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Case-based Reviews in Pediatric Emergencies



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CHAPTER 4

Acute Glomerulonephritis

RN Srivastava, Shina Menon

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INTRODUCTION

Clinical features of gross hematuria, edema, and hypertension constitute the acute nephritic syndrome or acute nephritis. Varying degrees of oliguria and impairment of renal function are usually associated. In most cases, postinfectious acute glomerulonephritis (AGN), usually poststreptococcal, is the underlying etiology. Other, much less common, causative conditions include renal vasculitis (e.g., Henoch-Schönlein purpura), collagen vascular disorders, immunoglobulin A (IgA) nephropathy, and acute interstitial nephritis. Systemic features are often present in these disorders. Extensive laboratory investigations are usually not required in case of postinfectious AGN, which rapidly resolves with supportive management. In most other conditions and in patients with increasing renal dysfunction, detailed laboratory evaluation and a renal biopsy are performed and appropriate treatment carried out.

CASE 1

A 9-year-old girl was referred to pediatric renal clinic. She was perfectly well until 2 days back when she passed "light brownish red urine". There was no dysuria, flank pain, or fever. On the following day, she had complained of headache and was noted to have mild swelling on the face. She appeared to be passing lesser amounts of urine.

Her pediatrician had found her blood pressure (BP) to be raised to 140/90 mmHg. Urinalysis showed 2+ protein and microscopy revealed numerous red cells, red cell casts, and granular casts. Blood tests showed urea 90 mg/dL, creatinine 1.6 mg/dL, and potassium 5.2 mEq/L.

On inquiry, there was a history of mild sore throat about 10 days earlier that resolved over 3–4 days. No antibiotic was taken. There was no family history of a renal disorder. The parents were well educated.

Physical examination confirmed mild facial edema and hypertension. There was no other abnormality.

A chest X-ray film was normal. Blood samples were obtained for hemogram, total protein and albumin, cholesterol, antistreptolysin O (ASO) titer and C'3. The hemoglobin was 11.5 g/dL, leukocytes 11,056/mm^3, neutrophils 60%, protein 6.2 g/dL, and albumin 3.2 g/dL. Subsequent reports showed ASO titer 512 U and C'3 32 mg/dL.

A diagnosis of AGN was made. The parents were explained about the condition, the nature of kidney disease and the outcome and its treatment. She was managed on outpatient basis with close observation by the parents and her pediatrician.

She was advised rest at home, restriction of fluid-and-salt and protein, and control of hypertension with enalapril.

The urine output gradually increased, gross hematuria disappeared over the next 3 days and the weight declined by 1 kg. The levels of blood urea and creatinine were normal on day 7. Urine contained 1+ protein and red blood cells.

Normal diet and activity was gradually resumed.

The poststreptococcal etiology was confirmed by the findings of raised ASO titer and low C'3. These tests were repeated after a period of 3 months when ASO titer had declined and the C'3 level was within the normal range.

CASE REVIEW IN A NUTSHELL

This patient had presenting features typical of poststreptococcal AGN. It is important to make the correct assessment and avoid unnecessary laboratory investigations. Such patients do not need antibiotics or any specific medications. She was managed symptomatically and observed at home, as the parents were educated and understood the management with imposition of only the necessary restrictions. The condition improved quickly. It is important to be aware of the unusual presenting features. Patients with marked oliguria and profoundly deranged renal functions, severe hypertension need inpatient supportive care.

TOPIC REVIEW

Q. What is the etiology of the "syndrome" of acute nephritis?

Acute glomerulonephritis is mostly follows β-hemolytic streptococcal pharyngitis or pyoderma. It may, however, be caused by other organisms and be a part of systemic disorders affect the kidney (Box 1). Some of them (e.g., IgA nephropathy, acute interstitial nephritis) may have gross hematuria as the sole indicator of renal involvement.

