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# Collapsing Lesions and Focal Segmental Glomerulosclerosis in Pregnancy: A Report of 3 Cases

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The relationship between focal segmental glomerulosclerosis (FSGS) and pregnancy is complex and not completely elucidated. Pregnancy in patients with FSGS poses a high risk for complications, possibly due to hemodynamic factors, imbalance between angiogenic and antiangiogenic factors, and hormonal conditioning. Although poor clinical outcomes associated with collapsing FSGS are common outside of pregnancy, the prognosis during pregnancy is not well documented. We report 3 patients who developed collapsing FSGS during pregnancy, 2 of whom had presumed underlying FSGS. Two patients underwent biopsy during pregnancy, and 1, during the puerperium. None of the 3 patients improved spontaneously after delivery, and 1 experienced a rapid deterioration in kidney function and proteinuria after delivery. Aggressive immunosuppressive therapy led to a full response in 1 case (without chronic lesions) and to partial responses in the remaining 2 cases. These cases suggest that collapsing lesions should be considered in patients with FSGS who develop a rapid increase in serum creatinine level or proteinuria during pregnancy and that these lesions may at least partially respond to treatment.

## Introduction

The relationship between focal segmental glomerulosclerosis (FSGS) and pregnancy-associated endotheliosis, with or without preeclampsia, is complex. Together with endotheliosis, FSGS lesions are frequently reported in patients undergoing kidney biopsy during or shortly after pregnancy.[1-6] Some reports suggest that FSGS lesions represent the final evolution of endotheliosis, while in others, the 2 diseases are described as distinct.[1-10] Conversely, pregnancy in patients with already diagnosed FSGS is known to have a higher risk for complications, including preeclampsia, preterm delivery, and increases in proteinuria.[11-16]

Hyperfiltration stress, which is likely to increase in pregnancy, has been identified as a potential, albeit not confirmed, risk factor for the development of an overt clinical picture of FSGS. This can be an issue in women born with very low birth weight, a population known to have a reduced nephron number and to be particularly prone to developing FSGS.[17-21] These studies point to the importance of hemodynamic factors, namely pregnancy-related hyperfiltration, in the pathogenesis of lesions. However, in keeping with what has been observed in patients with cancer treated with antiangiogenic drugs, it is possible that the imbalance between angiogenic and anti-angiogenic factors is a contributing factor.[22] Regardless, a central role for podocyte damage as a common final pathway of these lesions is suggested; podocyte shedding increases during a pregnancy with preeclampsia.[23,24]

FSGS is a heterogeneous disease with differences in degrees of severity, evolution, and therapeutic indications. In pregnancy, the short-term prognosis in forms secondary to hyperfiltration may be favorable, but less is known about collapsing lesions, which usually have a poor prognosis outside of pregnancy.[13,15,25,26]

We report 3 patients who developed collapsing lesions in pregnancy, in 2 cases presumably superimposed on preexisting FSGS lesions (long-lasting kidney disease was suggested by ultrasonography and kidney biopsy results) and in 1 case apparently without previous pathology. These reports, observed in 2015 to 2018, suggest that the onset of collapsing lesions should be considered in patients with FSGS who have a brisk increase in serum creatinine (Scr) level or proteinuria in pregnancy, and that these lesions can respond well to treatment.

## Case Reports

### Case 1

A 23-year-old Hispanic woman was referred because of severe proteinuria in her first pregnancy to the National Institute of Perinatology. The patient and her family said they had been unaware that she was pregnant. She had congenital hypothyroidism that was never properly corrected, resulting in a mental developmental defect. Pregnancy duration was estimated using ultrasonography as being between 16 and 18 weeks. Physical examination revealed a thin woman (height, 145 cm; weight, 52 kg [usual baseline, 45 kg], body mass index, 21.4 kg/m²) with anasarca, reported as occurring and rapidly worsening in the last 2 to 3 weeks, with otherwise unremarkable physical findings. Blood pressure was 100/60 mm Hg.

