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# Stepwise Interpretation of Albumin Fraction Abnormalities on Serum Protein Capillary Electrophoresis in Nephrotic Syndrome: A Case Series

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Serum protein electrophoresis (SPEP) is commonly used in the diagnostic evaluation of nephrotic syndrome (NS) to rule out monoclonal gammopathies. However, NS itself and various other factors may cause transient bisalbuminemia, resulting in albumin fraction irregularities in capillary SPEP and complicating interpretation. This study proposes a stepwise approach for interpreting albumin fraction irregularities, illustrated through three representative cases of NS.

Case 1: a 44-year-old woman with isolated proteinuria, Case 2: a 36-year-old woman with diabetic foot infection, and Case 3: a 58-year-old man with ankylosing spondylitis, all presenting with nephrotic-range proteinuria. On initial SPEP case 1 exhibited a distinct sharp peak, while cases 2 and 3 showed irregular bands between the albumin and α1 regions. A stepwise interpretative approach was applied: First, technical errors were excluded by repeat analysis using alternative capillaries on the same analyzer. Second, potential biochemical interferences were evaluated. Third, SPEP was repeated using freshly collected samples. Fourth, persistent albumin fraction irregularities prompted consideration of biochemical albumin modifications, such as glycosylated or carbamylated forms. Fifth, other causes including drugs, heavy metal toxicity, liver disease, and systemic inflammation were excluded. Finally, additional tests such as immunofixation electrophoresis were performed to rule out monoclonal proteins. In the absence of monoclonal proteins, the condition was interpreted as bisalbuminemia related to NS. Using this approach, transient bisalbuminemia was considered the most likely cause in Cases 1 and 2, whereas the disappearance of the irregular band in Case 3 suggested a preanalytical artifact. Systematic interpretation of albumin fraction irregularities can help distinguish true pathology from benign or artifactual patterns in patients with NS.

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Case Report

## INTRODUCTION

Capillary serum protein electrophoresis (SPEP) is widely used in the diagnostic workup of nephrotic syndrome (NS), particularly to rule out monoclonal gammopathies. Improved resolution of albumin variants with modern capillary electrophoresis systems has been highlighted in recent analytical reviews.[1] However, albumin fraction irregularities (AFI) —such as split peaks or shoulders—can complicate interpretation, especially when overlapping with the clinical findings of NS.[2]

One such irregularity is bisalbuminemia,

characterized by the presence of two electrophoretically distinct albumin bands. This phenomenon may result from structural modifications of albumin and can be either inherited (familial) or acquired (transient).[3] Transient bisalbuminemia has been associated with various conditions, including diabetes mellitus (DM),[4] NS,[5] liver disease,[6] infections, and exposure to specific drugs.[7,8] In some cases, changes in the AFI may reflect disease activity in NS.[9] Modified albumin forms, such as glycated or carbamylated variants, alter the albumin's charge, affecting its electrophoretic mobility. Glycated albumin, modified by glucose, serves as a marker of short-term glycemic control. In contrast, carbamylated albumin results from modification by urea-derived compounds and is associated with kidney dysfunction and protein damage. Specifically, carbamylation occurs predominantly at lysine-549, causing a detectable shift in electrophoretic mobility and resulting in asymmetry.[10]

In addition to bisalbuminemia, several biochemical or analytical factors may also affect the albumin region in SPEP. Severe hyperlipidemia often causes cathodal distortion, while hyperbilirubinemia can impact the anodal region.[7,11] Aged or deteriorated capillaries are also known to produce artifactual changes that resemble abnormal albumin variants.[7]

These irregularities, though typically benign, may mimic pathological globulin peaks, leading to unnecessary testing—particularly in patients undergoing evaluation for monoclonal gammopathies.[12]

In this report, we present three cases of NS in which AFI were observed on capillary SPEP. Through these cases, we propose a structured, stepwise diagnostic approach to assist clinicians and laboratory specialists in accurately interpreting such findings and avoiding diagnostic pitfalls.