Box 1: Etiology of acute nephritis

- Postinfectious
  - Bacteria: Group A b-hemolytic *Streptococcus* (most frequent cause), *Staphylococcus*, *pneumococcus*, *Salmonella typhi*. Specific conditions: Shunt nephritis,
  - bacterial endocarditis
  - Viruses: Hepatitis B, HIV, cytomegalovirus, rubella
  - Parasites: Malaria, filaria
- Systemic vasculitis
  - Henoch-Schönlein purpura, systemic lupus erythematosus, renal angiitis
- Primary glomerulonephritis
  - Immunoglobulin A nephropathy, membrano-proliferative glomerulonephritis.
- Acute interstitial nephritis

HIV, human immunodeficiency virus.

Q. What is its course and outcome?

Acute poststreptococcal glomerulonephritis carries an excellent prognosis. Renal biopsy is no longer carried out in these cases. Earlier studies have shown that even severe histological abnormalities resolve over a period of time with full recovery of renal function. The only exception is a particularly severe renal injury where glomeruli show extensive crescent formation (the histological lesion is termed as "crescentic glomerulonephritis"). These patients usually show mixed features of nephritis with massive edema, hypo-albuminemia, and severe derangement of renal function, which rapidly progresses. Therefore, patients who do not improve within a week are subjected to renal biopsy. Crescentic glomerulonephritis is managed with aggressive immunosuppressive therapy.

Q. How common is poststreptococcal acute glomerulonephritis?

In some parts of India PSAGN still remains common, whereas in northern regions its incidence has declined, and AGN is more often due to a condition other than streptococcal infection.

Q. What precisely is the cause of poststreptococcal acute glomerulo-nephritis?

Poststreptococcal AGN occurs following pharyngitis or skin infections caused by group A β-hemolytic streptococci. Strains of streptococci capable of producing AGN are called nephritogenic streptococci. Serotype M49 is most commonly associated with skin infections and 1, 4 and 25 with throat infections.

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Poststreptococcal AGN related to pyoderma often occurs in epidemics or clusters, being more common in hot and humid climates, whereas that following pharyngitis is predominantly observed in colder regions.

Q. What is its pathogenesis?

Glomerular injury in PSAGN results from deposition of immune complexes in the glomerular capillaries. Circulating immune complexes may get trapped in glomerular capillaries, or such complexes may form in situ from antibodies reacting with streptococcal antigens deposited in glomeruli or with some component of glomerular wall. The antigen responsible for PSAGN is not definitely known. Streptococcal M protein or/and endostreptosin (derived from streptococcal cell cytoplasm) have been suggested to play a role. Complement 3 (C3) activation in PSAGN is via the alternative pathway. The immune deposits consist of IgG, C3, properdin, and C5 and do not contain C1q and C4 (the classical pathway components). Serum C3 levels are decreased.

Q. What is its pathology?

The glomerular abnormalities are very characteristic. Glomeruli are enlarged and the lobular pattern is accentuated. Proliferative and exudative changes are uniformly distributed. There is proliferation of both endothelial and mesangial cells with obliteration of capillary lumen giving the glomeruli a "bloodless" appearance. Neutrophil infiltration is striking in early stages. The capillary basement membrane and the arterioles do not show significant abnormality, and tubular and interstitial abnormalities are absent or insignificant. In a very small proportion of cases, extensive crescentic changes are present. Immunofluorescence examination shows granular deposits of immunoglobulin G (IgG) and C3 along the capillary walls and in the mesangium. Electron microscopy shows electron dense subepithelial deposits or "humps" deposits. The histological abnormalities resolve rapidly and within 6–8 weeks, there is considerable decrease in exudative changes. Mild mesangial hypercellularity may persist for 1–2 years but eventually disappear.

Q. What is its clinical profile?

In most cases, there is a history of sore throat or pyoderma. The latent period is 7–14 days in the former and 2–4 weeks in the latter. Active or healed lesions of impetigo may be present when AGN develops. Poststreptococcal AGN usually affects children between the ages of 5–12 years and is rare below the age of 3 years. Gross hematuria and mild facial edema are the most common presenting features. Urine is typically reddish brown or smoky or cola-colored. Some degree of oliguria is usually associated, but anuria is infrequent and if persistent suggests rapidly progressive glomerulonephritis. If the fluid intake has been unrestricted, edema may increase to involve the hands and legs. Edema is turgid, unlike the flaccid swelling as in nephrotic syndrome. The child often complains of headache due to hypertension, which usually is mild to moderate and results from volume overload.