Laboratory tests at referral are reported in Table 1. Thyroid-stimulating hormone level was > 300 mIU/L, and

**Table 1.** Key Laboratory Data for Patient 1

| | Baseline (other hospital; 12-14 wk gestation) | Referral (16-18 wk gestation) | Miscarriage (19-20 wk gestation) | Biopsy (3 wk after miscarriage) | Follow-up (2 y after biopsy) |
|---|---|---|---|---|---|
| Scr, mg/dL | 0.36 | 0.87 | 0.75 | 0.55 | 0.44 |
| eGFR, mL/min/1.73 m² | 152 | 93.9 | 112 | 132 | 142 |
| Serum albumin, g/dL | 3.0 | 3.0 | 2.9 | 2.5 | 4 |
| Proteinuria, g/24 h | NA | 5.1 | 7.6 | 15.8 | 0.10 |
| Viral markers | Hepatitis B & C, HIV: negative | | | | |
| Immunologic markers | ANA and ANCA negative, C3 and C4 normal | | | | |

*Note:* Free thyroxine, 0.50 ng/dL; reference values, 0.58 to 1.64 ng/dL.
Abbreviations and definitions: ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibody; eGFR, estimated glomerular filtration rate (calculated with the Chronic Kidney Disease Epidemiology Collaboration [CKD-EPI] equation); HIV, human immunodeficiency virus; Scr, serum creatinine.

serum albumin level was 3.0 g/dL, in the presence of massive proteinuria (protein excretion >10 g/L).

Kidney ultrasonography findings were normal. The patient was hospitalized and started on thyroid hormone replacement therapy. Due to risks associated with kidney biopsy in pregnancy, glucocorticoid treatment was initiated (3 boluses of 1 g of methylprednisolone followed by 40 mg of oral prednisone). Spontaneous fetal loss occurred 2 weeks later (male fetus weighing 269 g).

Given persistent proteinuria after 5 weeks of steroids, a kidney biopsy was performed 3 weeks after delivery, which demonstrated FSGS with collapsing lesions in 6 of 40 glomeruli without signs of chronic kidney involvement. Immunofluorescence was negative or nonspecific (Fig 1). One infusion of 375 mg/m² of rituximab was administered, attaining full remission within 1 month. At last follow-up, the patient was in good clinical condition, with a normal Scr level and protein excretion < 0.10 g/d.

## Case 2

A 27-year-old Hispanic woman in her first pregnancy was referred to the National Institute of Perinatology at 14 weeks' gestation because of hypertension and proteinuria
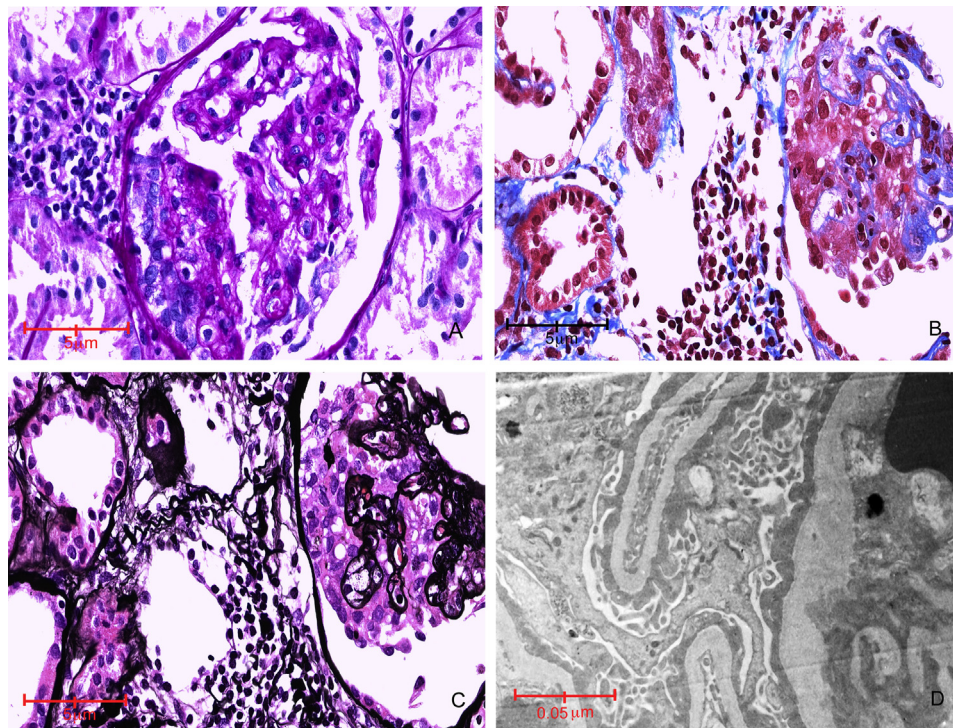


