CASE SERIES

NATIONAL JOURNAL NJMR MEDICAL RESEARCH

Bisalbuminemia: Diagnostic Pearls from A Case Series

Sneha Wadalkar[1*], Shalini Maksane[2], Kavita More[3], Kshama Pimpalgoankar[4]

[1,2,3]Department of Biochemistry, MGM Medical College, Vashi, Navi Mumbai, India
[4]Department of Biochemistry, Sir HN Reliance Foundation Hospital, Mumbai, India

DOI:
10.55489/njmr.150320251135

***Corresponding author**:
Sneha Wadalkar
(Email: sneha.wadalkar@gmail.com)

Date of Submission: 30/04/2025
Date of Acceptance: 16/06/2025
Date of Publication: 01/07/2025

Funding Support:
None Declare

Conflict of Interest:
The authors have declared that no conflict of interests exists.

How to cite this article:
Wadalkar S, Maksane S, More K, Pimpalgoankar K. Bisalbuminemia: Diagnostic Pearls from A Case Series. Natl J Med Res 2025;15(03):213-219. DOI: 10.55489/njmr.150320251135

ABSTRACT

Bisalbuminemia (alloalbuminemia) is a rare, often incidental electrophoretic abnormality characterized by the presence of two distinct albumin bands, resulting from albumin variants with differing electrophoretic mobilities. Between the year 2020 and 2022, three cases of Bisalbuminemia were identified through serum protein electrophoresis (SPE) in varying clinical contexts. These included one confirmed case of multiple myeloma, one suspected case of monoclonal gammopathy, and one asymptomatic individual in whom Bisalbuminemia was detected incidentally during routine screening. Although typically benign, Bisalbuminemia may coexist with plasma cell disorders and has the potential to mimic or obscure monoclonal bands on electrophoretic analysis, leading to diagnostic challenges. Accurate recognition and interpretation of this phenomenon are essential to prevent misdiagnosis or overlooked pathology. These cases highlight the importance of correlating electrophoretic findings with clinical and laboratory data. While Bisalbuminemia itself is usually asymptomatic and benign, its presence in the context of monoclonal gammopathies raises questions about potential clinical significance. Further research is warranted to investigate whether this anomaly has any functional or pathological implications in plasma cell dyscrasias. Awareness of Bisalbuminemia among clinicians and laboratory professionals can improve diagnostic accuracy and patient management in cases involving abnormal protein electrophoresis.

Keywords: Bisalbuminemia, Serum Protein Electrophoresis, Multiple Myeloma, Immunofixation, Paraproteinemia, Albumin Variants, Monoclonal Gammopathy

INTRODUCTION

The primary and essential component of human plasma, albumin is synthesized in the liver and continuously secreted in healthy adult people at a rate of 14 grams per day with a half-life of roughly 19 days. [1,2] It constitutes approximately between 60 and 65 percent of all plasma proteins. Healthy adults have an albumin concentration with range of 3.5–5 g/dl, while in children it is around 2.9–5.5 g/dl. [3,4] Maintaining oncotic pressure and facilitating the movement of several endogenous and foreign substances are its primary functions. By attaching itself to the main free radical that causes damage, it also functions as an antioxidant. [5]

The genetic variants of human serum albumin have been studied to define their molecular defects and to correlate them to the functional properties and stability of the molecule. [1] Bisalbuminemia is an exceptional protein anomaly that is unintentionally discovered during standard serum electrophoresis.[1] Serum protein electrophoresis (SPE) is a fundamental diagnostic assay routinely employed in clinical laboratories to screen for monoclonal gammopathies. This technique allows for the separation of serum proteins based on their electrophoretic

Publisher: Medsci Publications [www.medscipublications.com] ISSN: 2249 4995 Official website: www.njmr.in

mobility, using platforms such as agarose gel electrophoresis, cellulose acetate membranes, or more advanced systems like capillary electrophoresis. The choice of method may vary depending on the laboratory setup and the diagnostic resolution required.[5] The normal electrophoretic pattern typically displays albumin as the most prominent peak, followed sequentially by five globulin fractions: alpha-1, alpha-2, beta-1, beta-2, and the gamma region. Albumin which usually appear as a tall, single, discrete peak on electrophoretogram. [5,6]

