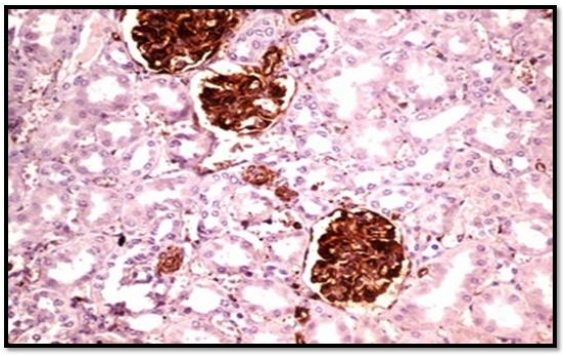

Figure 5: Positive control – Normal Glomerulilar Podocytes, IHC WT1 10X

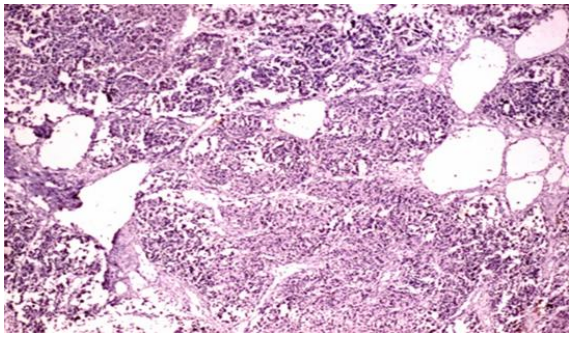

Figure 6: WT1- Negative Case

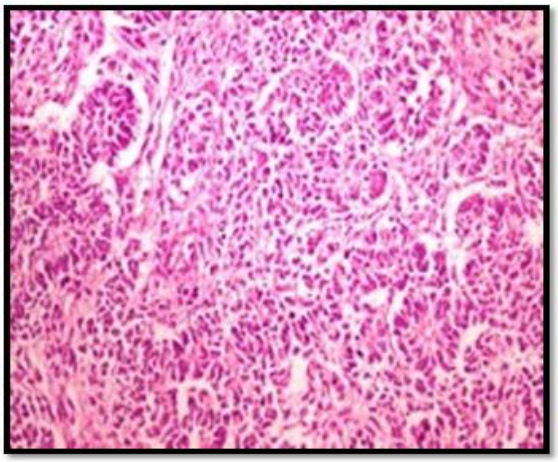

Figure 7: triphasic wilmstumor, H&E,10X

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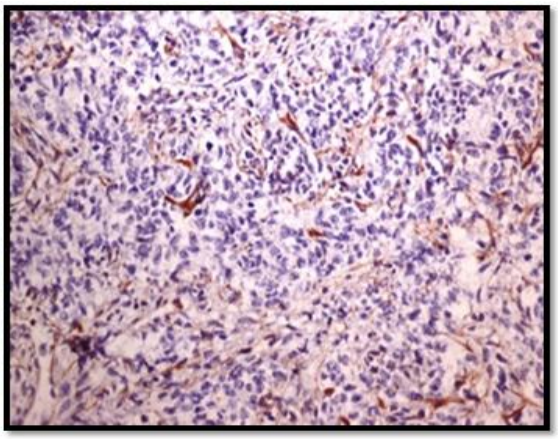

