# An In-depth Multidisciplinary Investigation in a Young Athlete Suffering from Syncope Caused by Myocardial Bridge

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#### Introduction

Laboratory medicine, in conjunction with genetic studies in sports medicine, is becoming increasingly crucial in monitoring athletes' health issues. Acute or intensive exercise can cause metabolic imbalances, muscular damage and the discovery of cardiovascular problems. The purpose of this study was to track the health of a basketball player using an integrated strategy that included biochemical and genetic studies as well as modern imaging methods in order to shed light on the reasons of recurrent syncope he experienced after activity. Biochemical tests revealed that the athlete's iron, ferritin and bilirubin levels were abnormal. Coronary CT Angiography revealed the presence of an intramyocardial bridge, implying that this was the source of the reported syncopes.

### Description

Laboratory medicine and medical genetics have evolved into specialties required for athletes' health monitoring. Intense physical exercise is known to induce cardiovascular diseases, thrombotic events, muscle damage and infections, particularly in susceptible individuals. The health state of competitive athletes must be monitored in order to assess the stress caused by rigorous physical exercise. Athlete laboratory monitoring assists physicians and sports trainers in developing focused training and recovery regimens to prevent injuries or irreversible harm. Recently, in order to explore athletes' health, the scientific community has found biomarkers helpful as an alarm bell for athlete protection in order to early highlight urinary infections; immune system activation, brain damage and muscular injuries heart problems, hormonal and vitamin imbalances [1].

Furthermore, cardiovascular examination and diagnostic imaging capable of recognizing heart diseases that are possibly lethal or muscle injuries, which would be difficult to diagnose at the medical/laboratory examination alone, are essential for the right clinical evaluation of athletes. At the same time, it is recognized that some kinds of hereditary/congenital cardiac disease or metabolic disorders, which can result in major accidents such as sudden death, may be missed in the absence of a family history and/or particular symptoms. As a result, the necessity to identify all elements that might characterize an athlete's phenotype is becoming obvious, shedding light on metabolic features as well as the mechanisms of adaptation and reaction of the particular athlete to the stress generated by severe physical exercise [2].

The use of biochemical, hematological and genetic testing to detect risk factors in athletes is becoming more popular at the competitive and elite levels. We examined changes in biochemical, hematological and instrumental

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cardiovascular parameters, as well as in-depth genetic analysis, to shed light on the aetiology of recurring syncope in an athlete who looked to be in excellent condition but had a few episodes of syncope. The biochemical study revealed an iron deficit. Iron insufficiency can occur in competitive athletes, especially adolescent female athletes. Regular and, more importantly, highintensity exercise increases iron losses in athletes by up to 70% as compared to inactive populations due to intense sweating and increased blood loss in urine and GI tract [3].

Inadequate dietary iron intake, on the other hand, may contribute to iron insufficiency in athletes. Ferritin is a sensitive and specific indicator of iron insufficiency. Ferritin is the primary iron storage protein within cells and its concentration in blood reflects the body's mineral stores. Although ferritin values less than 15 mg/mL are considered diagnostic for iron insufficiency in the general population, athletes should aim for ferritin levels of at least 30–40 mg/mL because they require more iron than less active patients. Our basketball player had iron deficiency without anemia, which required oral iron supplements and control blood tests after a minimum of three months of treatment [4].

At the same time, biochemical tests indicated that the athlete had Gilbert's syndrome, as demonstrated by hyperbilirubinemia and an increase in unconjugated bilirubin. Furthermore, NGS sequencing enabled genotyping of the (TA) polymorphism (rs3064744) in the UGT1A1 gene's TATA box. In Gilbert's patients, TA insertions in the TATA box of the UGT1A1 promoter are related with hyperbilirubinaemia. Gilbert syndrome is a genetic disorder that causes moderate hyperbilirubinemia. Common genetic variations have been linked to the beginning of illnesses, including cardiovascular disease.

Furthermore, the athlete had a vitamin D deficit, which was especially noticeable after a month of consistent high-intensity exercise. According to some publications, vitamin D deficiency is widespread among basketball players. Furthermore, multiple studies have shown that Vitamin D shortage in athletes is related with enhanced Vitamin D receptor expression in skeletal muscles, resulting in high intake of Vitamin D but low circulating levels. Finally, the lower levels in the second sample (collected in November) compared to the first (collected in September) might be attributed to strong circannual oscillations of vitamin D. Our findings support the notion that vitamin D circulating levels [5].

#### Conclusion

In today's professional sports, a multidisciplinary approach is required to monitor players and measure the stress that severe physical activity might produce. As a result, in addition to the athlete's medical history, the sports medical community now has a responsibility to integrate biochemical and hematological tests, which must be supported by instrumental evaluation, in order to create an overview of the athlete's health state. When extensive clinical, laboratory and instrumental assessments fail to identify a definite phenotype or examine the genetics and pathological processes of a recognized pathological disease, genetic evaluation may play a role.

## **Conflict of Interest**

None.

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