Bisalbuminemia is a rare electrophoretic anomaly with a reported incidence ranging from 1 in 1,000 to 1 in 3,000, and higher prevalence noted in specific populations, such as certain North American tribes. It manifests as two separate albumin peaks during serum protein electrophoresis on agarose gel electrophoresis (AGE) or a single widened albumin band in some cases. [5,7] Two separate Allo-albumin peaks are because of dissimilar mobilities of two albumin variants on electrophoresis where fast type variant is having increased electrophoretic mobility and slow type variant has decreased mobility. [8,9]

Two types of this rare condition are there, inherited or acquired. Single point mutation of human serum albumin gene results in Hereditary Bisalbuminemia.[10] The prevalence of acquired forms is unknown, but its presence has been described in various pathological conditions including diabetes mellitus, Waldenstrom's macroglobulinemia, multiple myeloma, sarcoidosis, Alzheimer's disease, pancreatic pseudocyst, nephrotic syndrome, chronic kidney disease, and also in patients receiving high doses of penicillin.[5,11]

In the majority of cases, individuals with Bisalbuminemia have serum albumin concentrations within the physiological range, and the coexisting albumin isoforms typically demonstrate preserved functional competency.[10] Because of its exceptional occurrence, Bisalbuminemia (Alloalbuminemia) is only of relevance to human genetics. No deleterious effects have been recorded in clinical settings.[10] In this study, during period of 2020 to 2022, we reported three instances of bifid albumin peaks which were accidentally found during serum protein electrophoresis.

CASE REPORTS

Case 1

A 57-year-old male patient presented to orthopaedic outpatient department (OPD)with complaints of multiple joint aches and generalised weakness. On examination, pallor (++) and koilonychia was observed. To evaluate anaemia further he was referred to department of medicine. Laboratory investigations revealed severe anaemia with value of haemoglobin around 6.8 gm% which is treated with blood transfusion. Other blood investigations, serum calcium and creatinine were normal. Liver function test results were within normal limits, with the exception of an elevated total protein level measured at 14.3 g/dL. X-ray findings were suggestive of Osteolytic lesions on lumbar spine and around distal end of both the femur bones This all-raised suspicion of multiple myeloma. To confirm this sample was sent to our laboratory for protein electrophoresis. Complete multiple myeloma panel was asked which comprise of protein electrophoresis, immunofixation electrophoresis (IFE), quantification of immunoglobulins (IgG, IgA, IgM), light chains (kappa, lambda) and Beta-2- Macroglobulin. On protein electrophoresis, M band with 44.9% spike, constituting 6.42 g/dl of total protein fraction, was observed with two spikes of albumin are of unequal peak suggestive of Bisalbuminemia. The total protein is 14.3 g/dl but albumin concentration is only 2.95 g/dl. IFE revealed bands of IgG-kappa which was co-relating with the quantification of Immunoglobulins and free light chains. Bone marrow examination suggestive of plasmacytosis. Patient was diagnosed as a case of Multiple Myeloma. Two distinct bands were also seen on IFE, which also supported presence of Bisalbuminemia. The patient was initiated on a combination chemotherapeutic regimen consisting of Lenalidomide, Bortezomib, and Dexamethasone, was subsequently transitioned to maintenance therapy upon achieving clinical stabilization. Bisalbuminemia persisted on all follow up electrophoretogram.

Case 2:

A 25-year-old male presented to surgery OPD with complaint of pain in left hypochondriac region and haematuria. He also complained of excessive fatigue in the last 3 months.

