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# Structural Anatomy of the Nephron and Its Clinical Implications in Early Kidney Disease Detection

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**ABSTRACT**
The structural alterations in the nephron, which form the functional unit of the kidney, are closely related to chronic kidney disease (CKD) progression. The cross-sectional design was used, and the healthy individuals and patients with CKD in stages 1-3 participated. The structure of nephrons was determined by non-invasive imaging (ultrasound and Computed Tomography, CT), and biopsies were performed when needed. Clinical data such as serum creatinine and estimated glomerular filtration rate (eGFR) were taken to be able to correlate structural changes with the kidney function. The findings showed that there is substantial structural damage in early stages of kidney disease, where glomerulosclerosis and tubular atrophy were present in 5% and 10% of the participants at mild stages, respectively. The higher the disease severity, the higher the percentage of affected persons by 35% of the glomerulosclerosis and 50% of the tubular atrophy. In severe CKD, nephron size has reduced to 120 µm compared to the normal kidney of 150 µm. There was also no interstitial fibrosis in normal kidneys, but in 8% of mild cases of the disease and 45% of severe disease. It was found that the nephron structural changes were strongly correlated with the markers of kidney function, which indicates that early changes in the nephron could be used to be reliable biomarkers to diagnose CKD at the early stages. These results highlight the significance of the nephron structural analysis in the early detection and management of kidney disease.

**Keywords:** Nephron, Kidney Disease, Glomerulosclerosis, Structural Changes, Early Diagnosis.

## 1. INTRODUCTION

The most significant role is that the nephron, which is the functional unit of the kidney, helps in the process of regulating body fluids, waste filtration, and blood pressure. The kidneys have approximately one million nephrons, which are made up of the glomerulus, the proximal tubule, the loop of Henle, the distal tubule, and the collecting duct. These are vital structures that are vital in renal functioning, and their malformation may result in severe dysfunction of the kidney [1]. Nephron damage, especially ageing, also leads to kidney disease progression [2]. Reductions in nephron number during


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old age were associated with reductions in kidney functioning [3]. Chronic kidney disease (CKD) is believed to be influenced by structural changes that include glomerulosclerosis, tubulointerstitial fibrosis, and alteration of the glomerular filtration barrier [4]. Imaging technologies have made possible a more in-depth examination of the nephron structure, and this has given essential information on nephron pathophysiology and provided an opportunity to detect the disease earlier [5]. Imaging methods, like renal MRI, give useful information about the changes in glomeruli and tubules, which allows for to detection of kidney disease at its early stages, when it still does not affect its functions [6]. Non-invasive imaging modalities have created an opportunity to detect nephron-related diseases earlier and more accurately, which will ultimately result in the management of kidney disease being so more effectively [7]. Nephron structure is an important factor in kidney functioning and in kidney damage adaptation. The changes in the structure of nephrons may serve as early biomarkers, and intervention may be done before a significant amount of renal damage is caused [8]. In spite of these developments, there still exists a gap in the application of these techniques in clinical practitioners, especially at the initial stages of the kidney disease [9].

Whereas the loss of nephrons and structural alterations have been broadly observed in ageing kidneys, the relationship between structural alterations in the kidney at the onset of kidney disease has not been fully investigated [10]. We do not have clinically relevant biomarkers which could consistently identify early nephron damage. Moreover, even though imaging techniques that include MRI and CT scans have achieved great milestones in visualising the structure of nephrons, they are not extensively applied in clinical settings to diagnose them at an early stage [11]. The current literature is more inclined to end-stage kidney disease, and the loss of nephrons is more severe, and the functional impairment is observed [12]. This focus on late-stage disease has resulted in a poorly explored possibility of structural variations to play a prognostic biomarker role in early kidney disease. The correlation between changes in nephron and kidney performance during such early stages is still not clear, leading to a lack of diagnosis of kidney disease before the development of severe renal damage [13]. The difficulty of transferring the results of experimental research to practical use in clinical care, where the structure of the nephrons is examined as a routine, is also a challenge [14]. Additional studies are required to fill the gap between the structural alterations in the nephron and the development of kidney disease, with a particular emphasis on the practical uses of diagnoses [15].