## CASE REPORT

### Case 1

A 44-year-old woman was referred for evaluation

**Table 1.** Patient's laboratory test results

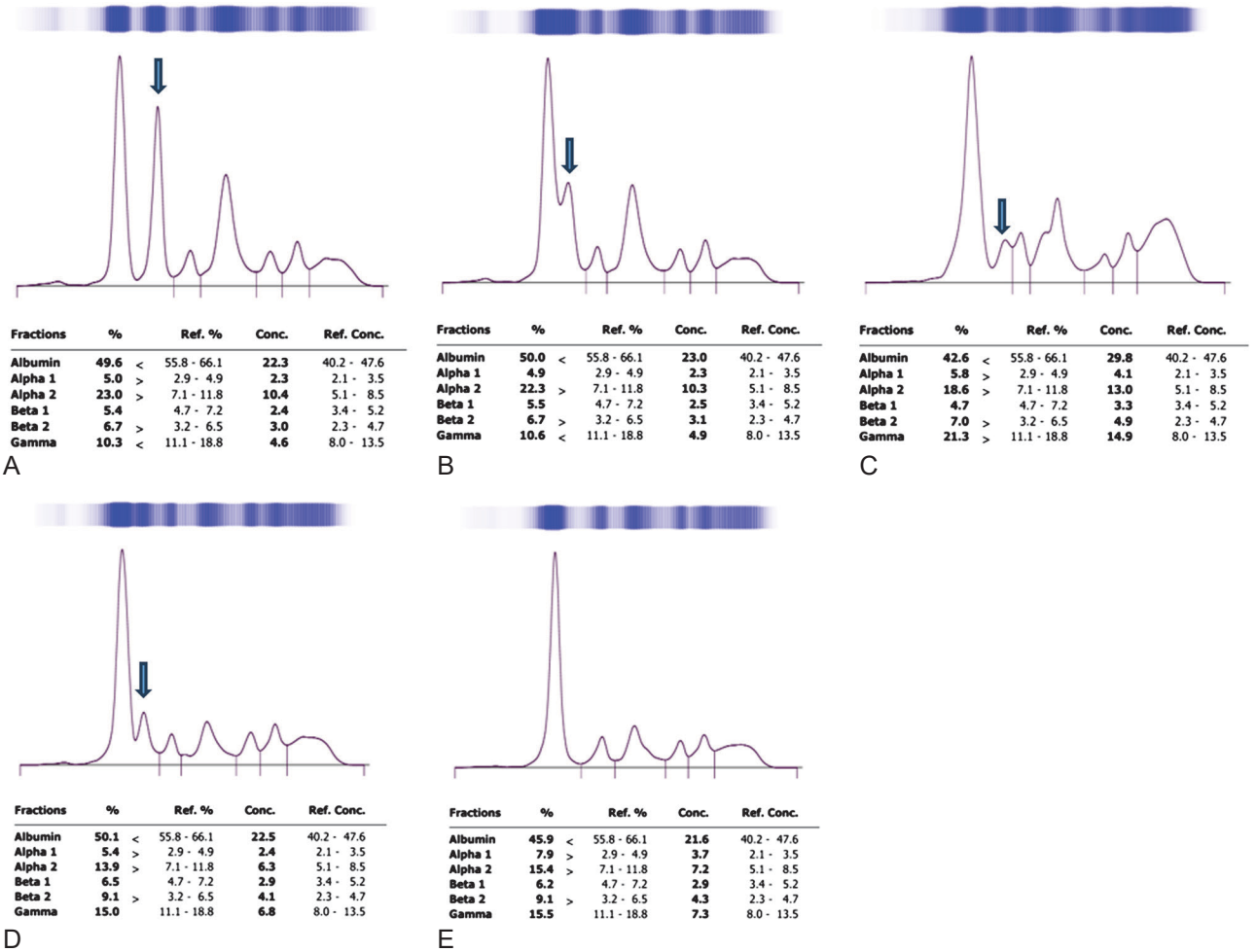
| Test | Result | | Reference İnterval | Result | Reference İnterval |
|---|---|---|---|---|---|
| **SERUM** | **Case 1** | **Case 2** | | **Case 3** | |
| Total protein (g/L) | 45 | 74 | 66-83 | 49 | 66-83 |
| Albumin (g/L) | 22 | 32 | 35-48 | 23 | 35-48 |
| Urea (mg/dL) | 17 | 42 | 15-40 | 69 | 15-40 |
| Creatinine (mg/dL) | 0.8 | 1.43 | < 0.9 | 1.34 | < 0.9 |
| eGFR (mL/min/1.73m$^2$)[a] | 89.3 | 46.8 | > 90 | 57.6 | > 90 |
| Total cholesterol (mg/dL) | 647 | 276 | 110-200 | 346 | 110-200 |
| HDL-cholesterol (mg/dL) | 50 | 19 | 40-85 | 69 | 40-85 |
| LDL-cholesterol (mg/dL) | 538 | 151 | 60-130 | 227 | 60-130 |
| Triglycerides (mg/dL) | 207 | 365 | 50-200 | 154 | 50-200 |
| Fasting blood glucose (mg/dL) | 91 | 167 | 70-100 | 92 | 70-100 |
| Total bilirubin (mg/dL) | 0.16 | 0.45 | < 1.2 | 0.25 | < 1.2 |
| Direct bilirubin (mg/dL) | 0.07 | 0.25 | 0-0.3 | 0.12 | 0-0.3 |
| CRP (mg/L) | - | 22 | < 5 | 1.7 | < 5 |
| Ig A (g/L) | 1.79 | 3.58 | 0.7-4 | 2.82 | 0.7-4 |
| Ig G (g/L) | 4.76 | 17.39 | 7-16 | 8.14 | 7-16 |
| Ig M (g/L) | 1.83 | 2.36 | 0.4-2.3 | 0.66 | 0.4-2.3 |
| Kappa free light chain (mg/L) | 42.1 | 80 | 6.7-22.4 | - | 6.7-22.4 |
| Lambda free light chain (mg/L) | 34.6 | 93.1 | 8.3-27 | - | 8.3-27 |
| Kappa / Lambda ratio | 1.21 | 0.859 | 0.31-1.56 | - | 0.31-1.56 |
| HbA1C (%) | - | 8 | 3.5-5,6 | - | 3,5-5,6 |
| URINE | | | | | |
| Protein (dipstick urinalysis) | 4+ | 4+ | Negatif | 4+ | Negatif |
| Protein/Creatinine (spot urine, mg/g) | 5995 | - | 0-150 | - | 0-150 |
| Albumin/Creatinine (spot urine, mg/g) | - | - | 0-30 | 7125 | 0-30 |