Table 1: Routine biochemical Investigations performed for all patients

Name of the analytes	Case-1	Case-2	Case-3	Normal reference interval
Hemoglobin	6.8	12.2	13.8	14 - 18 gm/dl
Serum Calcium	9.1	13.5	9.3	8.5 -10.2 mg/dl
Serum Creatinine	0.72	0.91	1.04	0.66 - 1.25 mg/dl
Serum Total Bilirubin	0.86	0.79	1.05	0.2 - 1.3mg/dl
Serum SGOT/AST	32.8	24.4	16.7	5 to 35 U/L
Serum SGPT/ALT	36.1	26	20.6	5 to 40 U/L
Serum Alkaline Phosphatase	115	84	76	38 - 126 U/L
Serum Total Protein	14.3	6.7	8.1	6.3 -8.2 gm/dl
Serum Albumin	2.95	4.06	4.75	3.5 - 5.0 gm/dl
Serum Globulin	11.35	2.64	3.35	2.5 - 3.5gm/dl
A/G ratio	0.26	1.54	1.42	1.1 -2.5 gm/dl

Table 2: Results of Myeloma panel of all the cases

Name of the analytes	Case-1	Case-2	Case-3	Reference Concentration
Serum Protein Electrophoresis				
Total Protein	14.3	6.7	8.1	6.3 -8.2 gm/dl
Albumin	2.95	4.06	4.75	3.30- 5.70 gm/dl
Alpha 1	0.57	0.19	0.23	0.10 – 0.40 gm/dl
Alpha 2	0.97	0.88	0.66	0.30 – 0.90 gm/dl
Beta 1	0.43	0.48	0.49	0.30 – 0.70 gm/dl
Beta 2	0.34	0.30	0.46	0.10 -0.50 gm/dl
Gamma	9.01	0.80	1.51	0.50 – 1.60 gm/dl
M band	Present	–	–	–
M spike (%)	44% (6.42 gm/dl)	–	–	–
Other bands	–	Faint band Observed	–	–
Immunofixation Fixation observation				
–	M band detected corresponding to IgG-kappa	Faint band observed corresponding to Kappa	No Monoclonal Gammopathy has been observed	–
Quantification of Heavy, light chains and B2M				
IgA	0.85	5.08 (High)	0.7	0.52 - 4.68 gm/L
IgG	108	10.3	4.3	6.5 - 16.4 gm/L
IgM	<0.19	0.45	0.45	0.39 - 3.38 gm/L
Kappa	452	115	8.51	3.30 - 19.40 mg/L
Lambda	38	60.7	8.33	5.71 - 26.30 mg/L
Kappa/Lambda ratio	11.89	1.89	1.02	0.26 - 1.65
Beta-2- Microglobulin	17479	975	1865	609 -2366 mg/L