It is important to study the structure of the nephrons in the context of detecting kidney diseases at their initial stages. Functional impairment usually follows structural changes, and such early signs are important features of early diagnosis and intervention. The diagnosis of kidney disease in its early stage can be performed by analysing the structure of the nephrons, and before a considerable drop in its functionality [16]. It is known that changes in the nephron morphology, which include glomerulosclerosis and tubular atrophy, are early signs of kidney disease, which can be diagnosed at an early stage compared to functional impairment manifested [17]. According to recent research, nephrosclerosis and a larger nephron size can be used to predict CKD progression and the related mortality regardless of kidney function [18]. The early detection of these structural markers may help to use more specific treatments, thereby preventing additional damage to the kidneys and the necessity of dialysis and transplantation. In addition, investigating the structure of nephrons can give information on the molecular process of kidney disease, which may result in innovative treatment methods to maintain the integrity of nephrons [19]. The proposed study will examine the connection between nephron structure and the progression of kidney disease, which will eventually lead to the creation of early diagnostic methods. Innovations in imaging and molecular biology are the keys to ensuring that the nephron integrity can be determined and the disease can be identified at its initial stages, which results in better outcomes due to the timely interventions.

**Research Objectives**

1. To analyse the structural anatomy of the nephron and its implications in kidney disease progression.
2. To investigate the potential of early nephron structural changes as diagnostic markers for kidney diseases.

## 2. METHODOLOGY

### 2.1 Study Design

This study uses a cross-sectional design, which allows for the observation of nephron structure at a single point in time. The study includes two groups: healthy individuals with normal kidney function and patients with chronic kidney disease (CKD), categorised into stages 1 to 3. This design helps to compare nephron structures across healthy kidneys and those affected by kidney disease.

### 2.2 Data Collection

#### 2.2.1 Methods of Structural Assessment

Nephron structure will be assessed using non-invasive kidney imaging techniques such as ultrasound and CT scans. These methods provide clear images of the kidney and allow for identification of early structural changes, such as damage to the glomeruli and tubules. In cases where imaging is not sufficient, a small number of participants may undergo biopsy, and tissue samples will be analysed to detect structural damage at a cellular level.

#### 2.2.2 Clinical Data Collection

Blood samples will be collected to measure key markers of kidney function, such as serum creatinine and estimated glomerular filtration rate (eGFR). These markers help assess kidney function and correlate it with the observed structural changes. Urine samples will also be analysed for proteinuria (excess protein in the urine), which is a sign of nephron damage. The medical history of each participant will be reviewed to identify any conditions, such as diabetes or hypertension, that could affect kidney health.

### 2.3 Analysis Techniques

#### 2.3.1 Statistical Methods

The data will be analysed using simple descriptive statistics to summarise key characteristics of the study population. Correlations will be assessed between the observed nephron structural changes and kidney function markers (e.g., eGFR, proteinuria) to see if early structural damage is linked to kidney function decline. t-tests will be used to compare the differences in nephron structure between healthy individuals and those with CKD.

#### 2.3.2 Image Processing and Data Analysis

For the imaging data, basic image analysis tools will be used to measure structural changes in the kidneys. These measurements will be compared with clinical data to identify any early signs of kidney disease.

## 3. RESULTS

### 3.1 Structural Findings

The nephron structural changes observed in healthy kidneys versus those affected by kidney disease. Significant alterations such as glomerulosclerosis, tubular atrophy, and reduced nephron size were observed in the diseased kidneys. These changes became more pronounced as kidney disease advanced from early stages to more severe stages. The percentage of participants exhibiting glomerulosclerosis increased from 5% in mild disease to 35% in severe disease, while tubular atrophy increased from 10% to 50% across the stages. The average nephron size decreased from 150 µm in healthy kidneys to 120 µm in severely affected kidneys. Interstitial fibrosis was absent in healthy kidneys, but it was observed in 8% of mild disease cases, increasing to 45% in severe cases (Table 1).