[a]eGFR was calculated according to the formula CKD-EPI.[14] CRP, C-reactive protein (mg/L); eGFR, Estimated glomerular filtration rate; HbA1c, Glycated hemoglobin A1c; HDL, High-density lipoprotein cholesterol; Ig, Immunoglobulin; LDL, Low-density lipoprotein cholesterol.

of isolated proteinuria detected during a routine check-up. A urine dipstick test revealed 4+ proteinuria. Laboratory results showed marked hypoalbuminemia and severe hyperlipidemia which were consistent with NS (Table 1). Albumin-to-creatinine ratio in spot urine analysis confirmed nephrotic-range proteinuria. These findings warranted further investigation to rule out multiple myeloma. SPEP demonstrated a distinct, sharp cathodal peak between the albumin and α1 bands (Figure 1A).

## Case 2

A 36-year-old woman with type 1 DM was admitted for the treatment of a diabetic foot infection. She had recently been prescribed amoxicillin/clavulanic acid and ciprofloxacin but discontinued both due to nausea. Poor glycemic control was confirmed by elevated fasting blood glucose and HbA1c levels (Table 1). On admission, dipstick urinalysis revealed 4+ proteinuria, indicating nephrotic-range proteinuria. Laboratory results showing marked hypoalbuminemia and significant dyslipidemia were consistent with NS (Table 1). These findings warranted further investigation to rule out multiple myeloma. SPEP revealed an irregular band between the albumin and α1 regions, accompanied by an elevated α2-globulin band, which are typical findings in NS (Figure 1C).



| Fractions | % | | Ref. % | Conc. | Ref. Conc. |
|---|---|---|---|---|---|
| Albumin | 49.6 | < | 55.8 - 66.1 | 22.3 | 40.2 - 47.6 |
| Alpha 1 | 5.0 | > | 2.9 - 4.9 | 2.3 | 2.1 - 3.5 |
| Alpha 2 | 23.0 | > | 7.1 - 11.8 | 10.4 | 5.1 - 8.5 |
| Beta 1 | 5.4 | | 4.7 - 7.2 | 2.4 | 3.4 - 5.2 |
| Beta 2 | 6.7 | > | 3.2 - 6.5 | 3.0 | 2.3 - 4.7 |
| Gamma | 10.3 | < | 11.1 - 18.8 | 4.6 | 8.0 - 13.5 |

A

| Fractions | % | | Ref. % | Conc. | Ref. Conc. |
|---|---|---|---|---|---|
| Albumin | 50.0 | < | 55.8 - 66.1 | 23.0 | 40.2 - 47.6 |
| Alpha 1 | 4.9 | | 2.9 - 4.9 | 2.3 | 2.1 - 3.5 |
| Alpha 2 | 22.3 | > | 7.1 - 11.8 | 10.3 | 5.1 - 8.5 |
| Beta 1 | 5.5 | | 4.7 - 7.2 | 2.5 | 3.4 - 5.2 |
| Beta 2 | 6.7 | > | 3.2 - 6.5 | 3.1 | 2.3 - 4.7 |
| Gamma | 10.6 | < | 11.1 - 18.8 | 4.9 | 8.0 - 13.5 |