The severity of PSAGN is variable and mild cases may just have microscopic hematuria with slight proteinuria. Those at the other end of the spectrum may have oligo-anuria and severe hypertension.

Q. How about its atypical presentations?

The child may present with one or more of the complications of AGN. A history of sore throat and gross hematuria may be absent and the edema mild. Occasionally, urinalysis may not show significant abnormality. A correct diagnosis and prompt management are crucial.

- Acute pulmonary edema: Expansion of extracellular volume may lead to congestive heart failure and pulmonary edema. There is dyspnea and restlessness. The blood pressure is high but in later stages when heart failure and shock supervene, there may be hypotension. A chest X-ray film typically shows mild cardiomegaly and features of pulmonary edema. The condition may be

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wrongly diagnosed as bronchopneumonia or myocarditis
- Hypertensive encephalopathy: Children with AGN may develop hypertensive encephalopathy at comparatively lower levels of blood pressure. Drowsiness and convulsions may be the presenting features, and the illness resembles encephalitis. Elevated blood pressure and urine abnormalities confirm the diagnosis
- Acute renal failure: In occasional cases, oliguria-anuria and azotemia are the chief features and gross hematuria and edema may be absent.

Q. What should be the laboratory investigations in a suspected case?

Gross hematuria is present in a majority of moderate-to-severe cases. Proteinuria is usually mild, but occasionally in the nephrotic range. Microscopic examination shows dysmorphic red cells, red cell casts, and neutrophils. In initial stages, urine may contain a large number of neutrophils, which is often mistakenly regarded to indicate urinary tract infection. A fresh, uncentrifuged urine specimen should be examined for casts as these disintegrate on standing and centrifugation. Hyaline and granular casts also may be seen.

The levels of blood urea and creatinine are elevated. In patients with acute renal failure (ARF), there may be hyponatremia, hyperkalemia and acidosis. A chest X-ray film may show cardiomegaly and pulmonary congestion. Anemia and hypoalbuminemia are secondary to renal sodium and water retention with consequent hemodilution. Renal function studies show a decrease in glomerular filtration rate and renal blood flow, increased distal tubular reabsorption of sodium and water leading to hypervolemia and expansion of extracellular volume.

Evidence of preceding streptococcal infection is indicated by a rise in antistreptolysin O (ASO) titer, which is elevated in 60–80% cases. Early antibiotic treatment may attenuate this response. The rise in ASO titer begins 1–3 weeks after the streptococcal infection, reaches a peak in 3–5 weeks and then falls to insignificant levels in 6 months. It is not related to the severity or the prognosis of disease. In pyoderma, ASO titer is less commonly elevated whereas antihyaluronidase and anti-deoxyribonuclease B are more often raised. The streptozyme test detects several streptococcal antibodies but has low sensitivity and specificity and is not necessary for routine use. Streptococci can occasionally be cultured from the throat or skin infection.

Approximately 90% patients have decreased serum C3 levels that return to normal in 6–8 weeks. Serum C5 levels are mildly reduced but those of C4 are normal. Persistent hypocomplementemia is rare in PSAGN and suggests an alternative condition such as membranoproliferative glomerulonephritis, lupus nephritis or glomerulonephritis related to endocarditis or occult abscesses. The levels of ASO titer or C3 are not related to the severity of the disease.

Q. What should be the therapeutic approach?

The treatment is essentially symptomatic. Mild cases may be managed at home. Those with moderate or severe hypertension and oliguria require hospital care. Strict bed rest is not necessary. Circulatory congestion and edema can be treated with restriction of salt and water and judicious use of diuretics. Moderate-to-severe hypertension with or without encephalopathy usually responds to treatment with nifedipine or enalapril and furosemide.

If oliguria is present and the level of blood urea is elevated, dietary protein should be appropriately restricted. Penicillin may be administered for 7 days if active pyoderma or residual pharyngitis is present, but it has no influence on the course of the disease. Daily weight, urine output, and blood levels of urea and electrolytes are monitored. With careful management, the child should lose weight depending upon the degree of edema. In patients having acute pulmonary edema and shock, adequate supportive measures should

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be promptly instituted preferably in an ICU. Furosemide is given intravenously in a dose of 4–6 mg/kg. Dopamine or dobutamine should be infused and ventilatory support provided.