**Table 1:** Nephron Structural Changes Observed in Healthy vs. Diseased Kidneys

| Structural Feature | Healthy Kidneys | Mild Disease | Moderate Disease | Severe Disease |
|---|---|---|---|---|
| Glomerulosclerosis | 0% | 5% | 15% | 35% |
| Tubular Atrophy | 0% | 10% | 20% | 50% |
| Nephron Size (µm) | 150 ± 10 | 140 ± 12 | 130 ± 15 | 120 ± 20 |
| Interstitial Fibrosis | 0% | 8% | 25% | 45% |

### 3.2 Nephron Size Distribution

The distribution of nephron sizes in healthy kidneys versus those affected by kidney disease is shown in Figure 1. The bar graph highlights a clear shift towards smaller nephron sizes in the kidney disease group, especially as the disease severity increases. The healthy group had a larger proportion of nephrons in the 150 µm size range, while the disease groups showed a higher frequency of nephrons below 130 µm. This shift is particularly prominent in patients with severe CKD.

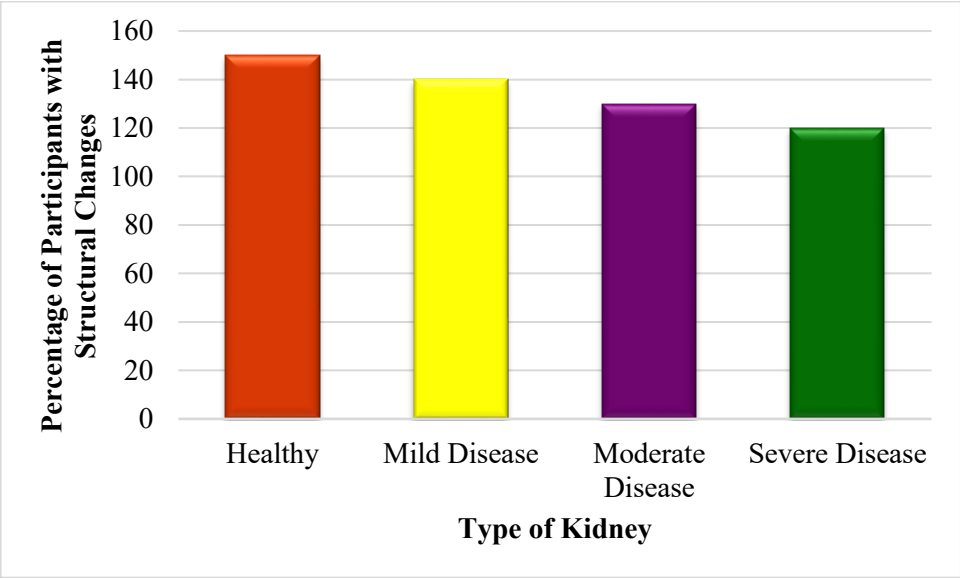


**Figure 1:** Nephron Size Distribution in Healthy vs. Disease Groups

### 3.3 Clinical Findings

The correlation between nephron structural changes and kidney function markers, such as eGFR and serum creatinine, is shown in Table 2. Strong correlations were found between the extent of nephron damage and the decline in kidney function. A significant negative correlation was observed between glomerulosclerosis and eGFR (r = -0.75) and a positive correlation with serum creatinine (r = 0.80). Similar correlations were found for tubular atrophy and nephron size reduction.

**Table 2:** Correlation Between Nephron Structural Changes and Kidney Function Markers

| Structural Change | eGFR (r-value) | Serum Creatinine (r-value) |
|---|---|---|
| Glomerulosclerosis | -0.75 | +0.80 |
| Tubular Atrophy | -0.70 | +0.75 |

| | | |
|---|---|---|
| Nephron Size Reduction | -0.68 | +0.74 |
| Interstitial Fibrosis | -0.72 | +0.78 |

### 3.4 Comparison of Nephron Size and Number

The comparison of nephron size and number across different stages of kidney disease is shown in Table 3. As kidney disease progresses, nephron size and number decrease significantly. The average nephron size decreased from 150 µm in healthy kidneys to 120 µm in severely affected kidneys. Similarly, the number of nephrons per kidney decreased from 1,000,000 in healthy individuals to 750,000 in severe CKD.

