A retrospective descriptive review of children diagnosed with Henoch–Schönlein purpura at Red Cross War Memorial Children's Hospital over a 5-year period (2015 - 2019)

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Background. Limited data are available on the prevalence, variation in clinical features and outcomes of IgA vasculitis in low–middle-income countries.
Objective. To describe IgA vasculitis cases encountered at a South African (SA) children's hospital.
Methods. A retrospective review of patients with a discharge diagnosis of IgA vasculitis at Red Cross War Memorial Children's Hospital between 2015 and 2019. Patient demographics, clinical characteristics, laboratory findings, management and short-term outcomes were summarised using conventional descriptive statistics.
Results. The mean age of the study sample (*N*=49) was 77 months; male-to-female ratio was 1:1. Rash presented in 48 children (97%), arthralgia in 41 patients (84%) and abdominal pain in 18 patients (37%). Oedema manifested as scrotal oedema in one patient and as facial oedema in three cases. Kidney involvement was evident in 26 cases (53%), with associated proteinuria or haematuria, while isolated microscopic haematuria occurred in six patients (12%). Complications were infrequent: five patients (10%) had IgA nephritis on biopsy and one (2%) had a gastrointestinal bleed. The mean (standard deviation) length of hospital stay was 1.6 (2) days. At one year of follow-up, two patients (4%) had persistent proteinuria; only one patient (2%) still had haematuria.
Conclusion. The clinical course of IgA vasculitis in this cohort of SA children was mostly self-limiting, consistent with international literature. However, patients with persistent haematuria or proteinuria required longer-term follow-up. Collaborative studies in SA and sub-Saharan Africa may provide a more accurate picture of the epidemiology of childhood IgA vasculitis and its complication rates.

Keywords: IgA vasculitis, Henoch–Schönlein purpura, HSP, children, proteinuria, Africa

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Since its initial description in the early 1800s, IgA vasculitis (previously known as Henoch–Schönlein purpura) has become recognised as the most prevalent childhood vasculitis, currently having a steady annual incidence rate of between 3 and 27 cases per 100 000.[1] The classification of IgA vasculitis has been revised multiple times since the 1990 version set by the American College of Rheumatology. The most recent classification, referred to as the EULAR/PRINTO/PRES 2010 classification,[2] has been reinforced by a 2019 update from the European Single Hub and Access point for paediatric Rheumatology in Europe (SHARE).[3]

IgA vasculitis is a systemic small-vessel vasculitis of unknown aetiology, which typically presents as a triad of palpable purpura, arthralgia, abdominal pain and sometimes haematuria, and may be present in children. Atypical presentations are rare and often reflect a complication of the disease course.[4] The hallmark of IgA vasculitis is the deposition of IgA1-predominant immune deposits along affected vessel walls in the skin, gastrointestinal mucosa, joints and glomeruli. Other areas of the body may also be affected, although this is rare. To date, no causative genetic mutations of IgA vasculitis have been identified, although genetic mapping does suggest a strong involvement in the *HLA-DQA1/DQB1* region.[5] Age <10 years and a preceding upper respiratory tract infection are consistent risk factors for IgA vasculitis, although the mechanism by which these factors contribute to pathogenesis has yet to be comprehensively defined.[6] IgA vasculitis predominantly follows a self-remitting course, typically occurring between the ages of 2 and 10. Long-term complications are rare, but when these do occur, they are almost exclusively due to kidney involvement.[7] The degree of kidney involvement with proteinuria or haematuria differs between cohorts, often ranging from 30% to 50%. Despite progression to permanent kidney involvement occurring in less than 1% of cases, the possibility is of prognostic importance.[8]

The clinical course of IgA vasculitis does not seem to vary greatly between high- and low–middle-income countries (LMICs), although some literature suggests IgA vasculitis may present with more severe symptoms in LMICs and be more common in European and Asian populations. Owing to the scarcity of data from LMICs, and South Africa (SA) in particular, it is difficult to demonstrate the prevalence of the condition.[9] In SA, most epidemiological data on IgA vasculitis and its associated complications appear to have been inferred from

studies on the prevalence of chronic kidney failure or from kidney biopsy reviews.[10,11] A recent publication by Scott *et al.*[12] highlighted the lack of data on paediatric rheumatic conditions in LMICs and also reported IgA vasculitis as the most common type of childhood vasculitis.