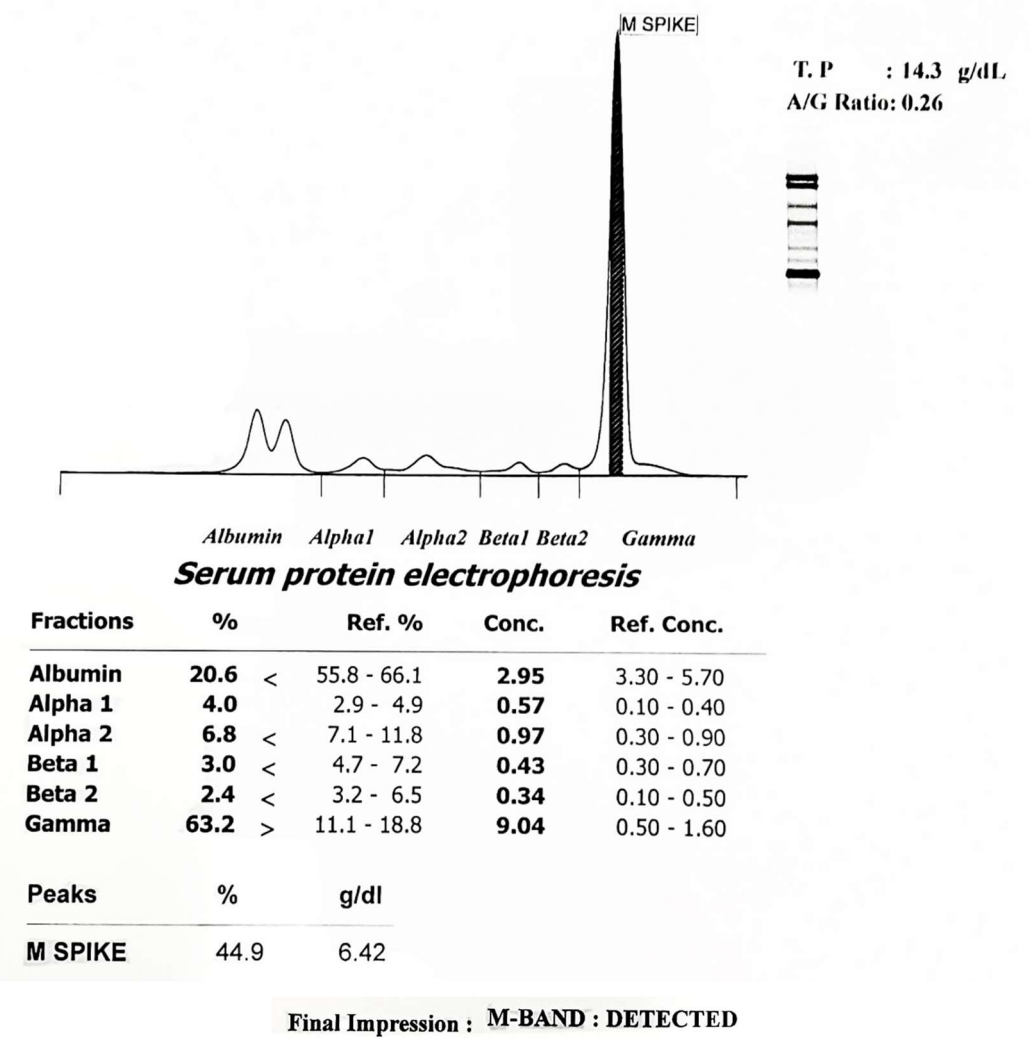
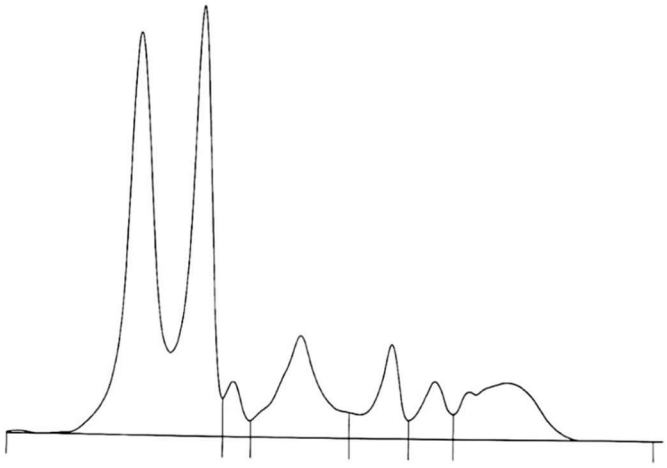


Figure 1: Serum protein electrophoresis profile of case 1 at the time of presentation

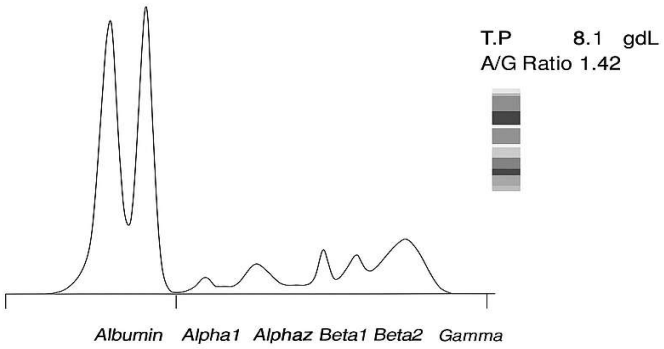


Serum protein electrophoresis

Fractions	%		Ref. %	Conc.	Ref. Conc.
Albumin	60.6		55.8 - 66.1	4.06	3.30 - 5.70
Alpha 1	2.8	<	2.9 - 4.9	0.19	0.10 - 0.40
Alpha 2	13.1	>	7.1 - 11.8	0.88	0.30 - 0.90
Beta 1	7.1		4.7 - 7.2	0.48	0.30 - 0.70
Beta 2	4.5		3.2 - 6.5	0.30	0.10 - 0.50
Gamma	11.9		11.1 - 18.8	0.80	0.50 - 1.60