Figure 8: TRIPHASIC WILMS TUMOR, IHC WT1 10X

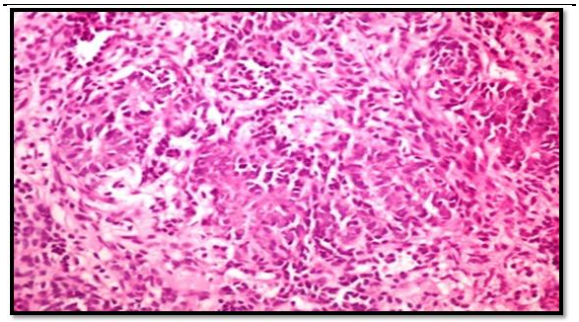

Figure 9: BIPHASIC WILMS TUMOR, H&E 10X

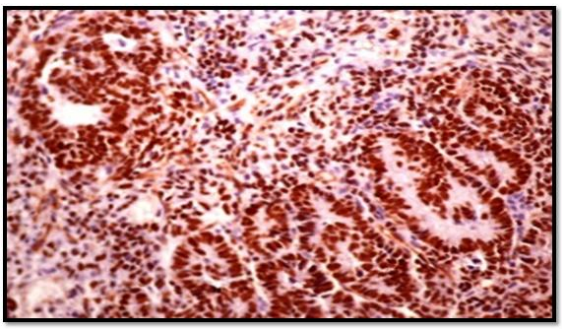

Figure 10: BIPHASIC WILMS TUMOR, IHC WT1 10X

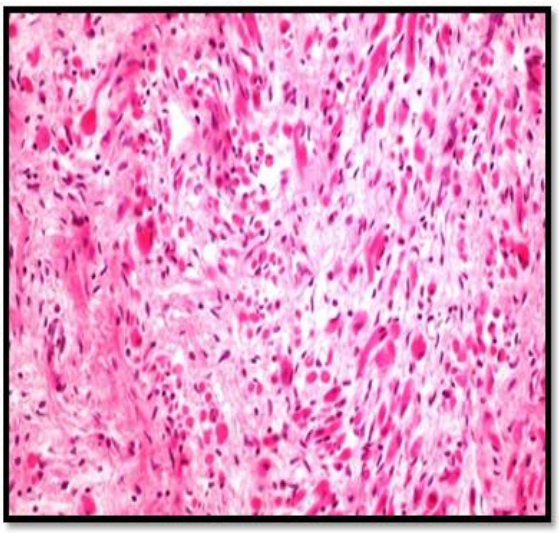

Figure 11: Stromal Predomian Nephroblastoma (Rhabdomyomatous Differentiation) H&E10X

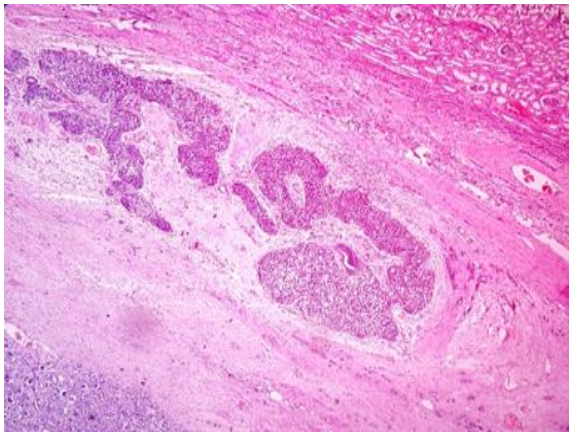

Figure 12: tumor infiltration to capsule H& E 10X

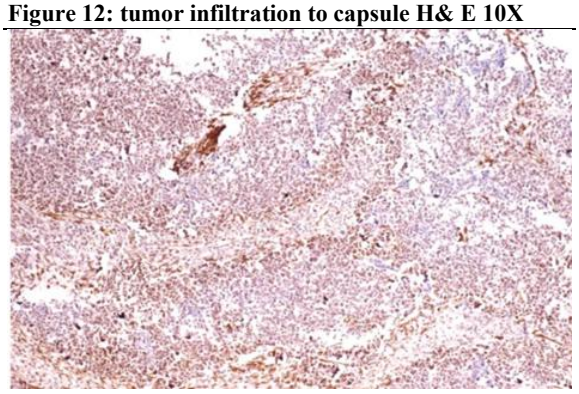

Figure 13: WT1 Staining INDEX+1 BASED ON % OF CELLS

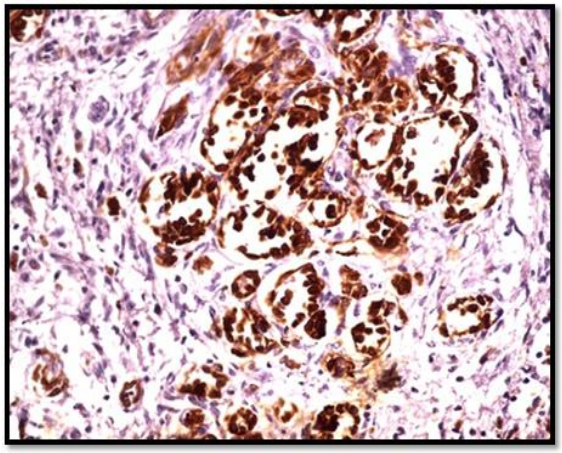

Figure 14: Strongest WT 1 IhcpositivityIn Neoplastic Glomeruli

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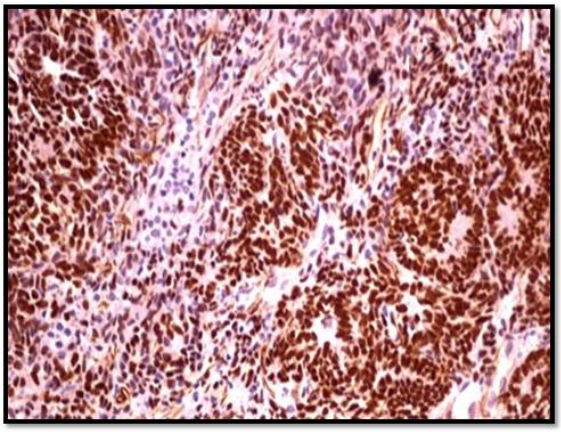


