Research Square

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Analysis of Factors Influencing IgA Vasculitis-associated Nephritis in Pediatric Patients

Li LIU
The First Affiliated Hospital of Xi'an Jiaotong University
Mengwei HOU
The First Affiliated Hospital of Xi'an Jiaotong University
Yue WU
The First Affiliated Hospital of Xi'an Jiaotong University
Xiaohong LIU
The First Affiliated Hospital of Xi'an Jiaotong University
Yanping HUANG
The First Affiliated Hospital of Xi'an Jiaotong University
Yonghua YANG
The First Affiliated Hospital of Xi'an Jiaotong University
Cui LIU
liucui0809@163.com

Xi'an International Medical Center Hospital

Research Article

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Abstract

Objective

To investigate factors influencing childhood IgA vasculitis (Henoch-Schönlein purpura) and IgA vasculitis nephritis (Henoch-Schönlein purpura nephritis).

Methods

Clinical data from children diagnosed with IgA vasculitis and admitted to the Department of Pediatrics at the First Affiliated Hospital of Xi'an Jiaotong University from January 1 to December 31, 2021, were retrospectively collected and analyzed. Baseline characteristics were compared between the nephritis (IgAVN) and non-nephritis (IgAV) groups, and we performed multivariate logistic regression analysis to identify independent risk factors. Receiver operating characteristic (ROC) curves were constructed to assess the predictive value of variables identified as significant risk factors.

Results

Age and uric acid (UA) levels were significantly higher in the nephritis (IgAVN) group compared to the IgAV group ($P < 0.05$). Elevated UA was identified as an independent risk factor for IgAVN, with an odds ratio (OR) of 1.010 (95% confidence interval [95% CI]: 1.005–1.015). UA predicted IgAVN with a sensitivity of 64.2% and specificity of 74.6%, at an optimal cut-off value of 277 μmol/L. The area under the ROC curve (AUC) was 0.711 (95% CI: 0.639–0.783; $P < 0.05$).

Conclusion

UA levels in children with IgA vasculitis show moderate predictive value for the development of IgAVN. UA assessment may be integrated with urine sediment analysis, renal pathology findings, and other clinical indicators to jointly evaluate disease prognosis. (This study is a retrospective observational study and does not involve prospective intervention assignment. Therefore, clinical trial registration was not required.)

What is Known - What is New

Previous studies have suggested that gastrointestinal involvement (including a positive fecal occult blood test) in children with IgAV is associated with an increased risk of kidney involvement. However, the present study found no statistically significant difference in fecal occult blood test results between the IgAVN and IgAV groups. Previous studies have also indicated that older age is a risk factor for IgAVN in children. Nevertheless, after adjusting for both age and serum UA levels in multivariate logistic

regression analysis, only serum UA level was identified as an independent risk factor with predictive value.

Introduction

Immunoglobulin A vasculitis (IgAV), formerly known as Henoch–Schönlein purpura (HSP), is the most common immune-complex-mediated small-vessel vasculitis in children and adolescents. The incidence varies across countries and regions, ranging from 3.5 to 26.7 cases per 100,000 individuals per year among those under 15 years of age[1]. The peak incidence occurs between 4 and 6 years of age[2]. The main clinical features include palpable purpura, arthralgia or arthritis, abdominal colic, and hematuria and/or proteinuria. IgAV can affect multiple organ systems, including the kidneys, joints, gastrointestinal tract, and, less commonly, the central nervous system, reproductive system, heart, and lungs[3]. Although most cases are self-limiting and have a favorable prognosis, a subset of patients develop chronic or long-term organ damage. The long-term outcome is primarily determined by the severity of renal involvement[4]. IgAV nephritis (IgAVN), previously termed Henoch-Schönlein purpura (HSP) nephritis, represents the renal manifestation of IgAV and is the most common secondary glomerular disease in pediatric patients. A study indicates that approximately 30% of children with IgAVN continue to exhibit persistent kidney disease at the end of a 3-year follow-up period[5]. Currently, no definitive preventive measures for IgAVN exist. However, the identification of predictive biomarkers could enable more accurate prognostic assessment, guide the frequency and scope of monitoring in children with IgAV, and facilitate early intervention to delay disease progression and improve outcomes.