Q. What is the course and prognosis of the disease?

The symptoms usually begin to resolve in the first week with loss of edema and fall in the elevated blood pressure levels. Gross hematuria rapidly clears but microscopic hematuria may be detected for 6–12 months or longer. Proteinuria subsides earlier but orthostatic or intermittent proteinuria may be observed, which is of no significance. Occasionally, mild-to-moderate hypertension may persist for a few weeks. Patients, who develop ARF, especially if prolonged, may be left with residual impairment of renal function.

Q. What is crescentic glomerulonephritis (rapidly progressive glomerulonephritis)?

Crescentic glomerulonephritis refers to a particularly severe form of glomerular injury characterized by extensive crescent formation. Crescentic glomerulonephritis is most commonly associated with a severe acute nephritic syndrome with heavy proteinuria, which may progress to renal failure over a few days to weeks. However, a rapidly progressive course may occur in other forms of glomerulonephritis or interstitial nephritis, whereas crescentic glomerulonephritis may not invariably have a fulminant course.

Q. What is its etiology and classification?

In children, crescentic glomerulonephritis is usually associated with postinfectious glomerulonephritis, collagen vascular diseases and vasculitis, but in many cases, it presents as an isolated disorder. The condition is classified according to immunohistological examination of renal biopsy (Box 2).

BOX 2: Disorders associated with crescentic glomerulonephritis

- Antiglomerular basement membrane disease:
  - Goodpasture's syndrome
- Immune complex disease:
  - Postinfectious glomerulonephritis
  - Immunoglobulin A nephropathy
  - Henoch-Schönlein purpura
  - Lupus nephritis
  - Membranoproliferative glomerulonephritis
  - Idiopathic
- Pauci-immune disease:
  - Idiopathic
  - Microscopic polyarteritis
  - Wegener's granulomatosis

Q. What about the pathologic changes?

The light microscopic appearance of the affected glomeruli is very striking. The crescents are large, several cell layers thick and initially cellular, and appear to compress and distort the capillary tufts. With passage of time, the crescents become fibrous and eventually the glomerulus gets sclerosed. Renal biopsies in children with pauci-immune glomerulonephritis show features of glomerular inflammation and necrosis; varying degrees of vasculitis may also be present. A majority of patients with pauci-immune crescentic glomerulonephritis have antineutrophilic cytoplasmic antibodies (ANCA), and many have systemic features of vasculitis.

Immunofluorescence examination identifies three distinct groups. There may be linear deposition of IgG and C3 along the capillary basement membrane indicating antiglomerular basement membrane (anti-GBM) antibody mediated involvement. Such a pattern is seen in Goodpasture's syndrome, which is rare in children. More commonly discrete granular deposits of IgG and C3 are seen along the capillary walls and in the mesangium indicating immune complex-mediated glomerulonephritis, characteristically found in postinfectious glomerulonephritis (in children usually poststreptococcal). In pauci-immune crescentic GN, there is no deposition of immune reactants

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in the glomeruli, except for the nonspecific presence of fibrin in the crescents.

Q. How does it present clinically?

Crescentic glomerulonephritis usually manifests with severe acute nephritic features often associated with heavy proteinuria and moderate-to-severe edema. Renal insufficiency may be present at the onset and steadily worsen. Patients with anti-GBM antibody disease, microscopic polyarteritis, and Wegener's granulomatosis may also have pulmonary hemorrhage.

Whereas almost all patients with PSAGN with typical histological features show significant resolution within a week or two, those with crescentic glomerulonephritis do not show much improvement. In occasional cases, the onset may be insidious with features of steroid-resistant nephrotic syndrome.

A renal biopsy should be promptly carried out in children with severe acute glomerulonephritis, in whom a poststreptococcal etiology is not established or those who do not show rapid resolution. Immunofluorescence (or immunohistology) is necessary to categorize different types of crescentic glomerulonephritis. Hemogram, chest X-ray, blood cultures, serum complement, antinuclear antibody, anti-double stranded DNA antibodies and ANCA, hepatitis B and C serology, cryoglobulins, and anti-GBM antibodies are required to clarify the diagnosis.