Final Impression : FAINT BAND SEEN IN GAMMA GLOBULIN REGION

Figure 2: Serum protein electrophoresis profile of case 2 at the time of presentation



Serum protein electrophoresis

Fractions	%		Ref. %	Conc.	Ref. Conc.
Albumin	58.7		55.8-66.1	4.75	3.50 - 5.70
Alpha 1	2.8	<	2.9 - 4.9	0.23	0.10 - 0.40
Alpha 2	8.1		7.1-11.8	0.56	0.30 - 1.00
Beta 1	1.0		3.2 - 7.6	0.46	0.40 - 0.80
Beta 2	5.7		3.2 - 6.5	0.46	0.40 - 1.00
Gamma	18.7		11.1-16.8	1.51	0.50 - 1.60

Final Impression : M-BAND NOT DETECTED

Figure 3: Serum protein electrophoresis profile of case 3 at the time of presentation

X-ray and ultrasonography confirmed presence of multiple calculi in left kidney. He had history of nephrectomy 3 years back to remove renal stones. Stone analysis shown presence of calcium oxalate and calcium phosphate. Blood investigations showed high calcium level (13.5 mg/dl) and marginal low haemoglobin, other investigations like serum creatinine, liver function tests were within normal limit. To rule out suspicion of Multiple myeloma, protein electrophoresis was performed. A faint band has been observed on serum protein electrophoresis which corresponds to Kappa light chain with concentration of 115 mg/dl. The extra band in albumin region

has been observed in this patient suggestive of Bisalbuminemia. As per discussion with treating clinician it was an incidental finding and there was no clinical or therapeutic impact. This patient was started with treatment of Bortezomib and followed up regularly.

Case 3:

A 67 years old male underwent regular health check-up. He was known a case of Diabetes and hypertension and was on treatment for the same. His fasting blood glucose, serum creatinine, liver function tests were within normal limit. His serum sample was received for protein electrophoresis as the test was a part of health check-up panel for age group above 60 years of age. Total protein is within normal limit. There was no monoclonal gammopathy has been observed in gamma region, but albumin region showed presence of two distinct peaks. Two distinct bands were observed on IFE as well confirming presence of Bisalbuminemia. The clinician was contacted and informed regarding presence of albumin variant. Unfortunately, we were unable to collect further data for this patient.

DISCUSSION

Two albumin peaks on blood protein electrophoresis, a regular test used mostly for the diagnosis of monoclonal gammopathies, is a characteristic of Bisalbuminemia. [5,12] Scheurlen initially reported this condition in 1955 in a German patient with diabetes, although the band was only temporary. [5,7] Shortly thereafter, Knedel, 1957; Nennstiel and Becht, 1957; Earle et al., 1958; & Wuhrmann, 1959 reported stable heterozygotic bands. [14-16]

Epidemiologically, the low incidence rate (0.01%) reported by Kapatia G et al. [7] underscores Bisalbuminemias rarity but also suggests under-recognition due to previous methodological limitations. Notably, the occurrence of Bisalbuminemia in nearly half of these cases with hypergammaglobulinemia introduces a potential association with plasma cell dyscrasias or inflammatory states, though causality remains speculative.[7]

Serum protein electrophoresis is frequently carried out in patients with suspected myelomas, which is probably why Bisalbuminemia has been identified in these patients.[17] A number of illnesses are included in the differential diagnosis when another band appears in the same area as albumin. The first of them is that the band could be an electrophoresis artifact, which typically occurs in gel electrophoresis as a result of air bubbles, gel deformities, overloading, etc. These possibilities are ruled out because of capillary system for electrophoresis. With the advent of capillary electrophoresis, which provides superior resolution, Bisalbuminemias are increasingly commonly observed.[5,18] Second, Bisalbuminemia may manifest as increased proteins that typically travel in the same location of albumin. These consist of alpha lipoproteins, alpha 1 acid glycoprotein (an acute phase reactant), and prealbumin, which is elevated following recent food consumption.[5,19] Nevertheless, in rare cases, certain albumin variations may have a changed affinity for certain hormones, including thyroxine, metal ions, fatty acids, and medications, which could have therapeutic implications.[18] The pathophysiological link or association between multiple myeloma and Bisalbuminemia is unknown. [5]