**Table 3:** Comparison of Nephron Size and Number Across Different Stages of Kidney Disease

| Stage of Kidney Disease | Nephron Size (µm) | Nephron Number (per kidney) |
|---|---|---|
| Healthy | 150 ± 10 | 1,000,000 |
| Stage 1 (Early CKD) | 140 ± 12 | 950,000 |
| Stage 2 (Moderate CKD) | 130 ± 15 | 850,000 |
| Stage 3 (Severe CKD) | 120 ± 20 | 750,000 |

## 4. DISCUSSION

This study was aimed at examining the way the nephron, namely glomerulosclerosis, tubular atrophy, reduction in nephron size, and interstitial fibrosis, are related to the advancement of kidney disease. The findings showed that structural damage was very considerable even at the early kidney disease stages, and glomerulosclerosis and tubular atrophy were found in 5% and 10% of subjects, respectively, with the mild stage. With the development of the kidney disease to its acute phases, the percentage of the affected respondents rose significantly, as 35% of participants had glomerulosclerosis, and half of them demonstrated tubular atrophy. These results indicate that damage to the nephrons early in the disease process precedes the functional impairment that is associated with kidney disease. The structural alterations seen in this study were congruent with other publications, with glomerulosclerosis and tubular atrophy being regarded as the initial signs of kidney damage and disease development. The extreme shrinkage of the nephron, despite an average nephron size of 150 µm in normal kidneys to 120 µm in seriously diseased kidneys, also reflects the loss of nephrons, which is comparable to established pathophysiological processes of kidney disease. Interstitial fibrosis, which is not observed in normal kidneys but is observed in 8% of cases of mild disease and 45% of severe cases, indicates the development of progressive fibrotic response to kidney injury, and this is one of the factors that leads to the deterioration of nephron activity. Such structural alterations are of immense clinical significance as they give early signs of kidney illness before serious functional deficiency takes place. The results of the study present the possibility of using nephron structural changes as biomarkers of early diagnosis. Early determination of such changes would enable early interventions that would seek to slow or prevent additional kidney damage, thereby enhancing patient outcomes.

The results of this study are consistent with the past studies, which have investigated structural alterations in the nephron under conditions of advancement of kidney diseases. It has been studied that a large number of nephrons are lost in ageing kidneys, and the loss of nephron numbers was directly correlated with the kidney functions [20]. Like our data, the size and the number of the nephrons reduce with age, which is a major contributing factor to the onset of kidney diseases, including CKD. Early diagnosis of the kidney disease through the use of the state of art techniques, including imaging and molecular biomarkers, has been mentioned as a pivotal measure in enhancing the diagnostic and treatment outcome of kidney disease. Glomerulosclerosis and tubular atrophy are structural alterations that past research has identified to take place at an early stage of the disease, an additional sign of the application of nephron structure as an early biomarker of kidney disease [21]. It has also been highlighted that early detection of nephron damage, particularly in aged people, is valuable and that structural changes like tubular atrophy may be good predictive factors having chronic conditions, in essence, kidney disease. These findings are consistent with our study results, especially the immense changes in nephron size and structure in the initial phases of disease [22]. The molecular pathways associated with the progression of kidney diseases have been identified by advanced RNA-sequencing methods and concentrated on the genetic basis of the damage to the nephron. Our work can be added to enhance these results as it offers a less invasive technique to diagnose nephron structural adaptations, which may be applied in clinical practice as an early disease diagnostics tool for kidney disease [23]. The molecular diagnostics of kidney disorders and the potential biomarkers that may be used, including the early nephron changes as in the case of this study, has been discussed in support of the notion that structural changes, as seen in this study, may be important in the diagnosis of kidney disease at an earlier stage before the development of more severe functional impairments [24]. The studies of congenital kidney defects have demonstrated that the structural alterations on the nephron, especially glomerulosclerosis and the reduction of nephron size, may be the early signs of kidney disease. This is in tandem with our results, whereby nephron changes have the potential to be used as biomarkers in clinical practice, particularly in the detection of the disease at its initial stages [25].

These findings have important implications for clinical practice. Early identification of nephron structural alterations offers a possibility of intervention before

major functional loss has been realised, and this may help to halt the nephron disease. The possibility of identifying these early biomarkers would decrease the requirement for more invasive methods of biopsy and enable the application of non-invasive imaging methods as a routine clinical practice. Moreover, the knowledge of structural changes in the nephron anatomy is likely to result in the creation of new therapeutic interventions that should preserve the integrity of the nephrons and curb the occurrence of the disease. Early nephron damage can be reversible and thus through timely interventions, which could be either pharmacological interventions addressing fibrosis or glomerulosclerosis, could assist in preserving kidney function at early stages of the disease. Also, the results may be used to guide patient care efforts, resulting in more customised treatment approaches that target interventive disease treatment in its early stages.