Some studies have consistently identified age and gastrointestinal symptoms as independent risk factors for IgAVN[6–8]. However, other findings remain inconsistent or even contradictory. Carucci NS et al. reported no statistically significant differences in gender, neutrophil count, or lymphocyte count between patients with and without renal involvement[8]. Kim WK et al. found that female sex and an elevated neutrophil-to-lymphocyte ratio were associated with increased risk of kidney involvement in IgAV (HSP)[9]. Additionally, Jin Y et al. identified peripheral blood white blood cell count as a risk factor for renal crescent formation in IgAVN—a histopathological feature often indicative of poor prognosis[10]; Nie Y et al. reported that elevated D-dimer levels were significantly associated with renal involvement in IgAV[11]. In contrast, Zeybek et al. observed no significant differences in coagulation parameters or thrombosis risk between IgAV patients and healthy controls[12].

Therefore, this study aimed to analyze differences in blood-based indicators between patients with IgAVN and those with IgAV without nephritis to provide a basis for early prediction of progression from IgAV to IgAVN.

1. Materials and methods

1.1 Research Subjects

This study selected 206 pediatric patients younger than 14 years who were newly diagnosed with IgAV (HSP) and had not received any treatment. These patients were admitted to the First Affiliated Hospital of Xi'an Jiaotong University from January 1, 2021 to December 31, 2021. Two patients were excluded due to the absence of urine routine test results, and a total of 204 patients were included in the study. The diagnostic criteria for IgAVN (HSP nephritis) were based on the "Evidence-Based Guidelines for the Diagnosis and Treatment of Henoch-Schönlein Purpura Nephritis (2016)"[13]: During the first 6 months of the IgAV (HSP) disease course, hematuria and/or proteinuria occur. The diagnostic criteria for hematuria and proteinuria are as follows: 1) During the first 6 months of the IgAV (HSP) disease course, hematuria and/or proteinuria occur. The diagnostic criteria for hematuria and proteinuria are as follows: 1) Hematuria: visible hematuria or 3 or more microscopic hematuria red blood cells per high-power field (HP) within 1 week. 2) Proteinuria: satisfy any of the following: urine qualitative test shows positive for protein 3 times within 1 week; 24-hour urine protein quantification > 150mg or urine protein/creatinine (mg/mg) > 0.2; urine microalbumin is higher than the normal value 3 times within 1 week.

1.2 Case Data

Prepare the case report form and collect the epidemiological data of the research subjects, such as gender, age, date of birth, and admission time; screen the auxiliary test results of the research subjects at their first admission, including peripheral blood eosinophil count (EO#), platelet count (PLT), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), serum total immunoglobulin E (tIgE), serum uric acid (UA), fecal occult blood(OB), *Mycoplasma pneumoniae* immunoglobulin M antibody (MP-IgM), anti-*Streptococcal* hemolysin O (ASO), urine routine, 24-hour urine protein quantification, and urine microalbumin/creatinine ratio determination.

1.3 Statistical Analysis

This study was completed using the IBM SPSS Statistics 23 professional statistical analysis software. Quantitative data that followed a normal distribution were expressed as mean ± standard deviation ($\bar{x}\pm$ SD), while quantitative data that did not follow a normal distribution were expressed as median (interquartile range, P25–P75). Categorical indicators described the number and percentage of each category. Comparisons between two groups of quantitative data that followed a normal distribution were conducted using t-tests, while comparisons between two groups of quantitative data that did not follow a normal distribution were conducted using the Mann-Whitney U test. Count data were compared using the x^2 test or Fisher's exact probability method. After baseline comparison, indicators with statistically significant differences between the two groups were subjected to Logistic regression analysis, and the ROC curve was drawn and the AUC was calculated. The optimal cut-off value was at the point where the Youden index was the maximum, and its sensitivity and specificity were calculated. Statistical tests were all conducted using two-tailed tests, and a P value < 0.05 was considered to indicate statistically significant differences in the tested variables.