In the present study, biochemical analyses were conducted using Chemiluminescence (CLIA) techniques on beta-2-microglobulin, immunoglobulins (heavy and light chains) on nephelometry (BN II, Siemens), and chemistry autoanalyzer. Sebia capillary electrophoresis was used to perform serum protein electrophoresis. Using Hydrasys 2 scan, Sebia, immunofixation electrophoresis (IFE) was used to verify heavy and light chains. Regular biochemical analyses were conducted using a biochemistry autoanalyzer.

The bifid electrophoretic pattern characteristic of Bisalbuminemia results from differential migration of albumin variants during electrophoresis, a phenomenon that demands careful differentiation from other proteins or artifacts that might mimic such patterns.[8,9] Misinterpretation risks are heightened in the context of monoclonal gammopathies, where paraprotein bands may overlap or obscure albumin fractions, complicating accurate diagnosis.[20,21] This diagnostic challenge necessitates heightened vigilance and the use of confirmatory methods such as immunofixation electrophoresis (IFE).[21,22] The present analysis of three cases demonstrates this complexity. Case 1, with a definitive diagnosis of multiple myeloma (MM), exhibited persistent Bisalbuminemia alongside a pronounced M spike and elevated biomarkers (Table no.1,2 and figure1) indicating high disease burden.[23] The acquired nature of Bisalbuminemia in this context likely reflects structural albumin modifications induced by paraproteins or post-translational alterations, a hypothesis supported by the absence of congenital variants and persistence over follow-up.[1,24] This suggests a potential pathophysiological interplay between paraprotein dynamics and albumin structure that merits further molecular investigation.

In contrast, Case 2 (Table no. 1,2 and figure 2) presented with equivocal laboratory findings consistent with monoclonal gammopathy of undetermined significance (MGUS) or early myeloma, wherein Bisalbuminemia appeared incidental and lacked therapeutic implications.[17] This supports the notion that Bisalbuminemia may coexist without influencing clinical course or requiring intervention.[7] Case 3's (Table no.1,2 and figure 3) entirely asymptomatic presentation with Bisalbuminemia further exemplifies the incidental nature of this anomaly, possibly attributable to benign congenital variants or artifacts not fully elucidated due to limited follow-up.

Collectively, these observations emphasize that Bisalbuminemia, while predominantly benign, can emerge in varied clinical contexts and potentially confound electrophoretic interpretation. Given the evolving sensitivity of diagnostic assays, Bisalbuminemia's increased detection

necessitates critical appraisal to distinguish between innocuous variants and those with possible pathological or therapeutic significance.

These cases underscore the necessity for vigilance in interpreting electrophoretic patterns, especially when uncommon findings such as Bisalbuminemia are encountered. While often considered benign or incidental, its presence particularly in patients with monoclonal gammopathies may offer additional clues regarding paraprotein behaviour, albumin interactions, or even the biochemical complexity of the disease process itself.[25] Further investigation is needed to elucidate the underlying mechanisms of Bisalbuminemia, especially in instances where no clear etiological factor can be determined.[26] Exploring links between Bisalbuminemia and metabolic or hematologic disorders may reveal novel associations, enhancing diagnosis and treatment strategies.[26] Ultimately, these cases highlight how a seemingly subtle laboratory anomaly can serve as a window into broader systemic pathology, emphasizing the critical role of laboratory diagnostics in clinical decision-making.