Q. What is its treatment?

The patients are treated with aggressive immunosuppression. Early diagnosis is essential since later in the course of the disease when the crescents are mostly fibrotic with severe damage to capillary tufts, no therapy will be of any use. Three to six intravenous methylprednisolone pulses followed by long-term treatment with oral prednisolone, together with oral or intravenous cyclophosphamide for the first 3–6 months are usually employed. Plasmapheresis is used in anti-GBM antibody-induced disease and in patients having pauci-immune glomerulonephritis with pulmonary hemorrhage or those not responding to prednisolone and cyclophosphamide.

The long-term prognosis is uncertain. Even patients who seem to recover from acute renal insufficiency may gradually develop increasing renal damage. The outcome is related to the severity of the crescentic changes and the promptness of the institution of therapy.

Q. What is immunoglobulin A nephropathy?

Immunoglobulin A nephropathy is regarded to be the most frequent form of primary glomerulonephritis worldwide, particularly in Asia-Pacific countries. However, it is very uncommon in children in India. Immunoglobulin A nephropathy is diagnosed by demonstration of deposits of IgA in the mesangium on immunofluorescence examination of renal biopsy. Immunoglobulin G and C3 deposits are usually present, but IgA deposition is predominant. Similar abnormalities are seen in Henoch-Schönlein purpura nephritis.

Q. What is its clinical presentation?

Recurrent gross hematuria is the typical presentation. Flank pain may be associated. Episodes of gross hematuria often immediately follow upper respiratory infections, but microscopic hematuria may persist. In an occasional case, the disorder manifests with acute nephritic features or with heavy proteinuria and nephrotic syndrome, and more rarely with ARF and crescentic glomerulonephritis.

Q. How about its pathogenesis?

The underlying abnormality appears to be a defect in glycosylation of IgA1, which leads to formation of immune complexes. Circulating immune complexes get deposited in the mesangium. Environmental factors seem also responsible to account for the high prevalence of IgA nephropathy in South-East Asian countries. Plasma levels of IgA are increased in a variable proportion of patients, but do not correlate with the severity of renal histologic lesions.

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Q. What is its renal histology?

On light microscopic examination, focal, segmental proliferative lesions are characteristic. There is proliferation of mesangial cells with increase in mesangial matrix. In more severe cases, segmental fibrinoid necrosis, capsular adhesions, and small crescents may be seen. These proliferative lesions are observed early in the course of the disease and may gradually decrease after a few months, when focal segmental sclerosis or hyalinosis may be more prominent. Diffuse proliferative lesions are uncommon. In the rare patient presenting with rapidly progressive glomerulonephritis, extensive crescent formation is observed. Vascular lesions are usually absent. Deposits in the mesangium are predominantly composed of IgA1.

Q. What is its course and outcome?

The long-term course of the disorder is not clearly defined but the prognosis in children is much better than in adults. A small proportion of patients are reported to develop progressive renal injury and chronic kidney disease on long-term follow-up.

Factors indicating poor outcome include:

- Persistent heavy proteinuria
- Diffuse proliferation with sclerosis
- Extensive crescentic changes on renal biopsy.

Q. How to treat this condition?

Patients with microscopic hematuria with or without mild proteinuria (<1 g/1.73 m^2/day) do not require any specific medications. In those having heavy proteinuria and severe histological changes, prednisolone alone or together with azathioprine may be used. Other drugs including cyclophosphamide and mycophenolate have been employed with variable results. Patients with extensive crescentic changes have a poor outcome and need aggressive treatment. Administration of angiotensin converting enzyme inhibitors, enalapril, or lisinopril leads to reduction in proteinuria. Use of omega-3 fatty acids (fish oil, 6–12 g daily) may also retard the decline in renal function.