Healthcare services in LMICs are often resource constrained and face socioeconomic challenges that differ from those in high-income countries; this may be important if disease complications extend into chronicity and add further strain onto healthcare resources. The aim of the study was to address a knowledge gap by describing the epidemiology and short-term outcomes of children with IgA vasculitis at a large public quaternary children's hospital.

Methods

Study design

This was a retrospective observational study at Red Cross War Memorial Children's Hospital (RCWMCH) in Cape Town, SA.

Study population and setting

RCWMCH is a quaternary hospital that provides dedicated healthcare to children and adolescents in SA's Western Cape province. Most of the children accessing or referred for care at the facility come from low socioeconomic urban and peri-urban communities. However, patients may also come more widely from other provinces in SA and neighbouring countries to access comprehensive paediatric services that focus on subspecialities. Most children whose records were included in the study were managed through the general paediatric service component at the hospital.

Records from all children who met the EULAR 2010 classification criteria for IgA vasculitis and who presented at the hospital between 1 January 2015 and 31 December 2019 were included in the study. Cases for whom records were unavailable or if vasculitis was found not to be due to Henoch–Schönlein purpura were excluded from the study.

Definitions

For the purposes of this study, kidney involvement included cases with haematuria or proteinuria, both defined as the relevant bedside urine dipstick test showing a value of between 1+ (30 mg/dL) and 3+. Acute kidney injury is defined as a serum creatinine increase of ≥0.3. mg/dL (26.5 µmol/L) or an increase of ≥1.5 to 1.9 times the baseline level (Stage 1). An increase in serum creatinine of between 2.0 and 2.9 times the baseline level is defined as Stage 2.[13]

Grading classifications set out by the International Study of Kidney Disease in Children[8] were used for kidney biopsy results, as follows:

- Grade I – minimal mesangial proliferation without crescents or necrosis
- Grade II – mesangial proliferation with focal crescents or necrosis involving <50% of glomeruli
- Grade III – mesangial proliferation with diffuse crescents or necrosis involving >50% of glomeruli
- Grade IV – global sclerosis involving >50% of glomeruli.

Data collection

The hospital information system database known as Clinicom® was used to identify eligible patients (both inpatients and outpatients) seen at the facility between 1 January 2015 and 31 December 2019 based on their discharge coding diagnosis. The search was broadened to look for cases diagnosed as Henoch–Schönlein purpura, the alternative name for IgA vasculitis.

All identified files were retrieved from the hospital's medical records department and clinical notes were reviewed based on the 2010 EULAR criteria. Files that met the stipulated criteria for IgA vasculitis were included in the analysis (Fig. 1).

Demographic details (including sex, age and nutritional status), clinical features at presentation, dates of admission and discharge, investigations performed, treatment, complications and follow-up outcomes were recorded in a data sheet for each patient.

Statistical analysis

Data were analysed using the STATA statistical software package, release 16 (StataCorp, USA). Patient demographics, clinical characteristics, laboratory findings, management and short-term outcomes were summarised and presented in tables.

Conventional descriptive and inferential statistical methods were used to analyse the dataset. Normality was evaluated using the Shapiro–Wilks method for continuous variables. For continuous variables, means (and standards deviations, SD) or medians (and interquartile ranges, IQR) were reported, as appropriate. Categorical variables were shown as proportions and percentages. Associations between categorical variables were explored using the chi-square and Fisher's exact test, as appropriate. A significance level of $p<0.05$ was used.

Significant associations between kidney involvement and categorical variables such as rash, abdominal pain or arthritis were investigated.

Ethical considerations

Given the inherent challenge of obtaining individual consent retrospectively, an institutional waiver was granted by the hospital research committee. Stringent measures were implemented to anonymise patient data and ensure confidentiality. The collected data were stored securely and accessible only to the researchers.