Figure 15: (WT1 STAINING INDEX+2)

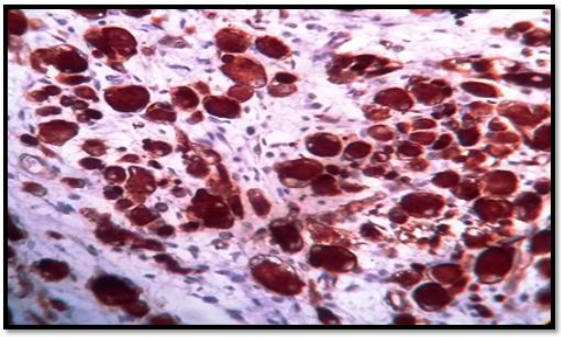
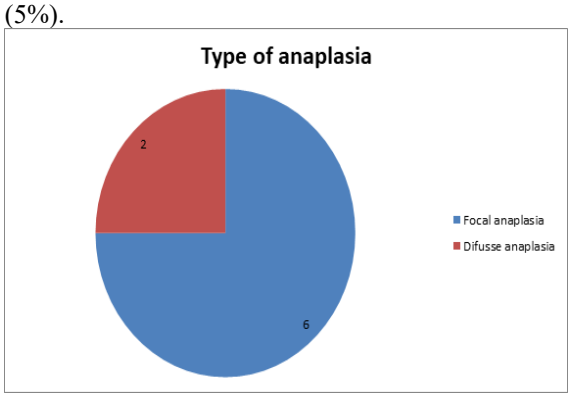


Figure 16: WT1 IHCSTAINING INDEX +3 BASED ON % OF CELLS

RESULTS

The tumors were classified according to WHO histologic classification of wilms tumor and the incidence of different histologic types noted.

The triphasic tumors were the commonest tumors accounting for 30 cases (75%), biphasic tumors were seen in 5 cases (12.5%), monophasic (mesenchymal) tumors were seen in 3 cases (7.5%) and monophasic (blastemall) tumors in 2 cases (5%). Favourable histology was seen in 32 cases (80%), unfavourable histology was seen in 8 cases (20%). In unfavourable histology-focal anaplasia seen in 6 cases (15%), diffuse anaplasia seen in 2 cases (5%) lymphnode metastasis seen in 2 cases (5%).



Graph 1 : Types of Wilms Tumor based on Anaplasia

Table 1: Various types of Wilms Tumor with WT1 expression

Type of tumor	No. of cases	WT1+ VE	WT1-VE
Triphasic tumors	30	26	4
Biphasic tumors	05	5	0
Monophasic mesenchymal tumors	03	0	3
Monophasic blastemal tumors	02	2	0
Total	40	33	7

Table 2: Wilms Tumor based on NWTS staging with WT1 expression.

NWTSG STAGE	No.of cases	Percentage	WT1+VE cases	WT1+VE cases
Stage I	27	67.5%	22	05
Stage II	8	20%	06	02
Stage III	4	10%	04	00
Stage IV	1	2.5%	01	00
Stage V	0	0%	00	00
Total	40	100%	33	07

Table 3: Wilms tumor based on SIOP Risk Groups with WT1 expression

SIOP Risk Group	No of cases	Percentage	WT1+VE cases	WT1 -VEcases
Low Risk	3	7.5%	02	01
Intermediate Risk	33	82.5%	27	06
Hiigh Risk	4	1%	04	00
Total	40		33	07

Table 4: Grading of WT1 Antibody (6F-H2) Stain based on intensivity

Staining index	Number of cases
Negative	07 (17.5%)
1+	10 (25%)
2+	23 (57.5%)

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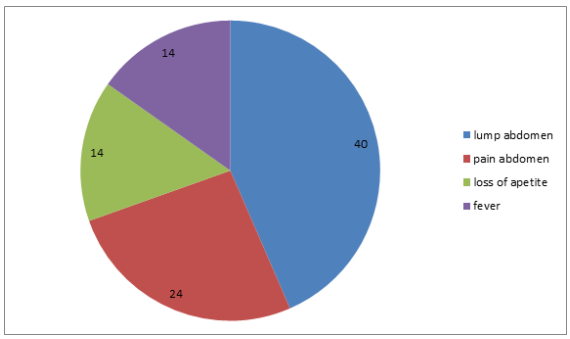

Graph 2: Clinical Presentation of Wilms tumor

DISCUSSION

Wilms' tumor is a pediatric malignancy of the kidney and one of the most common solid tumors in children. At present, the prediction of outcome is based mainly on histology and stage at the time of resection.