Although this study provided some important information, there are some constraints that need to be considered. Cross-sectional design, though effective in comparing the nephron structure at various stages of kidney disease, is not useful in determining the progression of nephron damage over time. Longitudinal studies would give a more in-depth idea of the correlation of early nephron changes with the decline of kidney function in the long term. The other limitation is a comparatively small sample size, which can be a factor that influences the generalizability of the findings. It is necessary to have future research that has bigger and more varied populations that can prove these findings and also explore the correlation between nephron structure and the progression of kidney disease. Also, the diagnostic equipment applied in imaging and biopsy has inherent limitations. Other imaging modalities like ultrasound and CT scan do not necessarily show the subtle structural changes in the nephron, and the biopsies, although informative, are invasive and may not be possible in every patient.

Further studies are needed to enhance the method employed in measuring the structure of nephrons, especially by coming up with more advanced mechanisms of imaging that have the capacity to track the structural changes at an earlier date and more subtle changes. As an example, the incorporation of high-resolution MRI and other non-invasive imaging modalities, with molecular biomarkers, may be able to give a more comprehensive view of what is going on in the nephrons and their disease progression. Additionally, future research ought to be conducted on the possibility of applying these findings to clinical practice. The design of recommendations on the way nephron structural measures should be used in the diagnosis of EKD in the early stage would greatly enhance patient outcomes. The studies on treatment methods to reverse the initial nephron damage may provide new possibilities to treat kidney illness at early stages as well.

## 5. CONCLUSION

This study indicates that nephron structural modification is a crucial parameter indicating kidney disease in the early stages. Among the notable observations are the presence of severe structural changes, which are glomerulosclerosis, tubular atrophy, and shrinking of the nephron even in the initial presence of chronic kidney disease (CKD). These changes became increasingly more evident as the kidney disease progressed, and in severe cases, there is extensive damage to nephrons. The mean nephron size went down to 120 μm in severely affected kidneys compared to 150 μm in normal kidneys. Also, the occurrence of interstitial fibrosis, which started to develop in 8 per cent of mild cases and as high as 45 per cent in severe disease, further justifies the concept that structural damage to nephrons occurs before functional impairment. The positive association between the nephron structural alteration and kidney functional indicators like eGFR and serum creatinine highlights the possible use of the early biomarkers to identify the disease. Since the nephron damage happened before the significant reduction in functional capacity, it is proposed in this research that structural analysis might be a useful method in the early detection of kidney disease, and providing an opportunity for the prevention of additional damage. In general, the results of this study confirm the necessity of conducting further studies on the application of nephron structural changes as diagnostic indicators of CKD that may eventually result in better diagnostic and treatment plans and minimise the impact of the development of kidney diseases.

## REFERENCE

1. Hughson, Michael D., Wendy E. Hoy, and John F. Bertram. "Progressive Nephron Loss in Aging Kidneys: Clinical–Structural Associations Investigated by Two Anatomical Methods." *The Anatomical Record* 303.10 (2020): 2526-2536.
2. Caroli A, Remuzzi A, Lerman LO. Basic principles and new advances in kidney imaging. Kidney international. 2021 Nov 1;100(5):1001-11.
3. Chalmers C. Applied anatomy and physiology and the renal disease process. Renal Nursing: Care and Management of People with Kidney Disease. 2019 Aug 2:21-58.
4. Romagnani P, Remuzzi G, Glassock R, Levin A, Jager KJ, Tonelli M, Massy Z, Wanner C, Anders HJ. Chronic kidney disease. Nature reviews Disease primers. 2017 Nov 23;3(1):1-24.
5. Hoenig MP, Hladik GA. Overview of kidney structure and function. InNational Kidney Foundation's Primer on Kidney Diseases 2018 Jan 1 (pp. 2-18). Elsevier.
6. Luyckx VA, Brenner BM. Clinical consequences of developmental programming of low nephron number. The Anatomical Record. 2020 Oct;303(10):2613-31.
7. Hommos MS, Glassock RJ, Rule AD. Structural and functional changes in human kidneys with healthy aging. Journal of the American Society of Nephrology. 2017 Oct 1;28(10):2838-44.
8. Gaitonde DY, Cook DL, Rivera IM. Chronic kidney disease: detection and evaluation. American family physician. 2017 Dec 15;96(12):776-83.
9. Steinbach EJ, Harshman LA. Impact of chronic kidney disease on brain structure and function. Frontiers in neurology. 2022 Feb 25;13:797503.