The study was approved by the Human Research Ethics Committee of the Health Sciences Faculty, University of Cape Town (ref. no. 652/2021) and the National Department of Health, South Africa (ref. no. RXH-RCC 312 WC_202111_015).

Results

Demographic data

Table 1 gives a summary of the sociodemographic characteristics of the study sample (N=49). The male-to-female ratio was 1:1. Most patients (n=28; 57%) were between 5 and 10 years old. Forty-one (84%) study participants had a normal weight-for-age classification, with four children being underweight and four overweight for their age.

The annual incidence of cases varied between 0.4 per 100 000 and 3.6 per 100 000 new patient visits at our institution over the course of the study period.

Clinical features at presentation

Almost all patients had palpable purpura (n=48, 97%) at presentation (Table 2); 41 patients (84%) were tested for thrombocytopenia. Joint involvement was the second most common manifestation (n=41; 84%) at presentation, followed by abdominal symptoms such as pain and nausea or vomiting. The complete triad of purpura, abdominal pain and arthritis at presentation was seen in 15 patients (31%).

Oedema was an uncommon finding, with scrotal oedema seen in one patient and facial oedema of the forehead in three others. Six children (12%) presented with isolated microscopic haematuria.

One 6-year-old had gastrointestinal bleeding; he presented with a typical rash, abdominal pain, oedema of the forehead and passing

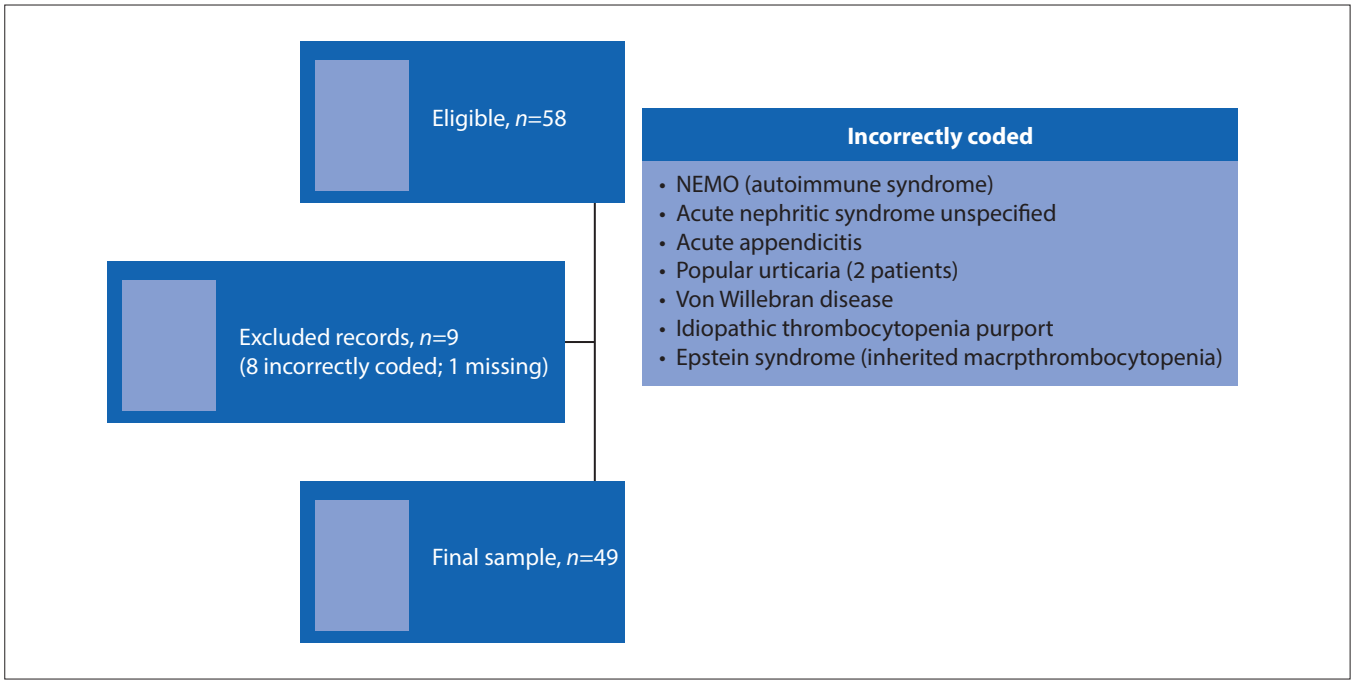

Fig. 1. Flow diagram showing participant recruitment.
(NEMO = nuclear factor kappa B essential modulator deficiency syndrome.)