**Figure 1.** Case 1. Light microscopy: hypertrophic podocytes with crown disposition around collapsed segments (A: periodic acid–Schiff stain, B: Masson stain, C: Jones methenamine stain; A-C: original magnification, ×40). (C) Endothelial edema and endothelial cells with foamy morphology. Glomeruli are of normal size, and tubule interstitial structures are overall preserved. (D) Ultrastructural analysis shows diffuse podocyte effacement (original magnification, ×5,000). Immunofluorescence staining was negative or nonspecific (Fig S1; Table S1).

(protein excretion, 2.3 g/d). She had a history of hypertension, discovered 18 months previously. Kidney function at that time was reported to be decreased with proteinuria present, but details were unavailable. Physical examination revealed mild peripheral edema and findings were otherwise unremarkable. Blood pressure was controlled with α-methyldopa (500 mg 3 times daily).

Laboratory tests at referral and during follow-up are reported in Table 2. Ultrasonography revealed kidneys of borderline-small size (right, 9.3 × 4.5 cm; left, 9.9 × 5.2 cm) with slightly irregular margins and loss of corticomedullary differentiation. The patient's clinical condition and proteinuria remained stable until gestational week 25, when proteinuria suddenly increased to protein excretion > 20 g/d, with stable Scr level. Because of the increase in proteinuria, kidney biopsy was performed at 26 gestational weeks, which revealed FSGS with collapsing lesions, tubular atrophy and regeneration, interstitial fibrosis, and occasional inflammatory infiltrate. Mild mesangial hypercellularity was observed, but immunofluorescence was negative for immunoglobulin A (Fig 2).

The patient was treated with 3 boluses of 1 g of methylprednisolone, followed by 30 mg/d of prednisone without reduction in proteinuria. Due to worsening hypertension, nifedipine and hydralazine were added at 30 and 32 weeks of gestation, respectively. The ratio between angiogenic and antiangiogenic factors was altered, suggesting superimposed preeclampsia (placenta growth factor, 8,575 pg/mL; soluble fms-like tyrosine kinase 1, 761.1 pg/mL; ratio, 88.76; reference ratio, >38 for a diagnosis of preeclampsia[27]). Fetal growth was normal and uterine and umbilical ultrasonography findings were normal for gestational age.

When hypertension control became difficult despite a 3-drug therapy (nifedipine, 120 mg/d; hydralazine, 150 mg/d; and α-methyldopa, 1,500 mg/d), cesarean delivery was performed at gestational week 33, delivering a healthy male baby weighing 2.1 kg (63rd centile for gestational age). Due to the lack of clinical response in the first week after delivery (after 8 weeks of steroid treatment), the patient was administered 375 mg/m$^2$ of rituximab and subsequently treated with tacrolimus, with partial reduction in proteinuria. At last follow-up, 11

months after delivery, she is in partial remission, with normal kidney function, on treatment with tacrolimus (3 mg/d) and angiotensin-converting enzyme inhibition (Table 2).

## Case 3

During her third pregnancy, a 27-year-old Hispanic woman was referred to the National Institute of Perinatology for hypertension, diagnosed at gestational week 35, during a routine check-up. Her medical history included 1 term pregnancy with cesarean delivery, which had otherwise been uneventful, and a spontaneous miscarriage at 10 gestational weeks 1 year earlier. Hypertension developed during this pregnancy; Scr level was 1.0 mg/dL and proteinuria was detected. However, she was not advised to undergo further analysis of kidney function. Physical examination revealed a thin woman (height, 158 cm; weight, 51.8 kg) with peripheral edema and blood pressure of 140/85 mm Hg. Examination findings were otherwise unremarkable.