In our study, peak incidence of Wilms tumor is seen in the age group of 2 to 5 years. This is similar to the study done by D.K stone et al (2015),[6] and contrast to the study done by Mishra et al (1998) in which the majority of patients were in the first two years of age group. In our study median age group was 3.5 years which is similar to the studies like done by D.K stone et al (2015) and Katarzyna et al (2010).[7,8] This is slightly higher than literature Mishra et al (1998) found median age of 2.5 years.[9]

A K Charles et al,[10] 1997 studied the expression of the Wilms' tumour gene WT1 in the developing human and in pediatric renal tumors: an immune histochemical study. Thirty one pediatric renal tumors were chosen to include 14 Wilms' tumors (13 rena land one extra renal) one with focal anaplasia, one cystic partially differentiated nephroblastoma, sixnephrogenic rests, four mesoblastic nephromas radiology, two malignant rhabdoid tumors of the kidney, two clear cell sarcomas of the kidney, and two renal cell carcinomas.

Mazen A.Ghanem et al,[7] 2000 Studied the prognostic value of WT-1 and EGR-1 in 61 Wilms' tumors of chemotherapeutically treated patients at the protein level, using an immunohistochemical approach. WT-1 was expressed in normal kidney tissues and in the blastemal and epithelial component of Wilms' tumor, whereas stromal tissue was negative. EGR-1 was expressed in normal kidney tissues and in the three main cell types of Wilms' tumor. The percentage of WT-1- and EGR-1- positive cells in a particular area was scored semi quantitatively as ,10, 10–25, 25–50, and >50%. The specimens were regarded as positive when the percentage of positive cells was >10%. In addition, the amount of blastema was estimated by counting the number of low power magnification fields of blastema.

In our study slight female predominance is seen (M:F =0.73:1).This is similar to studies like done by Be Fong et al,[11] (2004) and Mishra et al (1998). This is contrast to the studies like done by Katarzyna (2010) and Soyemi et al,[12] (2013) male predominance noted.

Befong Chen et al observed that Abdominal mass (80%), Abdominal pain (4%) Fever (0%), Hematuria (16%), Haralalrein hard et al,[13] study concluded that Abdominal mass (3.3%), Abdominal pain (83.3%) Fever (0%), Hematuria (6.6%). In our study patient presented Abdominal mass (100%), Abdominal pain (60%) Fever (14%), Hematuria (12%).

In the present series, we found triphasic tumors (75%) to be the most common tumors similar to Yoshiaki et al,[14] (65.8%) and Weirich et al,[15] (45.1%) In our series there were 30 cases of 40 Wilms tumors. The our series one case of cystic nephroblastoma noted similar to the study done by Katarzyna et al. In the present study only epithelial wilms tumors were absent, these are present all other studied like Yoshiaki et al (2012), Katarzyna et al (2008) and Weirich et al (2003).

In our series the biphasic tumors (12.5%) were higher when compared with Mishra et al (1998).The biphasic tumors were absent in studies done by Yoshiaki et al,[9] and Kataryna et al. In present study blastemal predominant wilms tumors (5%) were lower when compared with literature Yoshiaki et al (15.2%) and Kataryna et al (46.1%).

Tumor in various studies	TriphasicTumors	Biphasic tumors	Blastemal tumors	Mesenchymaltumors
Vujanic et al(16)	30%	43%	23%	2%
Katarzyna et al	10.2%	0%	46.1%	5.1%
Yoshiaki et al	65.8%	0%	15.2%	8.8%
Present study	75%	12.5%	7.5%	5%

In present series we found favourable histology was most common similar to studies done by Vujanic et al (87.9%) and Das et al,[17] (94.%). In our series Favourable histology were in 32 cases (80%). In present series Unfavourable histology higher when compared previous two studies.

In present series focal anaplasia noted in 6 cases (15%), diffuse anaplasia was found in 2 cases(5%) that was against in study done by Revsaude et al,[18] (FA-4.5%, DA-7.6%). No anaplasia noted in Be Fong chen et al & Ghanem et al. The anaplasia in present series higher (20%) when compared with study done by Charles et al (7%). In Charles et at study only focal anaplasia noted (1/14).

In the present series, as in all the other studies, we found lower stage (stage I & stage II) tumors be the most common tumors ranging from 56.2% (Katarzyna et al) to 88.8% (Das et al).In our series

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there were 35 cases of 40 wilms tumors. The present series higher stage (stage III, stage IV & stage V) tumors were lower when compared to studies done by Befongchen et al (52%) and Ghanem et al (37.1%).