Table 1. Demographic data of the study sample (*N*=49)

Variable	*n* (%)
Sex	
Male	24 (48)
Female	25 (52)
Age categories	
1 - 5 yr	15 (30)
5 - 10 yr	28 (57)
>10 yr	6 (13)
Age (months), mean (SD, range)	77 (31, 30 - 155*)
Nutritional status	
Mean weight-for-age (Z-score), (SD, range)	–0.13 (1.32, - 3 to 2.9)
Length of hospital stay (days), mean (SD)	1.6 (2)

SD = standard deviation.
*Hospital admission policy during the study period was that only new patients <13 years of age were seen at the study site.

Table 2. Clinical features of IgA vasculitis at presentation (*N*=49)

Clinical feature	*n* (%)
Preceding upper respiratory tract infection	20 (41)
Fever	6 (12)
Skin manifestation	
Palpable purpuric rash	48 (97)
Joint involvement	
Arthritis/arthralgia	41 (84)
Abdominal symptoms	
Abdominal pain	18 (37)
Gastrointestinal bleed	1 (2)
Nausea/vomiting	11 (23)
Kidney involvement	
Haematuria (isolated)	6 (12)
Proteinuria (isolated)	6 (12)
Proteinuria and microscopic haematuria	6 (12)
Localised oedema	4 (8)
Angioedema	3 (6)
Scrotal oedema	1 (2)

scanty, bloody mucoid stools. As his admission progressed, he also developed arthritis and haematuria. This patient had not received any non-steroidal anti-inflammatory drugs.

At initial presentation, 35 patients (71%) required hospital admission, of whom 31 (89%) were treated in the paediatric short-stay ward, two (6%) in the paediatric nephrology ward owing to kidney involvement, and two (6%) in the paediatric surgical ward, for severe abdominal pain and severe arthritis, respectively. The 14 patients who were not admitted were managed in the paediatric outpatient department.

Six patients had abdominal ultrasound scans, of whom two also had abdominal X-rays; all imaging findings were within normal limits.

Table 3 summarises the laboratory findings of patients for whom blood test results were available. White cell counts were not elevated and there was no evidence of thrombocytopenia; mean platelet count across the cohort was 428 × 10^9/L.

Kidney involvement

Kidney involvement, in the form of proteinuria or haematuria according to bedside dipstick urinalysis, was seen in 26 patients (53%), all of whom were followed up. Two patients had transient acute kidney injury, which resolved during admission.

One child, a 10-year-old male who initially presented with a rash and arthritis but normal kidney function, progressed to chronic kidney disease (2+ haematuria and 2+ proteinuria on dipstick urinalysis) 1 month after discharge, with an estimated glomerular filtration rate of 50 - 60 mL/min/1.73 m^2). He was followed up by the nephrology service. In addition, 15 children who were seen in the general outpatient clinic for routine follow-up were referred to

the paediatric nephrology service because of persistent haematuria or proteinuria.

Five children's records showed kidney biopsies for persistent proteinuria or haematuria; two of these children also had hypertension (Table 4). The length of time between diagnosis and a biopsy varied, with the earliest being 4 months after diagnosis and the latest being 4 years after diagnosis. Results were graded according to the International Study of Kidney Disease in Children classifications.

Treatment during the acute phase of admission in the short-stay ward

Analgesia

Most patients received simple analgesics for joint or abdominal pain; 36 (74%) received paracetamol and 21(43%) were given ibuprofen as add-on pain treatment. Three patients (6%) were given a weak opioid (tilidine) and one (2%) received oral morphine.