Laboratory tests at referral and during follow-up are reported in Table 3. Ultrasonography revealed small kidneys with poor corticomedullary differentiation. The patient was hospitalized at 36.2 gestational weeks for evaluation of her renal condition. The growth of the child was normal, Scr level had increased to 1.2 mg/dL, urinary sediment analysis revealed dysmorphic erythrocytes and granular casts, proteinuria was protein excretion of 11 g/d, and serum albumin level was 2.9 g/dL (Table 3). Fetal growth was normal, as were uterine and umbilical ultrasonographic examination findings. A cesarean delivery was performed at 36.4 weeks of gestation, delivering a healthy female baby weighing 2,590 g (41st centile for gestational age).

After delivery, Scr level rapidly increased to 1.8 mg/dL (day 1) and 2.1 mg/dL (day 2), and the patient was referred for kidney biopsy. On day 3 after delivery, Scr level further worsened to 5.6 mg/dL, with oliguria, and dialysis was initiated to treat hypervolemia. A rapidly progressive glomerulonephritis was suspected and while waiting for results of the kidney biopsy, the patient was treated with 3 boluses of 500 mg of methylprednisolone, plasmapheresis (3 sessions), and cyclophosphamide (1 g intravenously). The kidney biopsy showed collapsing lesions in 1 of 9

**Table 2.** Key Laboratory Data for Patient 2

| | Referral (14 wk gestation) | Biopsy (26 wk gestation) | Delivery (33 wk gestation) | Follow-up (9 mo after delivery) |
|---|---|---|---|---|
| Scr, mg/dL | 0.9 | 1.04 | 1.6 | 1.3 |
| eGFR, mL/min/1.73 m$^2$ | 88 | 73 | 43 | 58 |
| Serum albumin, g/dL | 3.8 | 2.13 | 1.6 | 3.6 |
| Proteinuria, g/24 h | 2.3 | 20 | 11 | 5.3 |
| Viral markers | Hepatitis B & C, HIV, parvovirus B-19, CMV: negative | | | |
| Immunologic markers | ANA, ANCA, lupus-like anticoagulant, antibodies anticardiolipin IgG/IgM, rheumatoid factor: negative; C3 C4: normal | | | |

Abbreviations and definitions: ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibody; CMV, cytomegalovirus; eGFR, estimated glomerular filtration rate (calculated with the Chronic Kidney Disease Epidemiology Collaboration [CKD-EPI] equation); HIV, human immunodeficiency virus; IgG, immunoglobulin G; Scr, serum creatinine.
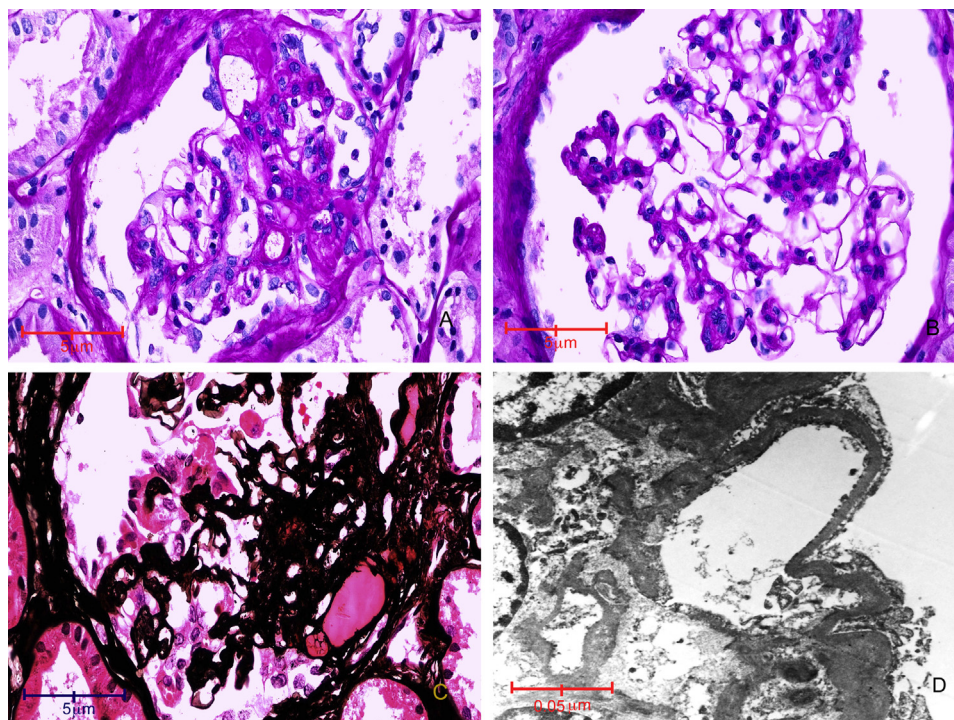
**Figure 2.** Case 2. (A-C) Light microscopy. (A) Segmental sclerosis with capsular synechia and areas of hyalinosis; (B, C) in some fields, segmental collapse; and (C) podocyte hypertrophy is observed (A, B: periodic acid–Schiff stain, C: Masson trichrome stain; A-C: original magnification, ×40). (D) At electron microscopy, diffuse effacement of foot processes is observed (original magnification, ×2,500). Immunofluorescence staining was negative or nonspecific (Figs S2-S6; Table S1).