B

| Fractions | % | | Ref. % | Conc. | Ref. Conc. |
|---|---|---|---|---|---|
| Albumin | 42.6 | < | 55.8 - 66.1 | 29.8 | 40.2 - 47.6 |
| Alpha 1 | 5.8 | > | 2.9 - 4.9 | 4.1 | 2.1 - 3.5 |
| Alpha 2 | 18.6 | > | 7.1 - 11.8 | 13.0 | 5.1 - 8.5 |
| Beta 1 | 4.7 | | 4.7 - 7.2 | 3.3 | 3.4 - 5.2 |
| Beta 2 | 7.0 | > | 3.2 - 6.5 | 4.9 | 2.3 - 4.7 |
| Gamma | 21.3 | > | 11.1 - 18.8 | 14.9 | 8.0 - 13.5 |

C

| Fractions | % | | Ref. % | Conc. | Ref. Conc. |
|---|---|---|---|---|---|
| Albumin | 50.1 | < | 55.8 - 66.1 | 22.5 | 40.2 - 47.6 |
| Alpha 1 | 5.4 | > | 2.9 - 4.9 | 2.4 | 2.1 - 3.5 |
| Alpha 2 | 13.9 | > | 7.1 - 11.8 | 6.3 | 5.1 - 8.5 |
| Beta 1 | 6.5 | | 4.7 - 7.2 | 2.9 | 3.4 - 5.2 |
| Beta 2 | 9.1 | > | 3.2 - 6.5 | 4.1 | 2.3 - 4.7 |
| Gamma | 15.0 | | 11.1 - 18.8 | 6.8 | 8.0 - 13.5 |

D

| Fractions | % | | Ref. % | Conc. | Ref. Conc. |
|---|---|---|---|---|---|
| Albumin | 45.9 | < | 55.8 - 66.1 | 21.6 | 40.2 - 47.6 |
| Alpha 1 | 7.9 | > | 2.9 - 4.9 | 3.7 | 2.1 - 3.5 |
| Alpha 2 | 15.4 | > | 7.1 - 11.8 | 7.2 | 5.1 - 8.5 |
| Beta 1 | 6.2 | | 4.7 - 7.2 | 2.9 | 3.4 - 5.2 |
| Beta 2 | 9.1 | > | 3.2 - 6.5 | 4.3 | 2.3 - 4.7 |
| Gamma | 15.5 | | 11.1 - 18.8 | 7.3 | 8.0 - 13.5 |

E

**Figure 1.** Capillary serum protein electrophoresis (SPEP) patterns demonstrating albumin fraction irregularities (AFI) observed in three nephrotic syndrome cases. (A) Initial sample from Case 1 showing a sharp cathodal split peak; (B) Follow-up sample from Case 1 demonstrating persistence of the irregularity, supporting transient bisalbuminemia; (C) Case 2 showing an irregular band between the albumin and α1 regions, likely related to glycated albumin in the setting of poor glycemic control; (D) Initial sample from Case 3 showing a cathodal shoulder suggestive of a possible artifact; (E) Follow-up sample from Case 3 in which the irregular band disappears, indicating a preanalytical cause. Blue arrows indicate the abnormal bands in the albumin region. SPEP performed on the Sebia Capillary 2 Tera system (Sebia, France).

**Case 3**

A 58-year-old man, a known case of ankylosing spondylitis, presented with a four-month history of lower limbs edema. Dipstick urinalysis showed 4+ proteinuria, and a kidney biopsy was planned to investigate possible amyloidosis. Laboratory tests revealed significant hypoalbuminemia and dyslipidemia, consistent with NS (Table 1). Albumin-to-creatinine ratio in spot urine analysis confirmed nephrotic-range proteinuria. These findings warranted further investigation to rule out multiple myeloma. SPEP revealed a cathodal shoulder between the albumin and α1 bands, accompanied by an elevated α2-globulin band, which are typical findings in NS (Figure 1D).

All capillary SPEP analyses were performed using the Sebia Capillary 2 Tera system (Sebia, France). Serum and urine immunofixation electrophoresis was conducted with the Sebia Hydragel 4 IF system. Biochemical analyses were carried out on the Roche Cobas 8000 analyzer (Roche Diagnostics GmbH, Germany). Detailed laboratory findings, including reference intervals, are provided in Table 1.

Evaluation of the cases using a stepwise approach

In all three NS cases, AFI such as cathodal shoulders and split peaks were observed, prompting a systematic diagnostic approach shown in Figure 2. The stepwise strategy applied was as follows (Figure 2):

First, technical artifacts such as aged capillaries were excluded by reanalyzing all samples using different capillaries on the same analyzer, with consistent results. In addition, no AFI were observed in the SPEP results of other patients analyzed in the same run.

Second, biochemical interferences were evaluated. All patients exhibited severe hyperlipidemia, which is known to cause cathodal distortion in some systems.[7] However, a study by Seneş *et al.*, which investigated the effect of lipemia on capillary SPEP using the same analyzer (Sebia Capillary 2 Tera system, France), reported no irregularity in the albumin band.[13] Thus, interference due to hyperlipidemia was excluded. All patients had normal bilirubin levels. Hyperbilirubinemia, hemolysis, clotting, and improper storage were also ruled out.