Immunosuppressants

Six patients managed by the paediatric nephrology service received some form of immunosuppression other than steroids during their clinical course, with the indication consistently being for IgA nephritis on biopsy or persistent haematuria. Five patients received mycophenolate mofetil and one received azathioprine. Tacrolimus was used as a second agent in two cases. Sixteen patients received oral prednisone for abdominal pain or arthritis.

Follow-up period

The mean (SD) length of hospital stay was 1.6 (2) days, although 10 patients (20%) required readmission, mostly owing to moderate or severe abdominal pain or arthritis. Mean (SD) time to readmission was 3.8 (2.4) days.

Table 3. Laboratory findings across patients presenting with IgA vasculitis (*N*=49)

Parameter	
White blood cell count (10^9/L), mean (SD), *n*=41	10.7 (3.7)
Platelet count (10^9/L), mean (SD), *n*=41	428 (99)
Urea (mmol/L), mean (SD), *n*=40	3.9 (1)
Creatinine (mmol/L) mean (SD), *n*=41	32 (7.5)
Serum complement C3 (g/L), mean (SD), *n*=12	1.5 (0.37)
Serum complement C4 (g/L), mean (SD), *n*=10	0.28 (0.11)
KDIGO* stage 1	2

SD = standard deviation.
*Kidney Disease: Improving Global Outcomes Stage 1: Serum creatinine increase of ≥0.3 mg/dL (26.5 µmol/L) or an increase of ≥1.5 - 1.9 times the baseline level.

No deaths were recorded in the cohort. The duration and site of follow-up varied, and were based on the discretion of the discharging physician. Some patients were referred to their local health facility and some were followed up at the study site; durations could not be categorised as <6 months or >6 months.

One year after discharge, nine children were still being monitored: two had persistent proteinuria, one had persistent haematuria, and six had recovered fully. Among the five children diagnosed with IgA nephritis on kidney biopsy, two had persistent proteinuria, two had recovered, and one had persistent haematuria.

A statistically significant association was found between abdominal pain and kidney involvement (p=0.041); however, no significant associations were found between kidney involvement and the presence of a rash (p=1.000), arthritis (p=1.000) or the combination of rash, abdominal pain and arthritis (p=0.205).

Discussion

Our study showed that IgA vasculitis is a relatively common paediatric condition. We found that most patients presented with the classic skin rash, 84% with arthritis and rash, and 31% with the triad of symptoms; kidney involvement was common although most cases were self-remitting without long-term complications. This is one of only a few epidemiological reports on IgA vasculitis in children in Africa, an important issue owing to the possibility, although uncommon, of long-term kidney disease, which may require additional resource allocation.

Our analysis showed that only one patient progressed to chronic kidney disease (estimated glomerular filtration rate <60 mL/min/1.73 m^2) and therefore higher stages of chronic kidney disease are less likely to be a significant problem than, for example, obstructive uropathy in children.

There are no comparable data on annual incidence of IgA vasculitis in LMICs. However, data from high-income countries show annual population incidence rates of 3.5 per 100 000 (Japan) and 26.7 per 100 000 (Scotland), with the highest rate reported among 4 - 6-year-olds in the UK (70.3 per 100 000).[14]

Our sample showed an even male-to-female ratio, whereas other studies' results generally point to a male predominance.[15] The mean age of study participants was similar to that seen in studies from high-income countries,[9] with 87% of our sample being younger than 10 and no patient being under 2 years of age.

