

Case Report: Asymptomatic Congenital Bisalbuminemia in a 24-Year-Old Male

Afif Ba^{1,2,*}, Ines Khadhraoui^{1,3}, Mariem Belhedi⁴, Manel Ayoub^{1,2}, Sana Aboulkacem^{1,3}, C Mazigh^{1,2}

¹Biochemistry Department, Main Military Hospital of Tunis, Tunisia

²Faculty of Pharmacy, Monastir, Tunisia

³Faculty of Medicine, Tunis, Tunisia

⁴Laboratory department, Habib Thameur Hospital, Tunis, Tunisia

ABSTRACT

Bisalbuminemia is a rare inherited or acquired condition characterized by the presence of two distinct albumin bands on electrophoresis. While typically benign, its recognition is crucial to avoid misinterpretation of laboratory results and unnecessary investigations. We present a case of asymptomatic congenital bisalbuminemia discovered incidentally in a 24-year-old male during a routine health check-up. This report highlights the importance of recognizing this benign condition and differentiating it from pathological causes of abnormal albumin patterns.

Keywords: Bisalbuminemia, Congenital, Asymptomatic, Albumin Electrophoresis, Benign Condition.

INTRODUCTION

Bisalbuminemia, first described by Scheurlen in 1955, is a phenomenon where two distinct albumin fractions are observed during serum protein electrophoresis [1]. It can be classified into congenital (inherited) and acquired forms. Congenital bisalbuminemia is an autosomal dominant trait, usually clinically silent, and often discovered incidentally [2]. Acquired forms can be transient and associated with conditions such as pancreatic disease, renal failure, or certain drug therapies [3]. This case report describes an incidental finding of congenital bisalbuminemia in an otherwise healthy young male.

CASE PRESENTATION

A 24-year-old male presented for a routine pre-employment health screening. He reported no significant past medical history, denied any chronic illnesses, hospitalizations, or regular medication use. He was asymptomatic, denied any fatigue, edema, unexplained weight changes, or gastrointestinal symptoms. His family history was non-contributory, with no known inherited disorders.

Physical examination was unremarkable. His vital signs were within normal limits, and systemic examination revealed no abnormalities. Routine laboratory investigations included a complete blood count, liver function tests, renal function tests, and serum protein electrophoresis. All parameters were within normal ranges, except for the serum protein electrophoresis.

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*Corresponding Author

Dr. Afif Ba,

Biochemistry Department, Main Military Hospital of Tunis, Tunisia, Tel: 0021658641541, E-mail: afifba173@gmail.com

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Investigations

Serum protein electrophoresis revealed two distinct

albumin bands of approximately equal intensity, migrating at different rates (Figure 1).

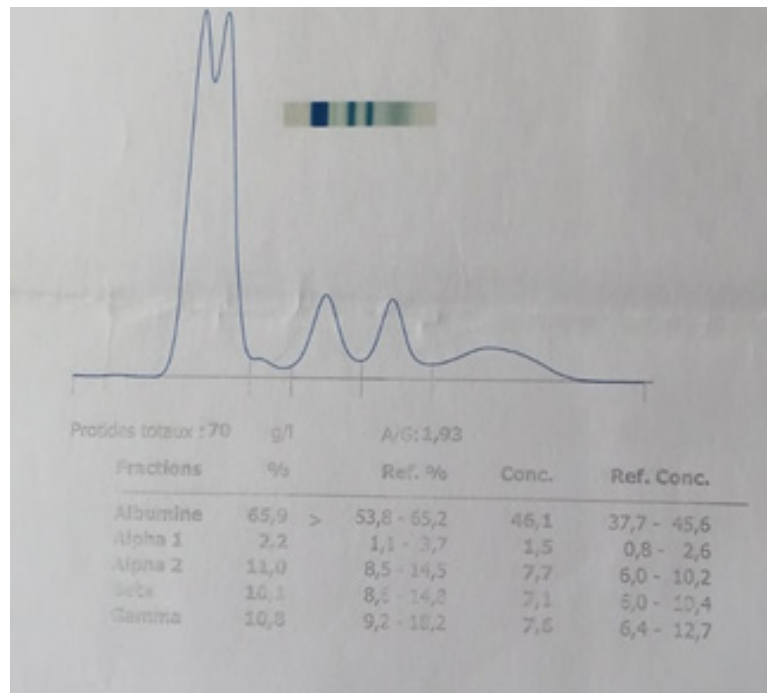


Figure 1. Serum electrophoresis trace of the patient.

The total serum protein and quantitative albumin levels were within the normal reference range. Other serum protein fractions (alpha-1, alpha-2, beta, and gamma globulins) were also normal. To confirm the finding, repeat serum protein electrophoresis was performed on a fresh sample, yielding identical results.

Given the asymptomatic nature of the patient and the consistent finding of two albumin bands, a diagnosis of bisalbuminemia was suspected. Family studies were suggested but could not be performed due to logistical constraints. However, in the absence of any underlying pathology and the stable nature of the finding, congenital bisalbuminemia was the most likely diagnosis.

Differential Diagnosis

The differential diagnosis for two albumin bands on electrophoresis primarily includes congenital bisalbuminemia and acquired bisalbuminemia [2,3].