Third, repeat testing was performed on newly collected samples in Cases 1 and 3. In Case 1, the sharp cathodal peak persisted, albeit less prominently (Figure 1B), suggesting transient bisalbuminemia. In contrast, Case 3 showed resolution of the cathodal shoulder in the follow-up sample (Figure 1E). Given the short interval between tests (five days), the absence of treatment, and persistent proteinuria, a preanalytical error was considered the most likely explanation.

Fourth, potential biochemical modifications of albumin such as glycated or carbamylated forms were considered. In Case 1, normal fasting glucose levels and the absence of diabetes mellitus (Table 1) suggested that glycated albumin was not involved. Normal urea levels and the absence of the asymmetry described by Favresse *et al.*[9] supported the conclusion that carbamylation was also not involved. In Case 2, poor glycemic control was confirmed by elevated fasting glucose and HbA1c (Table 1). The AFI was most likely attributable to glycated albumin. Although serum urea was mildly elevated, the absence of asymmetry again ruled out carbamylation. In Case 3, normal glucose levels and no history of diabetes mellitus excluded glycated albumin as a factor. Although the urea level was slightly elevated, the absence of asymmetry and the disappearance of the irregular band on repeat SPEP supported a non-biochemical, preanalytical cause.

Fifth, other known causes of transient bisalbuminemia such as exposure to high-dose β-lactam antibiotics, sulfonamides, ceftriaxone, salicylates, liver disease, infections, or heavy metal toxicity were not identified in any of the patients. Genetic bisalbuminemia was considered unlikely in all cases, given the absence of a family history. In addition, in Case 1, variability between the initial and follow-up SPEP results (Figure 1A–1B) further supported a non-genetic cause.

Finally, monoclonal gammopathy was excluded through negative serum and urine immunofixation electrophoresis in all cases. Although a monoclonal globulin peak in the albumin fraction is very rare, free monoclonal light chains can occasionally produce an extra gradient or atypical band between the albumin and α1 regions.[14] Since these patients were initially screened for monoclonal gammopathy, it was critical to avoid misinterpreting albumin abnormalities as pathological monoclonal peaks.

These findings supported a diagnosis of transient bisalbuminemia in Case 1 and 2. NS itself, an