Data on IgA vasculitis in Africa are scarce.[12] Given that most data are inferred from studies investigating the prevalence of chronic kidney failure or kidney biopsy reviews,[10,11] epidemiological data on the prevalence of IgA vasculitis and associated risk factors for complications are needed. Naija *et al.*[16] found 68 cases of IgA vasculitis over a 15-year period in a study at a university hospital in

Table 4. Kidney biopsy results in children with IgA vasculitis (*n*/*N*=5/49)

Age (years)	Sex	Diagnosis	Biopsy	Biopsy result*	Proteinuria/ haematuria†	Complications	Status at last follow-up
10	Male	August 2018	June 2020	Grade I	2/2	Hypertension	3+ proteinuria
7	Male	January 2018	April 2018	Grade IV	2/2	Hypertension	Recovered
6	Female	January 2019	May 2019	Grade I	2/3	-	Transferred to private facility
8	Male	June 2015	August 2018	Grade II	2/2	-	2+ proteinuria
5	Male	November 2016	November 2020	Grade III	3/3	-	Recovered

*International Study of Kidney Disease in Children grading classification:[13] Grade I – minimal mesangial proliferation without crescents or necrosis; Grade II – mesangial proliferation with focal crescents and/or necrosis involving <50% of glomeruli; Grade III – mesangial proliferation with diffuse crescents and/or necrosis involving >50% of glomeruli; Grade IV – global sclerosis involving >50% of glomeruli.
†Bedside urinary dipstick test 1+ (corresponds to 30 mg/dL) for haematuria and 1+ (corresponds to 30 mg/dL) for proteinuria.

Tunisia, and described IgA nephritis in 34 patients, of whom 14 had biopsy confirmation.

Bacteria, viruses and protozoa have all been implicated as triggers for IgA vasculitis, especially group A streptococci and parvovirus B.[6] Acute upper respiratory tract infection as a potential trigger for IgA vasculitis is suggested in our study, with a history of such an infection prior to presentation documented in 41% of cases. Similarly, in a study in Italy that involved 219 children, Coppo *et al.*[17] recorded a preceding upper respiratory tract infection in 41% of their sample. A systematic review by Hetland *et al.*[9] also concluded that upper respiratory tract infection often precedes IgA vasculitis cases.

Joint manifestation, seen in 41 patients (84%), was the second most common clinical feature of IgA vasculitis in our study. In a recent publication, in which records from 280 children in Italy were reviewed, Breda *et al.*[1] also found joint involvement to be the second most common feature; this is similar to findings by Reamy *et al.*,[18] who reported that up to 75% of children develop arthritis. The available literature reports that 5 - 25% of patients with IgA vasculitis may have joint manifestation preceding rash.[15] In our cohort, one patient presented with joint involvement only, contributing to diagnostic uncertainty until the development of the typical rash.

Gastrointestinal symptoms accounted for the third most common feature. Along with joint pain, these symptoms generally have a sudden onset; abdominal pain tends to be colicky in nature and may be associated with vomiting, and can even mimic an acute abdomen in its severity.[9,18] Intestinal complications such as gastrointestinal haemorrhage are rare, but have been reported; intussusception can also occur, with a mural haematoma serving as the lead point.[9,18] In our study, 18 patients (37%) had abdominal pain. One presented with gastrointestinal bleeding, thought to be superficial mucosal bleeding; no incidence of intussusception occurred. These children were more likely to receive steroid therapy. In a meta-analysis of 13 studies with close to 2 400 children across Japan, Korea, Spain, Brazil, Finland, Iran and Thailand, Chan *et al.*[19] found a significant association between abdominal pain and kidney involvement (defined as the presence of proteinuria, haematuria or blood cell casts). We also found a significant association between abdominal pain and kidney involvement.

Kidney impairment, which can lead to end-stage kidney disease, appears to be a key prognostic indicator for morbidity in IgA vasculitis. Narchi[20] estimated that the presence of both proteinuria and haematuria might indicate a 15% risk, whereas the combination of nephritic–nephrotic syndrome carries a risk of 50% for progressing to end-stage kidney disease. Close monitoring of patients with IgA vasculitis is therefore emphasised in order to identify those with kidney involvement and who may require referral to the paediatric nephrology service. The SHARE initiative recommends monitoring children by means of blood pressure measurements, early-morning urinalysis and estimated glomerular filtration rate to assess kidney function.[3]

Overall, 25 children (51%) in our cohort had kidney involvement, defined by the presence of microscopic haematuria at presentation; Breda *et al.*[1] reported kidney involvement in 37 out of 208 children (18%), with a mean age of 6.4 years across the group. In contrast, Pillebout *et al.*[21] reported 66% of a cohort of 50 children with IgA vasculitis to have kidney involvement. In a review of seven kidney biopsies of IgA nephritis at RCWMCH between 2005 and 2010, Sinclair[10] found that six had been within 6 months of diagnosis; at that time, there was no standardised immunosuppression regimen.