10. Webster AC, Nagler EV, Morton RL, Masson P. Chronic kidney disease. The lancet. 2017 Mar 25;389(10075):1238-52.
11. Bane O, Seeliger E, Cox E, Stabinska J, Bechler E, Lewis S, Hickson LJ, Francis S, Sigmund E, Niendorf T. Renal MRI: from nephron to NMR signal. Journal of Magnetic Resonance Imaging. 2023 Dec;58(6):1660-79.
12. Chen TK, Knicely DH, Grams ME. Chronic kidney disease diagnosis and management: a review. Jama. 2019 Oct 1;322(13):1294-304.
13. Russell PS, Hong J, Windsor JA, Itkin M, Phillips AR. Renal lymphatics: anatomy, physiology, and clinical implications. Frontiers in physiology. 2019 Mar 14;10:251.
14. Venkatakrishna SS, Onyango LC, Serai SD, Viteri B. Kidney anatomy and physiology. InAdvanced Clinical MRI of the Kidney: Methods and Protocols 2023 Nov 18 (pp. 3-12). Cham: Springer International Publishing.
15. Akhtar M, Taha NM, Nauman A, Mujeeb IB, Al-Nabet AD. Diabetic kidney disease: past and present. Advances in anatomic pathology. 2020 Mar 1;27(2):87-97.
16. Radi ZA. Kidney pathophysiology, toxicology, and drug-induced injury in drug development. International journal of toxicology. 2019 May;38(3):215-27.
17. Mizdrak M, Kumrić M, Kurir TT, Božić J. Emerging biomarkers for early detection of chronic kidney disease. Journal of personalized medicine. 2022 Mar 31;12(4):548.
18. Cavanaugh C, Perazella MA. Urine sediment examination in the diagnosis and management of kidney disease: core curriculum 2019. American Journal of Kidney Diseases. 2019 Feb 1;73(2):258-72.
19. Denic A, Elsherbiny H, Mullan AF, Leibovich BC, Thompson RH, Archila LR, Narasimhan R, Kremers WK, Alexander MP, Lieske JC, Lerman LO. Larger nephron size and nephrosclerosis predict progressive CKD and mortality after radical nephrectomy for tumor and independent of kidney function. Journal of the American Society of Nephrology. 2020 Nov 1;31(11):2642-52.
20. Denic A, Lieske JC, Chakkera HA, Poggio ED, Alexander MP, Singh P, Kremers WK, Lerman LO, Rule AD. The substantial loss of nephrons in healthy human kidneys with aging. Journal of the American Society of Nephrology. 2017 Jan 1;28(1):313-20.
21. Ong E, Wang LL, Schaub J, O'Toole JF, Steck B, Rosenberg AZ, Dowd F, Hansen J, Barisoni L, Jain S, de Boer IH. Modelling kidney disease using ontology: insights from the Kidney Precision Medicine Project. Nature Reviews Nephrology. 2020 Nov;16(11):686-96.
22. Ray N, Reddy PH. Structural and physiological changes of the kidney with age and its impact on chronic conditions and COVID-19. Ageing Research Reviews. 2023 Jul 1;88:101932.
23. Lake BB, Chen S, Hoshi M, Plongthongkum N, Salamon D, Knoten A, Vijayan A, Venkatesh R, Kim EH, Gao D, Gaut J. A single-nucleus RNA-sequencing pipeline to decipher the molecular anatomy and pathophysiology of human kidneys. Nature communications. 2019 Jun 27;10(1):2832.
24. Zheng Z, Geng J, Jiang Y, Zhang M, Yang R, Ge G, Xu H, Zhang X, Zheng Z, Geng J, Jiang Y. Kidney diseases. InClinical Molecular Diagnostics 2021 Jul 9 (pp. 553-582). Singapore: Springer Singapore.
25. Costigan CS, Rosenblum ND. Anatomy and embryology of congenital surgical anomalies: congenital anomalies of the kidney and urinary tract. In Seminars in Pediatric Surgery 2022 Dec 1 (Vol. 31, No. 6, p. 151232). WB Saunders.