2. Results

2.1 Baseline Data Comparison

Among the 204 children included in the study, 122 were boys (81 with nephritis and 41 without), and 82 were girls (56 with nephritis and 26 without). The average age was 103.27 ± 39.09 months. The average age of the IgAVN group was 107.36 ± 41.23 months, and that of the IgAV group was 94.91 ± 33.03 months. The analysis results showed that the age of the IgAVN group was significantly greater than that of the IgAV group (P < 0.05), and the UA of the IgAVN group was significantly higher than that of the IgAV group (P < 0.05). There were no statistically significant differences in gender, OB, MP-IgM, CRP, ASO, PLT, EO#, ESR, and tIgE between the two groups. (Table 1)

Table 1 Comparison of Baseline Data between the Nephritis Group and the Group without Nephritis

Items	IgAVN (n=137)	IgAV (n=67)	t/Z/χ2/F/U	P
Gender[n (%)]	Male 81 (59.12)	Male 41 (61.19)	0.080	0.777
	Female 56 (40.88)	Female 26 (38.81)		
Age (months)	107.36±41.23	94.91±33.03	-2.324	0.021*
OB[n (%)]	Positive 19 (15.45)	Positive 10 (18.87)	0.315	0.575
Missing data n=28	Negative 104 (84.55)	Negative 43 (81.13)		
MP-IgM[n (%)]	Positive 53 (59.55)	Positive 26 (72.22)	1.770	0.183
Missing data n=79	Negative 36 (40.45)	Negative 10 (27.78)		
CRP[mg/L:n(%)]	□10□123(90.44)	□10□60(89.55)	-0.241	0.810
Missing data n=1	10-20□8(5.88)	10-20□3(4.48)		
IgAVN (n=136)IgAV (n=67)	□20□5(3.68)	□20□4(5.97)		
ASO[IU/mL:n(%)]	□53□72(79.12)	□53□32(65.31)	-1.842	0.065
Missing data n=64	53-100□9(9.89)	53-100□6(12.245)		
IgAVN (n=91)IgAV (n=49)	100-200□5(5.49)	100-200□4(8.16)		
	200-400□1(1.10)	200-400□6(12.245)		
	□400□4(4.40)	□400□1(2.04)		
PLT($\times10^9$/L)	328.00(267.00~405.50)	319.22±85.17	3.208	0.075
EO# ($\times10^9$/L)	0.16(0.09~0.25)	0.15(0.08~0.24)	4309.500	0.973
ESR (mm/h)	18.00(10.00~36.75)	16.00(9.00~25.00)	1397.500	0.359
tIgE(IU/mL)	69.00(25.00~224.50)	47.50(21.00~129.25)	3075.500	0.094
UA(μmol/L)	301.50(247.00~354.75)	248.01±58.56	2592.500	0.000*

Abbreviations: IgAVN: IgA Vasculitis Nephritis; IgAV: IgA Vasculitis; MP-IgM: Pneumonia Mycoplasma IgM Antibody; CRP: C-reactive Protein; ASO: Anti-Streptolysin O; PLT: Platelet Count; EO#: Eosinophil Count; ESR: Erythrocyte Sedimentation Rate; tIgE: Total IgE; UA: Uric Acid; *P<0.05.

2.2 Logistic Regression Analysis

Including the indicators with differences in baseline data in the Logistic regression analysis showed that UA was an independent risk factor for IgAVN occurrence, with an OR value and its 95% CI of 1.010 (1.005, 1.015), and the OR value was greater than 1. (Table 2)

Table 2 Logistic Regression Analysis Results of Blood Source Indicators and IgA vasculitis nephritis

						95% CI of EXP(B)	
Items	B	S.E.	Wald	dfP	Exp(B)	Lower Bound	Upper Bound
Age(months)	0.002	0.005	0.160	1 0.689	1.002	0.993	1.011
UA	0.010	0.002	16.802	1 0.000*	1.010	1.005	1.015
constant	-2.313	0.680	11.566	1 0.001	0.099		

Abbreviations: UA: Uric Acid; *P<0.05.

2.3 Receiver Operating Characteristic (ROC) Curve

The ROC curve analysis showed that the AUC of UA for predicting IgAVN was 0.711 (P < 0.05), with a 95% CI of (0.639, 0.783), a sensitivity of 64.2%, a specificity of 74.6%, and the optimal cut-off value was 277 μmol/L. (Table 3, Fig.1)

Table 3 Area Under the Receiver Operating Characteristic Curve of the Subjects

				Asymptotic 95% CI	
Outcome Variable	AUC	S.E.[a]	Asymp. Sig.[b]	Lower Bound	Upper Bound
UA	0.711	0.037	0.000*	0.639	0.783

a.Under non-parametric assumptions; b. Null hypothesis: the actual area = 0.5; *P < 0.05.

3. Conclusion

This study found that there were significant differences in age and UA levels between the IgAVN group and the IgAV group. Among them, higher UA levels were an independent risk factor for IgAVN and had certain predictive value for IgAVN, with high specificity.