In a systematic review of 12 studies involving 1 133 children, Narchi[20] found that no long-term kidney impairment occurred in those with a normal urinalysis at 6 months, which suggests urinalysis needs to continue for 6 months in patients who have a normal urinalysis at presentation.

Treatment regimens in IgA vasculitis are controversial. Few randomised controlled trials have been conducted and in the absence of complications or kidney involvement treatment generally focuses on symptomatic relief of pain.[9] As there is no consensus on treatment in IgA nephritis, management is centre dependent and the decision for a kidney biopsy prior to treatment is made on a case-by-case basis given factors such as severity of the condition, acute kidney injury, nephrotic syndrome and initial response to treatment.[22] Recommendations from literature for administering corticosteroid immunosuppressants are based on a robust randomised controlled trial and are meant to reduce the intensity and duration of joint and abdominal pain, and not to prevent kidney disease.[18]

The use of corticosteroids in the symptomatic relief of abdominal and joint pain has been extensively documented in the treatment of IgA vasculitis.[22] Frequent use of steroids was observed in our study (n=23; 47%). Adjuvant symptomatic pain relief using simple analgesia was adequate for almost all patients.

Study limitations

Limitations of this retrospective descriptive study include the small sample size and limited comparable data on the annual incidence of IgA vasculitis in Africa and other LMICs. Furthermore, because IgA vasculitis is generally considered a benign condition, some children may have gone undiagnosed if seen at and discharged from a primary-care level or even when managed by district-level paediatricians.

The absence of a control group and the lack of randomisation in the study design limit the ability to establish causality and the ability to derive definitive conclusions about the risk factors for IgA vasculitis. In addition, the reliance on medical records for data collection may have introduced bias or incomplete information. As study participants were recruited based on the coded discharge data at the study site, there may have been cases of IgA vasculitis not accounted for during the study period owing to underreporting.[23]

Conclusion

Despite its limitations, the study adds to the limited literature on IgA vasculitis in LMICs as it was conducted in a quaternary hospital in SA that provides comprehensive paediatric service to a wide population of children and adolescents and used a standardised data capture sheet, ensuring consistent and accurate data collection. We were able to describe the epidemiology and short-term outcomes of children with IgA vasculitis at the facility, allowing for comparisons of descriptions of IgA vasculitis in other settings.

Our findings suggest that the clinical course of IgA vasculitis in SA is similar to that reported in other countries. Given prior research that has shown kidney involvement to be the most serious long-term outcome, prioritising children with kidney involvement for longer-term follow-up is emphasised. However, further research is needed to fully understand the pathogenesis and risk factors for kidney involvement in this population. Our study also highlights the need for continued research and surveillance of IgA vasculitis in LMICs to improve diagnosis, management and outcomes for affected children.