Q. What is acute interstitial nephritis?

Gross hematuria may occasionally be caused by exposure to drugs (such as ampicillin, ciprofloxacin, methicillin, rifampicin, phenytoin, furosemide, nonsteroidal anti-inflammatory drugs, and several others). Fever, rash, mild arthralgia, and flank pain may be associated. In about 15% cases, acute kidney injury may develop being of the nonoliguric form. Eosinophilia and eosinophiluria may be present. Acute interstitial nephritis is considered to be caused by cell-mediated immune injury. Renal histology shows acute interstitial inflammatory abnormalities with associated damage to tubular cells. Immunofluorescence examination usually does not show immune deposits.

Infiltrative changes can be diffuse but often they are patchy, mostly in the deep cortex and outer medulla. They are composed of T cells, monocytes/macrophages, plasma cells, and eosinophils. Interstitial edema is associated.

Q. How to treat it?

The offending agent should be promptly removed, which often leads to rapid recovery function. A short course of treatment with prednisolone (1–2 mg/kg/day, tapered off over 3–4 weeks) may hasten recovery. In patients with acute kidney injury, 3–5 pulses of intravenous methylprednisolone (30 mg/kg/day) may lead to rapid improvement of renal function.

Q. What is its course?

Patients, who develop renal failure, especially if prolonged, may be left with residual impairment of renal function.

Q. Are there some disorders with acute nephritic features with systemic abnormalities?

A number of conditions may present with one or more features of acute nephritis. Systemic manifestations may be mild or develop subsequently. Presence of purpuric rash or other cutaneous lesions, joint involvement, fever and

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systemic involvement, and family history of renal disease identifies the disorders that need detailed clinical and diagnostic evaluation and expert management.

CONCLUSION

Acute occurrence of gross hematuria, edema, and hypertension indicates an underlying renal disorder which is referred to as "acute nephritic syndrome". Varying degrees of oliguria and renal functional impairment are associated. The most common etiology is acute postinfectious glomerulonephritis, usually related to group A β-hemolytic streptococcal pharyngitis or pyoderma.

Renal vasculitis, IgA nephropathy, and collagen vascular disorders occasionally present with similar features are usually associated with systemic involvement (fever, arthropathy and rash). Occasionally, PSAGN manifests with atypical features such as pulmonary edema, encephalopathy and ARF. Careful evaluation and supportive management are carried out, and unnecessary tests and hazardous drugs avoided.

The outcome in PSAGN is satisfactory and almost all patients make a complete recovery. However, in unresolving cases renal biopsy is performed to detect crescentic glomerulonephritis, which requires aggressive immunosuppressive therapy and carries a guarded prognosis.

KEY LEARNING POINTS

- Clinical features of gross hematuria, edema, and hypertension constitute the acute nephritic syndrome or acute nephritis
- Varying degrees of oliguria and impairment of renal function are usually associated
- In most cases, postinfectious acute glomerulonephritis (AGN), usually poststreptococcal, is the underlying etiology
- Extensive laboratory investigations are usually not required in case of postinfectious AGN, which rapidly resolves with supportive management.

SUGGESTED READINGS

1. Bagga A, Menon S. Acute and rapidly progressive glomerulonephritis. In: Srivastava RN, Bagga A, (Eds). *Pediatric Nephrology*. New Delhi: Jaypee Brothers Medical Publishers; 2015:136-154.
2. Eison TM, Ault BH, Jones DP, Chesney RW, Wyatt RJ. Post-streptococcal acute glomerulonephritis in children: Clinical features and pathogenesis. *Pediatr Nephrol*. 2011;26:165-180.
3. KDIGO. Clinical Practice guidelines for glomerulonephritis. *Kidney Intern*. 2012;2:139-274.
4. Srivastava RN, Menon S. Acute glomerulonephritis revisited. In: Gupte S, Gupte SB, Gupte M (Eds). *Recent Advances in Pediatrics-23 Hot Topics*. New Delhi: Jaypee Brothers Medical Publishers; 2015:262-273.

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About the Author



Suraj Gupte MD FIAP FSAMS (Sweden) FRSTMH (London), an alumnus of the Postgraduate Institute of Medical Education and Research, Chandigarh, India, occupies pride of place for outstanding contribution as pediatric educationist, author, researcher, and innovator globally.

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