In our series there were 5 cases of 40 wilms tumors (12.5%). In present series stage V tumors were absent when compared with other studies.

In present study we found most common risk group was intermediate that is similar to study done by Klin padialr et al,[13] (90%). In our series intermediate risk group cases were 33 out of 40 cases of wilms tumors. In our series high risk group cases (4/40) were lower when compared to study done by Katarzyna et al (47.6%).

Expression of WT-1 is not present in all nephroblastomas, and may be present in various other tumours. In nephroblastomas, it is confined to the nucleus and correlates with tumour histology: areas of stromal differentiation and terminal epithelial differentiation show very low levels or no expression of WT-1, whereas areas of blastemal and early epithelial differentiation show high levels of WT-1.

Stromal areas of the tumor did not express WT-1. The negative stromal elements included differentiated mesenchymal tissue, in which adipose tissue and skeletal muscle were seen.WT-1 mRNA has not been detected in the stromal component of Wilms' tumors, nor have recent morphological studies demonstrated the WT-1 protein in the stroma. These results suggest that there is pathogenetic heterogeneity in Wilms' tumors, with stromal-predominant tumors having complete loss of expression the gene.

In present series WT -1 positivity found to be 82.5% (33/40) that is similar to studies done by Charles et al (83.3%) and Carpentieri et al,[19] (71%).The WT-1 sensitivity little higher in our series when compared to study done by Ghanem et al (60%). The seven Wilms' tumors that showed no positive staining, not associated with WAGR syndrome; They were apparently normal children. Uninvolved kidney in the same sections from all cases showed normal staining. Approximately 10% of sporadic Wilms' tumors show WT1 mutations, and may therefore lack WT1 expression. The proportion of negatively staining Wilms' tumors in our series is in keeping with this generally accepted figure.

In present series the uninvolved kidney showed a very intense nuclear staining of glomerular podocytes for WT1, the tubules were negatively stained both were used as internal positive and negative controls respectively, similar to Be Fong Chen et al. Nuclear immunoreactivity of various intensity was observed in blastemal and epithelial elements of the nephroblastomas. The strongest staining was in the neoplastic glomerular component. All the triphasic tumors showed cytoplasmic stain in the stromal cells The heterologous rhabdomyoblasts showed negative staining. The endothelial cells of blood vessels show obvious cytoplasmic staining.

In present series the presence of WT-1 nuclear immune reactivity considered, it is useful to distinguish blastemal predominant nephroblastoma from CCSK or neuroblastoma.

Cytoplasmic WT1 immunoreactivity seems to be more nonspecific than nuclear staining. Cytoplasmic WT1 staining in our series could be seen in the stromal Cells. Cytoplasmatic WT1 staining was also reported in Be Fongchen et al, Carpentieri et al.

In the latter study, weak cytoplasmic staining could be seen in some tubular elements of nephroblastomas. In Carpentieri's study, the cytoplasmic pattern was seen in 75% of nephroblastomas and was almost exclusively stromal and weak. In Be Fong Chen et al study all the tumors showed cytoplasmic stain in the stromal cells.

In present series blastemal WT-1expressionin low NWTSG was found 54%.it was higher than study done by Be Fong chen et al (23%). The higher blastemal expression in lower stages indicates rapid clinical progression. These cases were not respond to routine treatment. In our series blastemal WT-1 expression in higher NWTS stages were found to be 100%, that is higher than study conducted by Be Fong Chen et al(67%).

In present series WT-1 expression of epithelial component was found to be 88.5% (31/35), it was higher than studies done by Ghanem et al.

In present series WT-1 expression in anaplasia was noted in two cases which is similar to study done by Ramani & Cowell et al.[20] they were found positive staining for WT1 in all their Wilms tumours including those with anaplasia. In Charles et al study had a single case with focal anaplasia that was focally positive in the non-anaplastic areas, but negative in anaplastic cells. The reason for this disparity is not clear, but, generally, tumours showed WT1 staining only in some areas of blastema and epithelium. Anaplasia may have arisen from parts of the tumour showing no WT1 expression.

CONCLUSION

WT1 is a very sensitive marker for diagnosis of nephroblastoma of kidney.

The blastemal WT1 immunohistochemical expression is independent prognostic marker for clinical progression.

The Majority the cases blastemal WT1 expression correlated with NWTS & SIOPS.

The minority of cases belonging to low NWTS stages also showed higher blastemal WT1 expression. We need to do close follow-up for those cases.

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