Brugada syndrome (BrS) is an inherited channelopathy which is usually caused by mutation in the SCN5A gene. The coved ST-segment elevation in the right precordial leads is diagnostic and can predispose to polymorphic ventricular tachycardia (VT) and sudden death (figure 1). In Ireland, BrS cases with secondary prevention implantable cardioverter defibrillators (ICDs) are unable to drive. This recommendation was based on the consensus statement of the European Heart Rhythm Association in 2009. However, increasing data suggests that educating patients to avoid heavy meals and alcohol before sleep, prompt treatment of pyrexia, and avoid certain drugs greatly reduces risk of VT. In addition, Quinidine and more recently epicardial ablation have proven effective. Furthermore, our threshold to implant a device is higher, due to a clearer definition of arrhythmogenic syncope and the reduced use of programmed electrical stimulation (PES). These developments prompted a review of current national guidelines, and our own data.

Introduction

Brugada syndrome (BrS) is an inherited channelopathy which is usually caused by mutation in the SCN5A gene. The coved ST-segment elevation in the right precordial leads is diagnostic and can predispose to polymorphic ventricular tachycardia (VT) and sudden death (figure 1). In Ireland, BrS cases with secondary prevention implantable cardioverter defibrillators (ICDs) are unable to drive. This recommendation was based on the consensus statement of the European Heart Rhythm Association in 2009. However, increasing data suggests that educating patients to avoid heavy meals and alcohol before sleep, prompt treatment of pyrexia, and avoid certain drugs greatly reduces risk of VT. In addition, Quinidine and more recently epicardial ablation have proven effective. Furthermore, our threshold to implant a device is higher, due to a clearer definition of arrhythmogenic syncope and the reduced use of programmed electrical stimulation (PES). These developments prompted a review of current national guidelines, and our own data.

Methods

Retrospective analysis of BrS patients who attend an adult inherited cardiac conditions clinic (Tallaght University Hospital and Mater Misericordiae University Hospital). Local records and the national device database Heart rhythm Ireland were utilised.

Results

A total of 10 BrS patients with ICDs were identified. Mean age was 50 years, and 7 were male. One case underwent implantation for primary prevention due to high-risk features. Regarding the secondary prevention cohort; 2 had survived a cardiac arrest, 3 had syncope without documented arrhythmia, and 4 had non-sustained VT or a positive PES. A subcutaneous ICD was utilised for 2 patients. Mean follow up time since implant was 7 years and 3 months. Defibrillation therapy was required for 2 patients, each of which was appropriate and occurred during sleep. Both have been free of ventricular arrhythmias since commencement of Quinidine or having undergone an epicardial ablation.

Conclusion

BrS secondary prevention ICDs have rarely been performed in Ireland. In terms of risk when driving, ventricular arrhythmias only occurred during sleep, a pathognomonic trigger in BrS. Given the improved understanding of BrS and the development of effective treatments, several countries have removed the indefinite driving restriction for a category one license. Our national data is consistent with this trend and can now be included in a review of Irish driving recommendations.
transferred to a tertiary cardiology centre for electrophysiology studies. An ajmaline test was negative, although ventricular ectopics were noted. There were no ectopics elicited during electrophysiology studies. There was a suggestion of a subtle J wave on a number of her ECGs and intermittent short PR interval (figure 2), which would imply an early repolarisation syndrome such as J wave syndrome. Of note, during her stay in the coronary care unit, she developed intermittent tongue swelling and generalised urticarial rash which required treatment with steroids and antihistamines. She was transferred to a specialist centre, for further diagnostics including cardiac magnetic resonance imaging, electrophysiology studies and genetic screening for long QT syndrome. These investigations were all unremarkable, including a negative ajmaline test. A single chamber transvenous implantable cardiac defibrillator was inserted and she was discharged with beta-blockade.

Discussion
Polymorphic ventricular tachycardia, has a multitude of causative factors including QT prolonging drugs, cardiac ischaemia, underlying genetic arrhythmias such as and catecholaminergic polymorphic ventricular tachycardia (CPVT) and inherited sodium and potassium channel mutations, most notably, long QT syndromes and Brugada syndrome. It can also be as a result of early repolarisation syndromes such as J wave syndrome. Myocarditis has now been linked to the covid vaccine, with a generally benign course of illness observed. It is unclear in our case, whether an underlying genetic predisposition, in combination with the covid vaccine and medications which can cause prolonged QT intervals, provoked this episode of polymorphic ventricular tachycardia. This patient, had never observed cardiac symptoms including chest pain or palpitations, leading a very active lifestyle prior to this event. We suspect an underlying early repolarisation syndrome, as a potential precipitant of this cardiac arrest. There has been an estimated rate of 11.1 cases of anaphylaxis, per 1 million Pfizer-BioNTech Covid-19 vaccines. Cardiovascular compromise, due to anaphylaxis, is well described in the acute setting. This patient had symptoms of a prolonged allergic reaction to the vaccine, as noted by her continued allergic symptoms days after her initial anaphylaxis. This may have contributed to the development of cardiovascular collapse in this case.

64 SIX NATIONS UNDER AGE RUGBY AND BMI, WHAT PRICE SUCCESS?

On March 20th, 2022, the Irish Times featured the personal profiles of the successful Irish under 20 years team in the Six Nations contest against France, England, Scotland, Wales and Italy. The mean weight was 100 Kgs and 8/31 met the U.S. Center for Disease Control criteria for obesity with BMI >30 Kg/M2, (risk category 1,) theoretically putting them at risk for later life premature morbidity.

On reviewing the entire six nations forwards cohort, 61/94 had a BMI >30. No backs (n 89) had a BMI >30 and mean BMI was 26.4. All 33 prop forwards had a BMI >30 and 11 had a BMI >35, (CDC risk category 2.) 18/21 hookers had a BMI >30 whereas 2/20 locks had a BMI above 30 and 9/32 back row forwards had a BMI >30.

Much of this weight gain is facilitated by ‘bulking up’ with high caloric intake with supplemental protein under medical and nutritionist supervision. All of these players would be contracted professional or academy members.

For comparison the average BMI for the all age Kerry Gaelic football team in 2019 was 24.7. Rafal Nadal and Roger Federer have BMIs at 24.8. The Liverpool FC team (2022), average BMI is 23.1.

The BMI has known limitations in predicting excess body fat in individuals and Athletes but has proved useful in epidemiological studies and in the clinical management of morbid obesity.

The marked discrepancy between front row forwards and backs does suggest that the BMI difference cannot all be attributed to by higher muscle mass alone. With under age players one has to ask what are the downsides, if any ?, of rapid weight gain at this age. It certainly points to a need for long term follow up for all high performance athletes using supervised protein supplementation and whose playing time weights exceed the norm for their age and height.