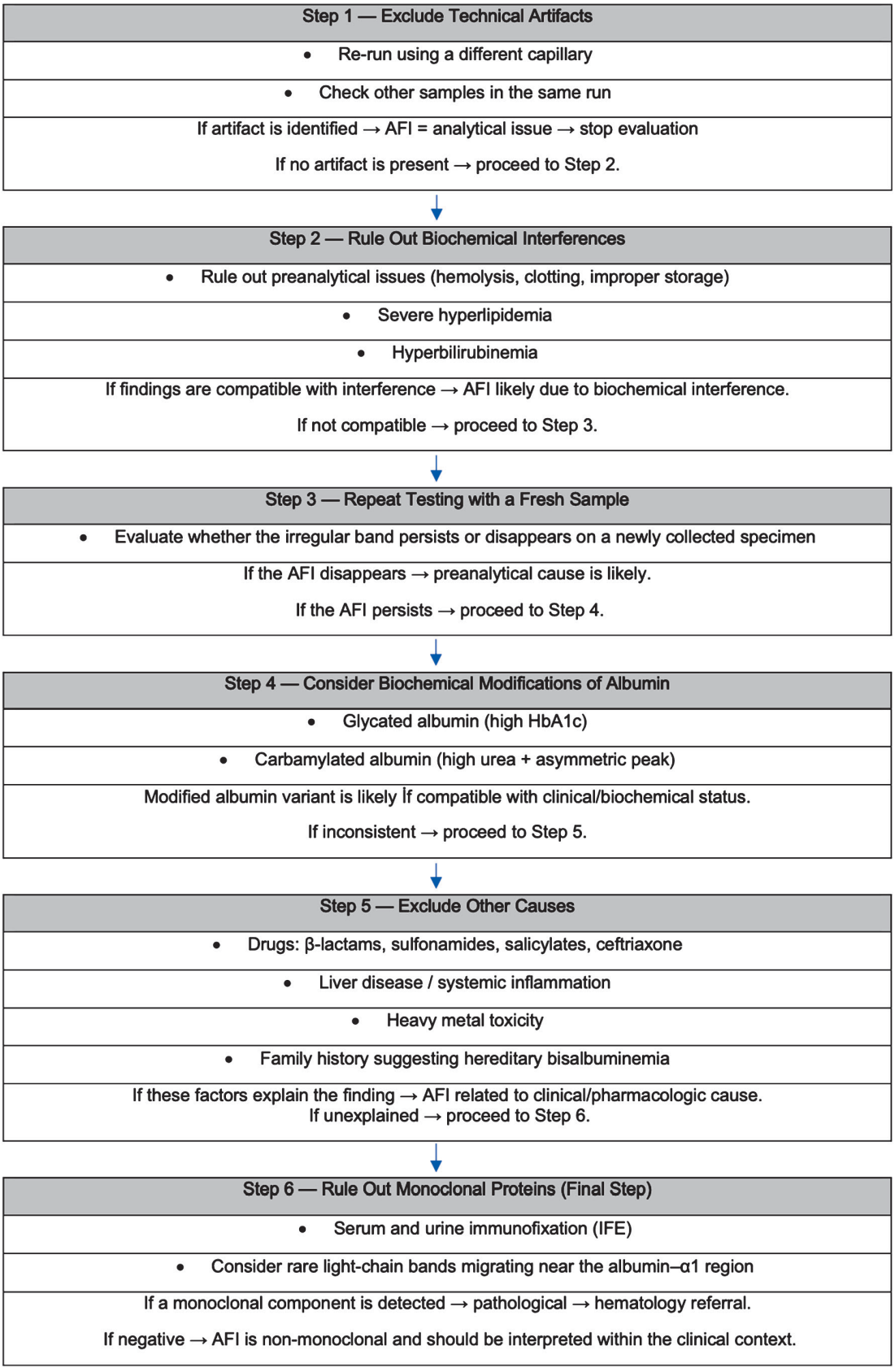



**Figure 2.** Stepwise diagnostic algorithm for interpreting albumin fraction irregularities (AFI) in capillary serum protein electrophoresis.

established trigger, was considered the most likely cause in Case 1. Although the underlying mechanism remains unclear, hypotheses include altered albumin binding due to oxidative stress or protein modification by nephrotic-range proteins.[6,10] Similarly, transient bisalbuminemia due to glycated albumin was the most plausible explanation for the irregular band observed in Case 2, although the lack of follow-up data precluded a definitive conclusion. As noted above, the irregular band observed in Case 3 was most consistent with a sample-related preanalytical error.

## DISCUSSION

In this case series, we presented three patients with NS, each exhibiting distinct AFI on capillary SPEP. Case 1 had a sharp, persistent cathodal albumin split consistent with transient bisalbuminemia. Case 2 displayed an irregular albumin band likely reflecting poor glycemic control (glycated albumin). Case 3 showed a transient cathodal shoulder that resolved on repeat testing, suggesting a preanalytical artifact. In all cases, monoclonal gammopathy was excluded by immunofixation testing, and common interferences (e.g., lipemia, hyperbilirubinemia) were ruled out. Together, these observations demonstrate the diverse potential causes (biochemical, structural, or artifactual) underlying AFI in NS and underscore the importance of a structured diagnostic approach to avoid misinterpretation.

Reports of AFI in NS are scarce, but the phenomenon has been noted both as albumin variants and as transient acquired patterns in certain diseases.[1] True bisalbuminemia (also termed alloalbuminemia) is characterized by a bifid or broadened albumin band and can be either hereditary or acquired. For example, a report documented a patient with congenital bisalbuminemia and NS who exhibited "bisalbuminuria," confirming that the variant albumin appeared in the urine.[5]

Ogawa *et al.* reported a 51-year-old patient with NS, FSGS, and Waldenström macroglobulinemia who developed a double albumin peak on capillary electrophoresis; notably, the split band disappeared after five days of corticosteroid therapy despite ongoing heavy proteinuria.[9] Similarly, Akhmouch *et al.* documented a pregnant woman with minimal change disease, whose SPEP revealed a second albumin band during partial remission; that band vanished in complete remission and reappeared with relapse of NS.[15] The acquired bisalbuminemia observed in NS is generally transient and tends to parallel disease activity, supporting the concept of disease-related albumin modification rather than a fixed structural variant. This is consistent with Case 1, which shows variability between initial and follow-up SPEP results, as shown in Figure 1A-1B.