4. Discussion

Uric acid (UA) is the final oxidative product of purine metabolism. This study provided additional evidence that elevated serum UA levels are an independent risk factor for IgA vasculitis nephritis (IgAVN) in children. The underlying mechanism may involve the accumulation of UA, which leads to vascular endothelial dysfunction, lipid metabolism disorders, oxidative stress, and inflammatory responses[14-20], thereby contributing to the development of various cardiovascular diseases, metabolic syndrome, and chronic kidney disease. Some researchers propose that excessively high UA concentrations in the renal tubules can cause UA to precipitate, forming microcrystalline deposits that stimulate renal tubular

epithelial cells. Supporting this hypothesis, Saglam M K et al. reported that among 61 children with simple hyperuricemia, 35 exhibited a notable number of small high-echo spots in their renal calyces[21].

Current research has extensively elucidated the signaling pathways through which UA induces vascular endothelial damage: UA activates the *NLRP3*/IL-1β signaling pathway via reactive oxygen species generation and potassium ion efflux, thereby triggering assembly of the *NLRP3* inflammasome—a cytoplasmic protein complex of the innate immune system. This activation promotes the release of IL-1β and IL-18, exacerbating inflammation in both blood vessels and kidneys[22]. In rat models under hyperuricemic conditions, kidney epithelial expression of *NLRP3*/ASC was upregulated, and both inflammasome-dependent caspase-1 activation and non-inflammasome-related P-Smad2/3 signaling were implicated in UA-induced epithelial-mesenchymal transition (EMT), inflammation, and fibrosis[23]. Furthermore, in hyperuricemia, long non-coding RNA (lncRNA)-*HOTAIR* promotes endothelial cell pyroptosis by competitively binding miR-22 to modulate *NLRP3* expression[24].

These findings have identified novel therapeutic targets for mitigating UA-induced vascular endothelial damage: the histone deacetylase (HDAC) inhibitor trichostatin A (TSA) or the selective HDAC6 inhibitor Tubastatin A (Tub A) alleviated UA-induced vascular endothelial cell injury (VECI) by upregulating fibroblast growth factor 21 (FGF21) expression and subsequently activating the PI3K/AKT pathway[24]. Additionally, UA can suppress the nuclear factor E2-related factor 2 (Nrf2) pathway, thereby inducing intracellular oxidative damage and inflammation; simvastatin (SIM) has been shown to counteract these effects by modulating the Nrf2 pathway, thus reversing UA-triggered oxidative stress and inflammatory exacerbation[19].

This study highlights both innovative aspects and clinical relevance: 1) Our findings, together with prior evidence, support a dual role for UA as a biomarker and a potential therapeutic target in pediatric IgA vasculitis (IgAV) management. Future clinical practice should emphasize monitoring and intervention targeting serum UA during treatment and follow-up of children with IgAV. For instance, incorporating agents that inhibit UA production or block *NLRP3* activation into standard IgAV therapy may yield additional clinical benefits and potentially slow or prevent the onset and progression of IgAVN. 2) Given the mechanistic insights presented here, serum UA management may also be critically important in other vascular diseases beyond IgAV, warranting the application of these principles to broader research on vascular pathologies. 3) All data in this study were objective indicators extracted directly from the medical record system, excluding subjective clinical symptoms; such laboratory-based metrics are unaffected by observer bias and thus enhance data reliability. 4) The selected variables represent routine diagnostic tests for IgAV patients and exclude supplementary experimental biomarkers. This approach ensures wide applicability without increasing testing costs and facilitates clinical translation for frontline diagnosis and treatment.

Nevertheless, this study has several limitations: 1) As a single-center retrospective analysis with a limited sample size, our findings require validation in a larger, multicenter prospective study—planned by our research group—to improve statistical power and overall evidence quality. 2) The current analysis

included only a subset of potential indicators and omitted certain risk factors identified in earlier studies as well as recently discovered immune markers; future prospective work will incorporate expanded biomarker panels and integrate basic research approaches, such as animal models, to identify additional prognostic factors and elucidate underlying molecular mechanisms in IgAV. 3) Due to CRP and ASO values exceeding assay detection limits in some cases, categorical rather than continuous quantitative analysis was employed, potentially introducing bias. 4) Data were collected only from the early disease phase and did not capture long-term outcomes; all subjects were inpatients, and post-discharge follow-up—often conducted in outpatient settings or at other institutions—was not included, potentially affecting outcome classification (e.g., presence or absence of kidney injury). 5) Clinical manifestations of IgAVN do not always correlate with the severity of renal histopathological damage, which more accurately reflects lesion severity and long-term prognosis; however, renal biopsy data were not incorporated into our statistical analysis. In upcoming studies, our team plans to retrieve pathological results from our institutional biological specimen bank to enable more precise assessment of renal injury.