glomeruli, with the remaining nephrons showing glomerulomegaly, signs of focal segmental sclerosis, interstitial infiltrate, and tubular regeneration, without immune deposits (Fig 3). When biopsy results became available, she was switched to oral prednisone and tacrolimus treatment. Kidney function partially improved and at the last follow-up, 4 months after delivery, Scr level was 2.29 mg/dL and proteinuria was protein excretion of 1.55 g/d (Table 3).

## Discussion

Previously unsuspected kidney disease may be discovered during pregnancy.

The incidence of collapsing FSGS in pregnancy is not known; we were able to find only 2 such reports, with 2 other reports (4 cases) in which collapsing lesions were superimposed on thrombotic microangiopathy (TMA).[28-31] The first 2 cases were reported in the first trimester of pregnancy (6 and 8 weeks' gestation). In the first case, systemic lupus erythematosus was present, and in the second, a viral infection and genetic predisposition coexisted.[28,29] In the 4 additional cases, collapsing glomerulosclerosis was associated with TMA; these patients developed acute kidney injury in the postpartum period.[30,31] The association with TMA[31-33] is intriguing. TMA is frequently found in preeclampsia, and some experts consider glomerular endotheliosis a specific variant of TMA.[4] However, in none of our cases were lesions typical of endotheliosis found, and all responded partially or completely to immunosuppressive treatment.

**Table 3.** Key Laboratory Data for Patient 3

|  | Referral (35 wk gestation)[a] | Delivery (36.4 wk gestation) | Biopsy (3 d after delivery) | Follow-up (4 mo after biopsy) |
|---|---|---|---|---|
| Scr, mg/dL | 1.0 | 1.2 | 5.6 | 2.2 |
| eGFR, mL/min/1.73 m² | 77.1 | 31.5 | 9 | 29.7 |
| Serum albumin, g/dL | 3.2 | 2.9 | 2.9 | 4.9 |
| Viral markers | Hepatitis B & C, HIV: negative | | | |
| Immunologic markers | ANA, ANCA negative; C3 and C4 normal | | | |