Additionally, Badr *et al.* described a series of diabetic patients (including one with NS) who exhibited albumin band variants; one patient even had both bisalbuminemia and bisalbuminuria. They observed that acquired bisalbuminemia is common in the setting of diabetes mellitus, consistent with our Case 2 findings.[4,16]

Multiple mechanisms can produce AFI in NS.[17] For example, chronic hyperglycemia as in Case 2 leads to nonenzymatic glycation of albumin; notably, Vladutiu showed that poor glycemic control in diabetic patients had a faster-migrating (more anodal) albumin band on electrophoresis compared to normal.[18] Conversely, uremia generates cyanate, which carbamylates lysine residues on albumin, adding negative charges. Favresse *et al.* demonstrated that carbamylated albumin shifts to a more cathodal position on capillary electrophoresis (altering the albumin peak's symmetry).[10] Such partial chemical modifications could create a distinct secondary albumin band if only a fraction of the albumin pool is modified. Supporting this concept, Gross *et al.* found that glycated and carbamylated albumin are biochemically distinct and more toxic to renal tubular cells than unmodified albumin.[19]

Oxidative modifications may also contribute to AFI in NS.[20] Albumin oxidation products – for example, ischemia-modified albumin (IMA) – are elevated during active NS and inversely correlate with serum albumin levels, suggesting that relapse episodes induce oxidative damage to albumin.[21]

Furthermore, biophysical studies indicate that albumin from NS patients differs in net charge and conformation. For instance, Haeri *et al.* found that albumin isolated from children with NS had altered size and electrophoretic behavior, which could enhance its glomerular filtration and potentially contribute to podocyte injury.[22] These observations imply that the nephrotic process itself can generate structurally modified albumin species capable of altering the SPEP pattern.

Extrinsic factors such as drugs can also produce albumin band irregularities. High-dose β-lactam antibiotics (e.g. penicillins, cephalosporins) form covalent adducts with albumin, sometimes creating a slower-moving "penalbumin" band on SPEP. Consistently, recent reports describe multiple cases of bisalbuminemia associated with β-lactam therapy (including patients with NS).[16,23] Other medications (e.g. dicloxacillin, tolmetin) and even peptide hormones (e.g. pegvisomant) have been linked to transient bisalbuminemia, as have conditions that alter albumin homeostasis (such as pancreatic pseudocysts or autoimmune diseases).[4,15]

Another consideration is the electrophoresis method. Capillary zone electrophoresis (CZE), as used in our cases, provides superior resolution for detecting subtle protein variants compared to agarose gels. Indeed, the adoption of CZE has increased the detection of bisalbuminemia; Jaeggi-Groisman *et al.* noted several cases of bisalbuminemia that were missed on traditional agarose electrophoresis but detected by CZE.[24] Therefore, laboratories should be aware that modern SPEP techniques may reveal benign albumin splits not seen with older methods.

Clinically, recognizing AFI patterns in patients with nephrotic syndrome is crucial to prevent misdiagnosis. For instance, a sharp peak, shoulder, or unusual band in the albumin region can be mistaken for a small monoclonal "M-spike," potentially triggering costly evaluations (free light-chain assays, imaging, or hematology consultation) and an unnecessary alarm. Conversely, understanding that such irregularities often stem from non-pathological factors (albumin variants or artifacts) and evaluating them with a systematic stepwise approach helps avoid these pitfalls.

Our proposed stepwise interpretative algorithm (Figure 2) synthesizes these insights into a practical diagnostic tool. This structured framework, which sequentially addresses technical errors, biochemical interferences, albumin modifications, and possible paraproteins, provides a reproducible and clinically applicable strategy that is not explicitly described in prior reports. By systematically ruling out benign causes, the algorithm can help avoid diagnostic dilemmas and unnecessary interventions when AFI are encountered.

Despite our careful evaluation, this case series has inherent limitations. Most obviously, only three patients were included, limiting the generalizability of our observations. Although such a small sample is acceptable for a case series, any conclusions drawn must be cautious. Additionally, we did not directly measure modified albumin forms; interpretations regarding glycated or carbamylated albumin were inferred from clinical parameters rather than confirmed by specific assays. The absence of a glycated albumin measurement in Case 2 is a notable gap, and genetic testing to definitively exclude hereditary bisalbuminemia was not performed.

## CONCLUSION

In summary, AFI in NS present a diagnostic challenge but are typically benign or transient phenomena once properly evaluated. Our cases illustrate that such anomalies often misinterpreted as pathological peaks can result from reversible albumin modifications or technical artifacts rather than monoclonal gammopathy. By applying a careful stepwise interpretative framework, clinicians and laboratory specialists can correctly identify AFI, avoid unnecessary investigations, and improve the accuracy of nephrotic syndrome evaluations.

## ETHICS STATEMENT

All patient-related data in this manuscript have been fully anonymized to protect confidentiality. According to the institutional policy of our hospital, case reports that do not include identifiable personal information are exempt from ethics committee approval. Therefore, both ethical approval and informed consent were waived for this study.