In conclusion, serum UA levels among blood-derived indicators in patients with IgA vasculitis (IgAV) demonstrate significant predictive value for IgAV nephritis (IgAVN). Therefore, elevated UA levels in children with IgAV should prompt close monitoring of kidney function to facilitate early detection and timely intervention for renal injury, thereby improving clinical outcomes. Further clinical and basic research is warranted to develop a robust predictive model for IgAV-related kidney damage.

Abbreviations

AUC
Area Under the Curve
ASO
Anti-*Streptococcal* Hemolysin O
ASC
Apoptosis-associated Speck-like Protein Containing a CARD
CI
Confidence Interval
CRP
C-Reactive Protein
EO#
Eosinophil Count
ESR
Erythrocyte Sedimentation Rate
EMT
Epithelial-Mesenchymal Transition
FGF21
Fibroblast Growth Factor 21

HSP
Henoch-Schönlein Purpura
HP
High-Power Field
HDAC
Histone Deacetylase
IgA
Immunoglobulin A
IgAV
Immunoglobulin A Vasculitis
IgAVN
Immunoglobulin A Vasculitis Nephritis
IL
Interleukin
lncRNA
Long non-coding RNA
MP-IgM
Mycoplasma pneumoniae Immunoglobulin M Antibody
NLRP
NOD-like Receptor Family Pyrin Domain Containing
Nrf2
Nuclear Factor E2-related Factor 2
OR
Odds Ratio
OB
Fecal Occult Blood
PLT
Platelet Count
PI3K/AKT
Phosphatidylinositol 3-kinase/Protein Kinase B
ROC
Receiver Operating Characteristic
SIM
Simvastatin
tIgE
total Immunoglobulin E
TSA
Trichostatin A
Tub A
Tubastatin A

UA
Uric Acid
VECI
Vascular Endothelial Cell Injury

Declarations

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Competing Interests

The authors declare no financial or non-financial interests relevant to this work to disclose.

Author Contributions

LIU Li and HOU Mengwei contributed equally to this work. LIU Li contributed to the conception and design of the study. Material preparation, data collection, and analysis were performed by LIU Cui and HOU Mengwei. The first draft of the manuscript was written by YANG Yonghua, and all authors commented on previous versions of the manuscript. All authors have read and approved the final manuscript.

Ethics approval

This study was conducted in accordance with the principles of the Declaration of Helsinki. Specifically, ethical approval was obtained from the Bioethics Committee of the Medical Department of Xi’ an Jiaotong University (approval date: 2022-02-25, approval number: 2022-304).

Consent to participate

This is a retrospective observational study and does not contain any sensitive personal information such as names and addresses. Therefore, ethical approval was obtained from the Bioethics Committee of the Medical Department of Xi’ an Jiaotong University, and the need for informed consent was waived.

Consent to publish

The manuscript does not contain any personally identifiable information, such as images or videos. As a result, informed consent for publication from participants was not required.

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LIU Li and HOU Mengwei contributed equally to this work.

LIU Li (Attending Physician, Department of Pediatrics, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, China): Provided critical guidance during the conception and design phases of the study.

HOU Mengwei (Senior Network Information Engineer, Network Information Department, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, China): Assisted with data retrieval and the removal of sensitive information.

WU Yue (Associate Chief Physician, Cardiovascular Medicine Department, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, China): Contributed to the revision of the article.

LIU Xiaohong and HUANG Yanping (Chief Physician, Department of Pediatrics, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, China): Reviewed the professional content of the article and made thorough revisions.

YANG Yonghua (Associate Chief Physician, Department of Pediatrics, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, China): Involved in the drafting of the manuscript and critically revised it for important intellectual content.

LIU Cui (Attending Physician, Department of Pediatrics, Xi'an International Medical Center Hospital, Xi'an, Shaanxi, China): Participated in data analysis and manuscript writing.