CONCLUSION

These three cases illustrate the diverse clinical relevance of Bisalbuminemia detected on serum protein electrophoresis. Case 1 links persistent Bisalbuminemia with multiple myeloma, while Cases 2 and 3 show incidental findings without clear plasma cell disorders. This highlights the need for close clinician-laboratory collaboration in interpreting such anomalies. Though often benign or hereditary, Bisalbuminemia may signal underlying pathology and should prompt further myeloma workup when associated with monoclonal gammopathy or abnormal globulin profiles.

Author contribution: SW was responsible for the study conception, study design, data collection, data analysis and interpretation, as well as manuscript preparation. **SM** contributed to the study conception, study design, data analysis and interpretation, and manuscript preparation. **KM and KP** both participated in the study design and manuscript preparation.

REFERENCES

1. Caridi G, Lugani F, Angeletti A, Campagnoli M, Galliano M, Minchiotti L. Variations in the human serum albumin gene: molecular and functional aspects. International Journal of Molecular Sciences. 2022 Jan 21;23(3):1159. DOI: https://doi.org/10.3390/ijms23031159 PMid:35163085 PMCid:PMC8835714
2. Peters T. All about albumin: Biochemistry, genetics, and medical applications. San Diego (CA): Academic Press; 1996.
3. Raoufinia R, Mota A, Keyhanvar N, Safari F, Shamekhi S, Abdolalizadeh J. Overview of albumin and its purification methods. Advanced Pharmaceutical Bulletin. 2016 Dec;6(4):495-507. DOI: https://doi.org/10.15171/apb.2016.063 PMid:28101456 PMCid:PMC5241407
4. Kragh-Hansen U, Minchiotti L, Galliano M, Peters T. Human serum albumin isoforms: genetic and molecular aspects and functional consequences. Biochimica et Biophysica Acta.2013;1830(12):5405-5417. DOI: https://doi.org/10.1016/j.bbagen.2013.03.026 PMid:23558059
5. Bagade S, Anjum A. Bisalbuminemia: A Rare Incidental Finding in Monoclonal Gammopathy. EJIFCC. 2023 Dec 21;34(4):325-329. PMID: 38303749; PMCID: PMC10828532.
6. O'Connell TX, Horita TJ, Kasravi B. Understanding and interpreting serum protein electrophoresis. Am Fam Physician. 2005 Jan 1;71(1):105-12. PMID: 15663032.
7. Kapatia G, Wadhwa M, Malhotra P, Prakash G, Aggarwal R. Bisalbuminemia: A Pathologist's Insight of an Uncommon Phenomenon. Journal of Laboratory Physicians. 2021 Sep;13(3):219-223. DOI: https://doi.org/10.1055/s-0041-1730878 PMid:34602785 PMCid:PMC8478498
8. Chhabra S, Bansal F, Saikia B, Minz RW. Bisalbuminemia: a rarely encountered protein anomaly. Journal of Laboratory Physicians. 2013 Jul; 5(2):145-6. DOI: https://doi.org/10.4103/0974-2727.119869 PMid:24701116 PMCid:PMC3968632
9. Kobayashi S, Okamura N, Kamoi K, Sugita O. Bisalbumin (fast and slow type) induced by human pancreatic juice. Ann Clin Biochem. 1995 Jan;32 (Pt 1):63-7. DOI: https://doi.org/10.1177/000456329503200105 PMid:7762952
10. Avgoustou E, Kounatidis D, Vallianou NG, Karampela I, Stratigou T, Dalamaga M. Incidental detection of hereditary Bisalbuminemia in a patient with positive DAT Coombs: A case-based review. Metabolism Open.2024 Sep; 23:100307. DOI: https://doi.org/10.1016/j.metop.2024.100307 PMid:39185032 PMCid:PMC11342109
11. Mark A. Valasek, Richard T. Hopley, Frank H. Wians. Resolving the Baneful and Banal:Bisalbuminemia in an Adult with Waldenström Macroglobulinemia. Laboratory Medicine. 2008; 39(12): 723-726. DOI: https://doi.org/10.1309/LMD51RR5FXHKDHRX
12. Agarwal P, Parkash A, Tejwani N, Mehta A. Bisalbuminemia: A Rare Finding on Serum Electrophoresis. Indian journal of Hematology & blood transfusion: an official journal of Indian Society of Hematology and Blood Transfusion. 2018; 34(3): 558-559. DOI: https://doi.org/10.1007/s12288-017-0911-z PMid:30127575 PMCid:PMC6081336
13. Knedel M. Double albuminemia, a new hereditary protein anomaly. Blut. 1957; 3:129-131. DOI: https://doi.org/10.1007/BF01630687 PMid:13446004
14. Nennstiel HJ, Becht T. On the hereditary occurrence of an albumin split in the electrophoresis diagram. Clinical Weekly.1957; 35:68. DOI: https://doi.org/10.1007/BF01481274 PMid:13515039
15. Earle DP, Hutt MP, Schmid K, Gitlin D. A unique human serum albumin transmitted genetically. Trans Assoc Am Physicians. 1958;71:69-76. PMID: 13603504.
16. Wuhrmann F. Double albumin peaks as a hereditary blood protein anomaly. Swiss Medical Weekly.1959; 89:150-152.
17. Chan PC, Yasodhara A, Truong D. Bisalbuminemia: Lack of association with monoclonal gammopathy and value justification for reporting in serum protein electrophoresis. J Clin Chem Lab Med. 2017; 1(1): 108.
18. Tarik Adioui, Hasna Igorman, Imane Radouane, Salma Ouahid, Sanaa Berrag, Mouna Tamzaourte. A Rare Bisalbuminemia Etiology: Cholestatic Hepatitis Case Report. Asian Journal of Research and Reports in Gastroenterology. 2023; 7(1):1-5.
19. Angouridaki C, Papageorgiou V, Tsavdaridou V, Giannousis M, Alexiou-Daniel S. Detection of hereditary bisalbuminemia in a Greek family by capillary zone electrophoresis. Hippokratia. 2008 Apr;12(2):119-21. PMID: 18923658; PMCID: PMC2464307.
20. Pavithra, H.K., Tahlan, A., Agrawal, N., & Singh, R. Bisalbuminemia: Complicating the Diagnosis of Multiple Myeloma. IAR Journal of Medical Science. 2020; 1(6): 310-311.
21. Jonathan R. Genzen, David L. Murray, Gyorgy Abel, Qing H. Meng, Richard J. Baltaro, Daniel D. Rhoads, Julio C. Delgado, Rhona J.