Abbreviations and definitions: ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibody; eGFR, estimated glomerular filtration rate (calculated with the Chronic Kidney Disease Epidemiology Collaboration [CKD-EPI] equation); HIV, human immunodeficiency virus; Scr, serum creatinine.
[a]Scr level was reported to be 1.0 mg/dL 1 year previously; the presence of proteinuria was reported, but no quantitative data were available.
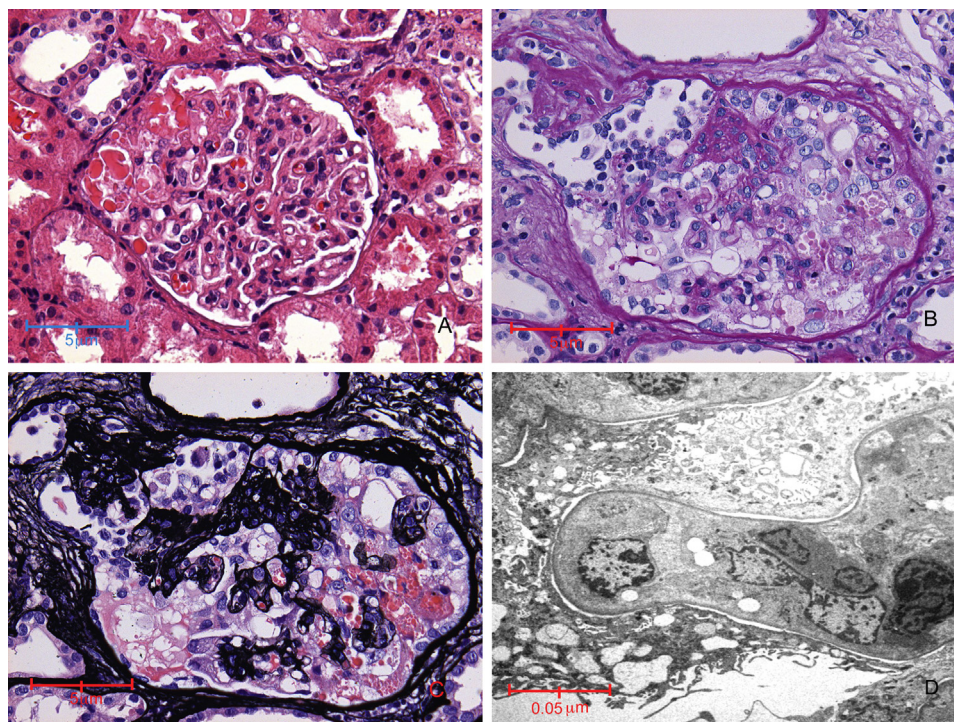
**Figure 3.** Case 3. (A-C) Light microscopy. (A) Glomerular hypertrophy; hypertrophic podocytes show vacuolar degeneration, segmental sclerosis (hematoxylin and eosin); (B, C) global collapse (B: periodic acid–Schiff stain; C: Masson trichrome stain; A-C: original magnification, ×40). (D) At electron microscopy, diffuse effacement of foot processes is observed (original magnification, ×2,500). Immunofluorescence staining was negative or nonspecific (Figs S7 and S8; Table S1).

Our cases were not associated with known viral infections or other autoimmune diseases, although there was incomplete availability of appropriate testing. Genetic analysis was likewise not available, a situation common to limited-resource settings. In particular, APOL1 variants have been associated with increased risk for progression of different kidney diseases, as well as de novo collapsing glomerulopathy after transplantation, and their frequency is probably higher in Hispanic individuals.[34-36]

Collapsing lesions in pregnancy have been infrequently reported, possibly because of the rarity of kidney biopsy in this setting. A common element in our 3 cases was good response to therapy, unusual in collapsing FSGS. It is difficult to interpret this finding; the presence of at least 1 component of superimposed preeclampsia was suspected in case 2. The effect of rituximab may have been enhanced by previous glucocorticoid use in case 1. Pregnancy may have served as a trigger on a particular morphologic or clinical background, which may explain the treatment response observed after delivery.

In summary, we report 3 cases of collapsing lesions in pregnancy, 2 of which were complications of FSGS. We observed a favorable, albeit in 2 cases incomplete, response to treatment. This underlines the importance of considering this diagnosis in patients with FSGS who develop a brisk increase in Scr level or proteinuria during

pregnancy because these lesions may at least partially respond to treatment.

## Supplementary Material

### Supplementary File (PDF)

**Figure S1:** Direct immunofluorescence microscopy of kidney biopsy specimen from case 1 shows nonspecific IgG trapping.

**Figure S2:** Direct immunofluorescence microscopy of kidney biopsy specimen from case 2 shows strong IgG positivity.

**Figure S3:** Direct immunofluorescence microscopy of kidney biopsy specimen from case 2 shows negative IgA staining.

**Figure S4:** Direct immunofluorescence microscopy of kidney biopsy specimen from case 2 shows IgM deposits.

**Figure S5:** Direct immunofluorescence microscopy of kidney biopsy specimen from case 2 shows κ light chain positivity.

**Figure S6:** Direct immunofluorescence microscopy of kidney biopsy specimen from case 2 shows λ light chain positivity.

**Figure S7:** Direct immunofluorescence microscopy of kidney biopsy specimen from case 3 shows weak IgG staining.

**Figure S8:** Direct immunofluorescence microscopy of kidney biopsy specimen from case 3 shows C3c dots.

**Table S1:** Details of the kidney biopsies.

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