Acquired forms are typically transient and can be associated with:

- Pancreatic disease (e.g., acute pancreatitis)
- Renal failure
- Drug-induced causes (e.g., penicillins, sulfonamides, certain anti-inflammatory drugs forming drug-albumin complexes)
- Hyperthyroidism (rarely reported)
- Interference from plasma expanders

In this case, the patient's excellent health, absence of symptoms, normal organ function, and lack of medication use ruled out acquired causes.

A family investigation was carried out with serum protein electrophoresis in the patient's mother and sister and a trace suggesting bisalbuminemia was found in the mother (Figure 2).

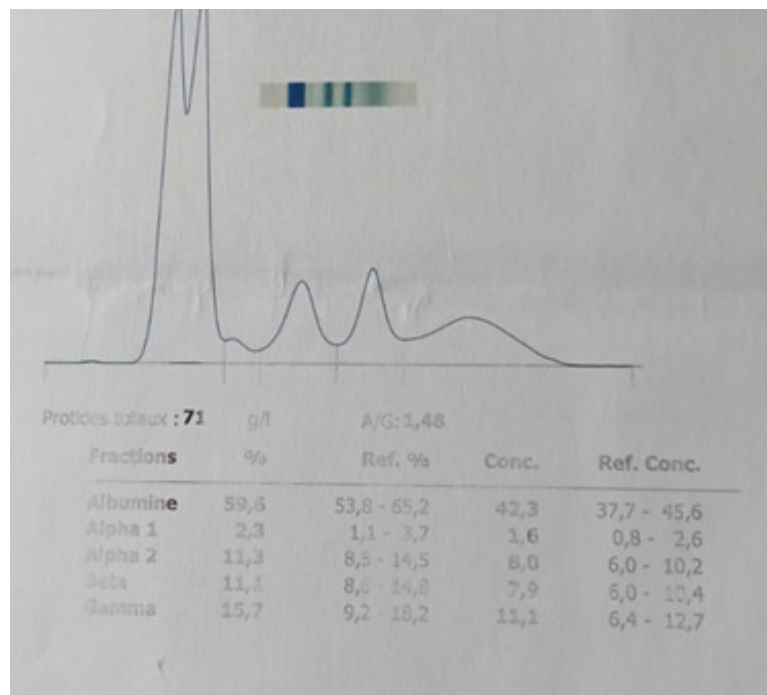


Figure 2. Serum electrophoresis trace of the patient's mother.

Outcome

The patient was reassured that the bisalbuminemia was a benign, congenital condition with no clinical significance, requiring no further intervention or follow-up. He was advised to inform future healthcare providers about this finding to prevent unnecessary investigations.

DISCUSSION

HCongenital bisalbuminemia is a rare genetic anomaly, with an estimated prevalence ranging from 1 in 1,000 to 1 in 10,000 individuals, varying among populations [2]. It is inherited in an autosomal dominant pattern, implying that affected individuals typically have one normal and one variant albumin allele. The structural difference in the variant albumin is usually a single amino acid substitution, which alters its charge and thus its electrophoretic mobility [4].

The clinical significance of congenital bisalbuminemia is generally negligible. The two albumin variants perform their physiological functions normally, including transport of various molecules, maintenance of oncotic pressure, and buffering of pH. Therefore, individuals with this condition are typically asymptomatic, as seen in our patient [2,3].

The importance of recognizing bisalbuminemia lies in avoiding misinterpretation of laboratory results and preventing unnecessary and often invasive diagnostic procedures. If bisalbuminemia is not recognized, the presence of two albumin bands could be mistakenly attributed to a monoclonal gammopathy, an abnormal immunoglobulin, or

a technical artifact, leading to further investigations such as immunofixation electrophoresis, bone marrow biopsy, or extensive imaging studies [5].

When bisalbuminemia is identified, a thorough clinical history and physical examination are crucial to rule out acquired causes. If the patient is asymptomatic and there are no signs or symptoms of an underlying disease, congenital bisalbuminemia is highly probable. Confirmation often involves family studies to identify the trait in relatives [2]. However, in an isolated, asymptomatic case, the diagnosis can be made clinically after excluding acquired causes.

Although definitive identification of the mutation responsible for bisalbuminemia provides limited clinically useful information, more than 60 different albumin variants have been characterized in human populations over the last three decades, the majority of which are caused by single-base changes in the structural gene [6]. These mutations are frequently observed at hypermutable CpG dinucleotides. In rare instances, the resulting condition of bisalbuminemia carries clinical consequences due to the mutation's effect on ligand-binding. Three mutations in particular—p.Leu90Pro, p.Arg242His, and p.Arg242Pro—induce a high binding affinity for thyroid hormones (T3 and T4). This aberrant binding capacity causes two distinct clinical syndromes: familial dysalbuminemic hyperthyroxinemia and familial dysalbuminemic hypertriiodothyroninemia [7,8]. Furthermore, rare cases of bisalbuminuria associated with bisalbuminemia underscore the limited utility of urinary protein electrophoresis in diagnosing bisalbuminemia [9].

CONCLUSION

This case report serves as a reminder of the existence of congenital bisalbuminemia, a benign genetic variant of albumin that is usually discovered incidentally. Healthcare professionals should be aware of this condition to prevent misdiagnosis, anxiety for the patient, and the expenditure of resources on unnecessary investigations. In an asymptomatic individual with two distinct albumin bands on electrophoresis, and no evidence of underlying pathology, a diagnosis of congenital bisalbuminemia is highly likely.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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