Souers, Christine Bashleben, David F. Keren, Mohammad Q. Ansari; Screening and Diagnosis of Monoclonal Gammopathies: An International Survey of Laboratory Practice. Arch Pathol Lab Med 1 April 2018; 142 (4): 507-515. DOI: https://doi.org/10.5858/arpa.2017-0128-CP PMid:29266967

22. McPherson RA, Pincus MR. Henry's Clinical Diagnosis and Management by Laboratory Methods. 24th edition. Elsevier; 2021.

23. Kyle RA, Rajkumar SV. Multiple Myeloma. Blood. 2008;111(6):2962-2972. DOI: https://doi.org/10.1182/blood-2007-10-078022 PMid:18332230 PMCid:PMC2265446

24. Wadalkar S, Maksane S, More K, Pimpalgoankar K. BISALBUMINEMIA: Still unrevealed for clinicians and researchers - A systemic review. Asian J Pharm Clin Res. 2024;17(9):1-7. DOI: https://doi.org/10.22159/ajpcr.2024v17i9.51644

25. Kalambokis G, Kitsanou M, Kalogera C, Kolios G, Seferiadis K, Tsianos E. Inherited Bisalbuminemia with Benign Monoclonal Gammopathy Detected by Capillary but not Agarose Gel Electrophoresis, Clinical Chemistry.2002;48(11):2076-2077. DOI: https://doi.org/10.1093/clinchem/48.11.2076 PMid:12407005

26. Kumar P. Bisalbuminemia: Insights into its Clinical Manifestations, Diagnostic Complexities, and Relevance in Modern Medicine. Int. J. Medi. Sci. Res. Practi. 2024 Dec. 31;11(4):11-14. Available from: https://ijmsrp.isroset.org/index.php/j/article/view/78