The First Affiliated Hospital of Xi'an Jiaotong University (Xi'an, Shaanxi 710061, China): Supplied the case data and the results of auxiliary examinations.

All contributors have reviewed and approved the manuscript. Individuals who did not contribute substantially are not listed. The authors declare no conflicts of interest. Data will be made available upon request.

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Figures

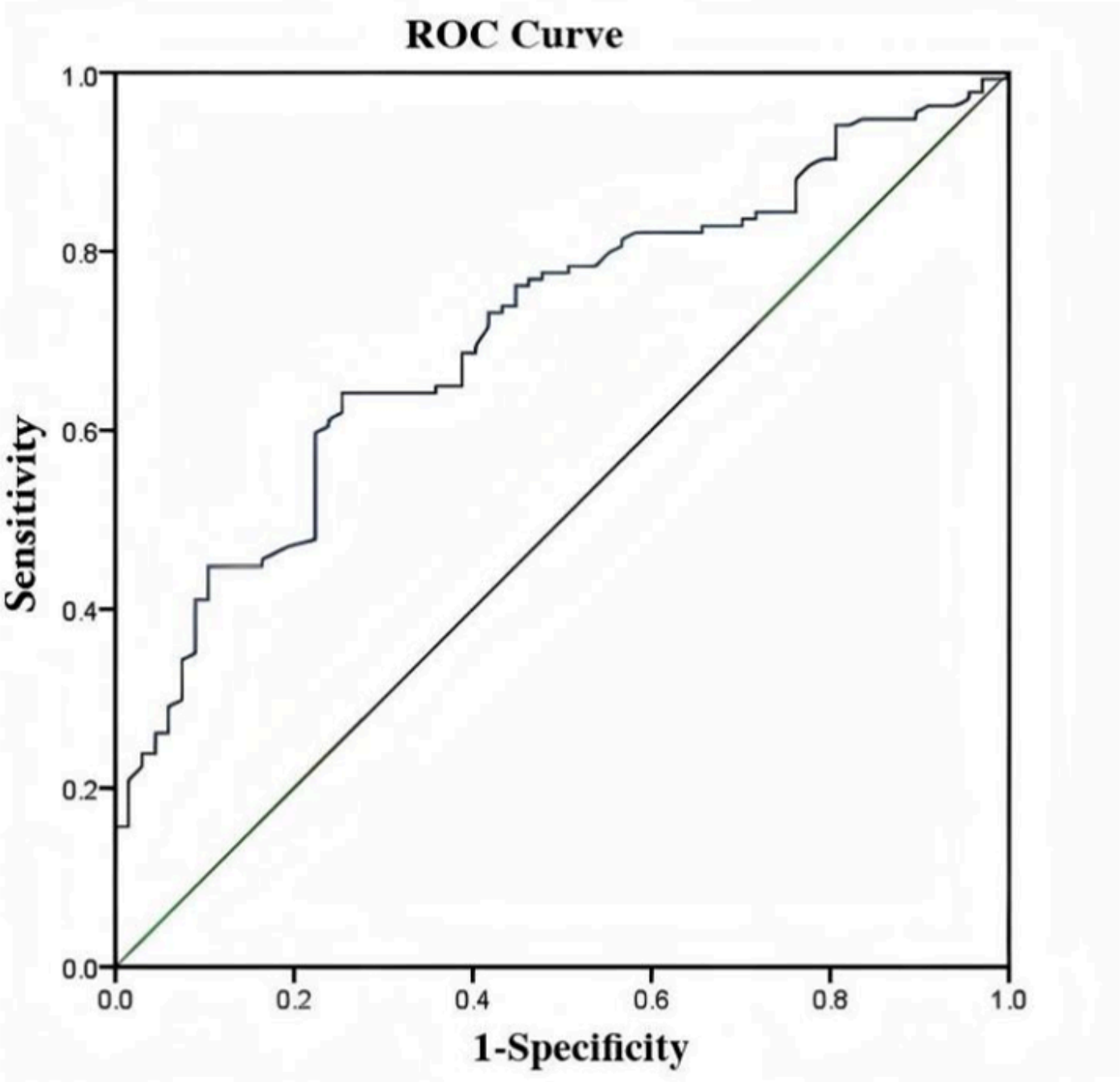


Figure 1

Receiver Operating Characteristic Curve for Predicting IgA Vasculitis Nephritis by Uric Acid

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- SupplementaryMaterial.zip