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1. Breda L, Carbone I, Casciato I, et al. Epidemiological and clinical aspects of immunoglobulin A vasculitis in childhood: A retrospective cohort study. Ital J Pediatr 2021;47(1):237.
2. Ozen S, Pistorio A, Iusan SM, et al. EULAR/PRINTO/PRES criteria for Henoch-Schönlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part II: Final classification criteria. Ann Rheum Dis 2010;69(5):798-806.
3. Ozen S, Marks SD, Brogan P, et al. European consensus-based recommendations for diagnosis and treatment of immunoglobulin A vasculitis-the SHARE initiative. Rheumatology (Oxford) 2019;58(9):1607-1616.
4. Grover N, Sankhyan N, Bisht JP. A five-year review of clinical profile in HSP. J Nepal Med Assoc 2007;46(166):62-65.
5. López-Mejías R, Carmona FD, Castañeda S, et al. A genome-wide association study suggests the HLA Class II region as the major susceptibility locus for IgA vasculitis. Sci Rep 2017;7(1):5088.
6. Heineke MH, Ballering AV, Jamin A, Ben Mkaddem S, Monteiro RC, Van Egmond M. New insights in the pathogenesis of immunoglobulin A vasculitis (Henoch–Schönlein purpura). Autoimmun Rev 2017;16(12):1246-1253.
7. Tracy A, Subramanian A, Adderley NJ, et al. Cardiovascular, thromboembolic and renal outcomes in IgA vasculitis (Henoch–Schönlein purpura): A retrospective cohort study using routinely collected primary care data. Ann Rheum Dis 2019;78(2):261-269.
8. Dyga K, Szczepańska M. IgA vasculitis with nephritis in children. Adv Clin Exp Med 2020;29(4):513-519.
9. Hetland LE, Susrud KS, Lindahl KH, Bygum A. Henoch-Schönlein purpura: A literature review. Acta Derm Venereol 2017;97(10):1160-1166.
10. Sinclair P. Henoch-Schönlein purpura – a review. Curr Allergy Clin Immunol. 2010;23(3):116-120.
11. Mitchell J. Descriptive study of biopsy proven IgA and Henoch-Schönlein purpura nephropathy in two government hospitals in Johannesburg (South Africa). Thesis. Johannesburg: University of the Witwatersrand, 2010. http://hdl.handle.net/10539/8794
12. Scott C, Sawhney S, Lewandowski LB. Pediatric rheumatic disease in lower to middle-income countries: Impact of global disparities, ancestral diversity, and the path forward. Rheum Dis Clin North Am 2022;48(1):199-215.
13. Kellum JA, Lameire N. Diagnosis, evaluation, and management of acute kidney injury: A KDIGO summary (Part 1). Crit Care 2013;17(1):204.
14. Watts RA, Hatemi G, Burns JC, Mohammad AJ. Global epidemiology of vasculitis. Nat Rev Rheumatol 2022;18(1):22-34.
15. Trapani S, Micheli A, Grisolia F, et al. Henoch Schönlein purpura in childhood: Epidemiological and clinical analysis of 150 cases over a 5-year period and review of literature. Semin Arthritis Rheum 2005;35(3):143-153.
16. Naija O, Bouzaraa J, Goucha-Louzir R, Lakhoua MR. [Predictive factors of severe Henoch-Schönlein nephritis in children: Report of 34 cases]. Tunis Med 2012;90(12):878-881.
17. Coppo R, Amore A, Gianoglio B. Clinical features of Henoch-Schönlein purpura. Italian Group of Renal Immunopathology. Ann Med Interne (Paris) 1999;150(2):143-150.
18. Reamy BV, Servey JT, Williams PM. Henoch-Schönlein purpura (IgA vasculitis): Rapid evidence review. Am Fam Physician 2020;102(4):229-233.
19. Chan H, Tang Y-L, Lv X-H, et al. Risk factors associated with renal involvement in childhood Henoch-Schönlein purpura: A meta-analysis. PLoS One 2016;11(11):e0167346.
20. Narchi H. Risk of long term renal impairment and duration of follow up recommended for Henoch-Schönlein purpura with normal or minimal urinary findings: A systematic review. Arch Dis Child 2005;90(9):916-920.
21. Pillebout E, Jamin A, Ayari H, et al. Biomarkers of IgA vasculitis nephritis in children. PLoS One 2017;12(11):e0188718.
22. Dudley J, Smith G, Llewelyn-Edwards A, Bayliss K, Pike K, Tizard J. Randomised, double-blind, placebo-controlled trial to determine whether steroids reduce the incidence and severity of nephropathy in Henoch-Schönlein purpura (HSP). Arch Dis Child 2013;98(10):756-763.
23. Daniels A, Muloiwa R, Myer L, Buys H. Examining the reliability of ICD-10 discharge coding in Red Cross War Memorial Children's Hospital administrative database. S Afr Med J 2021;111(2):137-142.

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