## REFERENCES

1. Lefrère B, Dedôme E, Garcia-Hejl C, Ragot C, Chianea D, Delacour H, et al. Bisalbuminemia: A case report. Rev Med Interne. 2018;39(12):950-4.
2. Faviou E, Nounopoulos C, Dionyssiou-Asteriou A. Bisalbuminemia from a clinical chemist's viewpoint: a case report and review of the recent literature. Minerva Med. 2006;97:287-93.
3. Kobayashi S, Okamura N, Kamoi K, Sugita O, Kamoi K. Bisalbumin (fast and slow type) induced by human pancreatic juice. Ann Clin Biochem. 1995;32:63-7.
4. Badr H, Young PE, Dong J, et al. Combined bisalbuminemia and bisalbuminuria: a rare finding on serum and urine electrophoresis. Clin Chim Acta. 2024;552:117635.
5. Hoang MP, Baskin LB, Wians FH Jr. Bisalbuminuria in an adult with bisalbuminemia and nephrotic syndrome. Clin

Chim Acta. 1999;284:101-7.

6. Mendler MH, Corbinais S, Sapey T, et al. In patients with cirrhosis, serum albumin determination should be carried out by immunonephelometry rather than by protein electrophoresis. Eur J Gastroenterol Hepatol. 1999;11:1405-11.
7. Regeniter A, Siede WH. Peaks and tails: evaluation of irregularities in capillary serum protein electrophoresis. Clin Biochem. 2018;51:48-55.
8. Arvan DA, Blumberg BS, Melartin L. Transient "bisalbuminemia" induced by drugs. Clin Chim Acta. 1968;22:211-8.
9. Ogawa M, Toda N, Kurahashi S, et al. Bisalbuminemia in nephrotic syndrome. Kidney Int. 2022;102:943.
10. Favresse J, Delanghe J. High-resolution capillary electrophoresis for the determination of carbamylated albumin. Clin Chem Lab Med. 2022;60(2):229-34.
11. Hellara I, Fekih O, Triki S, Elmay A, Neffati F, Najjar MF. Does bilirubin interfere with capillary electrophoresis of serum proteins? Ann Biol Clin (Paris). 2017;72:124-8.
12. Bagade S, Anjum A. Bisalbuminemia: A rare incidental finding in monoclonal gammopathy. EJIFCC. 2023;34(4):325-9.
13. Şeneş M, Alpdemir M, Kırtıl G, Öğüş E, Çolak Samsum E, Alpdemir MF. Effects of lipemia on capillary serum protein electrophoresis in native ultra-lipemic material and intravenous lipid emulsion added sera. Clin Chem Lab Med. 2022;61:1054-64.
14. Schneider W. "Albuminplasmocytoma" – a rare paper electrophoretic finding of a myeloma. Arztl Forsch. 1967;21(10):382-4.
15. Akhmouch I, Alayoud A, Bahadi A, et al. Bisalbuminemia during remission of nephrotic syndrome. Saudi J Kidney Dis Transpl. 2012;23(6):1251-3.
16. Ghosh A, Chowdhury N. An unusual case of bisalbuminemia in a 61-year-old male patient. 2025;121:60-1.
17. Lee P, Wu X. Modifications of human serum albumin and their binding effect. Curr Pharm Des. 2015;21.
18. Vladutiu AO. Faster electrophoretic mobility of albumin in diabetes. JAMA. 1980;244(1):27-8.
19. Gross ML, Piecha G, Bierhaus A, et al. Glycated and carbamylated albumin are more "nephrotoxic" than unmodified albumin in the amphibian kidney. Am J Physiol Renal Physiol. 2011;301(3):F476-85.
20. Mehta A, Shenoy R, BS D, Mundkur S, Biswas M. Electrophoretic pattern of serum and urinary proteins in nephrotic syndrome. Biomedicine. 2022;42:478-83.
21. Cakirca G, Guzelcicek A, Yilmaz K, Nas C. Increased ischemia-modified albumin levels in children with steroid-sensitive nephrotic syndrome. Pak J Med Sci. 2020;36(7).
22. Haeri HH, Eisermann J, Schimm H, et al. Profound changes in functional structure and dynamics of serum albumin in children with nephrotic syndrome: an exploratory research study. J Med Chem. 2023;66(17):12115-29.
23. Hadrach S. Bisalbuminemias caused by betalactamine antibiotic and nephrotic syndrome: case report. J Clin Case Stud Rev Rep. 2024;1:1-6.
24. Jaeggi-Groisman S, Byland CGH, Gerber H. Improved sensitivity of capillary electrophoresis for detection of bisalbuminemia. Clin Chem. 2000;46:882-3.
25. Levey AS, Stevens LA, Schmid CH, et al. Estimating GFR using the CKD-EPI creatinine equation: more accurate GFR estimates, lower CKD prevalence estimates, and better risk predictions. Am J Kidney Dis. 2010;55:622-7.

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