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ORIGINAL ARTICLE

Foetal bradycardia: A retrospective study in 9 Spanish centres☆,☆☆

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KEYWORDS
Bradycardia;
Heart block;
Fetal therapy;
Steroids

Abstract
Objective: The aim of this study is to review the current management and outcomes of foetal bradycardia in 9 Spanish centres.
Methods: Retrospective multicentre study: analysis of all foetuses with bradycardia diagnosed between January 2008 and September 2010. Underlying mechanisms of foetal bradyarrhythmias were studied with echocardiography.
Results: A total of 37 cases were registered: 3 sinus bradycardia, 15 blocked atrial bigeminy, and 19 high grade atrioventricular blocks. Sinus bradycardia: 3 cases (100%) were associated with serious diseases. Blocked atrial bigeminy had an excellent outcome, except for one case with post-natal tachyarrhythmia. Of the atrioventricular blocks, 16% were related to congenital heart defects with isomerism, 63% related to the presence of maternal SSA/Ro antibodies, and 21% had unclear aetiology. Overall mortality was 20% (37%, if terminations of pregnancy are taken into account). Risk factors for mortality were congenital heart disease, hydrops and/or

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ventricular dysfunction. Management strategies differed among centres. Steroids were administrated in 73% of immune-mediated atrioventricular blocks, including the only immune-mediated IInd grade block. More than half (58%) of atrioventricular blocks had a pacemaker implanted in a follow-up of 18 months.
Conclusions: Sustained foetal bradycardia requires a comprehensive study in all cases, including those with sinus bradycardia. Blocked atrial bigeminy has a good prognosis, but tachyarrhythmias may develop. Heart block has significant mortality and morbidity rates, and its management is still highly controversial.

PALABRAS CLAVE
Bradicardia;
Bloqueo auriculoventricular;
Terapia foetal;
Corticoides

Bradicardia foetal: estudio multicéntrico retrospectivo en 9 hospitales españoles

Resumen
Objetivo: Revisar el manejo actual y la evolución de la bradicardia fetal en 9 centros españoles.
Método: Estudio multicéntrico retrospectivo: análisis de todos los fetos con bradicardia diagnosticados en 9 centros españoles entre enero de 2008 y septiembre de 2010. Los mecanismos electrofisiológicos responsables de la bradicardia fetal se estudiaron mediante ecocardiografía.
Resultados: Se registraron 37 casos: 3 fetos con bradicardia sinusal, 15 con extrasistolia auricular no conducida y 19 con bloqueo auriculoventricular (AV) de alto grado. Bradicardia sinusal: el 100% asoció patologías severas. Extrasistolia auricular no conducida: excelente pronóstico, pero un caso desarrolló posnatalmente taquicardia supraventricular. Entre los bloqueos AV de alto grado, el 16% asociaban cardiopatía congénita con isomerismo, el 63% anticuerpos antiRo/SSA maternos y el 21% fueron de etiología desconocida. La mortalidad global de los bloqueos AV fue del 20% (37% si consideramos la interrupción voluntaria del embarazo). Factores de riesgo fueron: asociar una cardiopatía congénita, hídrops y/o disfunción ventricular. El tratamiento fue variable según el centro, se administraron corticoides en el 73% de los bloqueos de grado III inmunomediados y en el único caso de bloqueo de grado II inmunomediado. En un seguimiento medio de 18 meses, se implantaron marcapasos en el 58% de los bloqueo AV de alto grado.
Conclusiones: La bradicardia fetal sostenida precisa siempre de un estudio exhaustivo, incluso en el caso de la bradicardia sinusal. La extrasistolia auricular no conducida tiene buen pronóstico pero puede asociar taquicardia. El bloqueo AV de alto grado fetal tiene todavía una morbimortalidad significativa y su tratamiento es controvertido.

Introduction

Transient episodes of bradycardia are a common occurrence in the developing foetus and they are usually benign.[1] However, sustained foetal bradycardia unrelated to labour is a rare but potentially serious entity. The condition may be due to sinus bradycardia (SB), blocked premature atrial contractions (BPACs), or high degree atrioventricular block (AVB).[2] The latest pathology is very important due to its high mortality (17–43%)[3–5] and morbidity,[3] and to the international controversy surrounding its management.[6] In most cases, in the absence of congenital heart disease (CHD), conduction defects are due to the transplacental passage of maternal antibodies that attack the conductive tissue with subsequent fibrosis,[7] while in a smaller number of foetuses no cause is found for the block (non-immune or ''idiopathic'' AVB). In immune-mediated cases, the involved antibodies (anti-Ro/SSA) are necessary but not sufficient to cause disease,[8] which affects only 1–2% of children of mothers with these antibodies (the risk is tenfold if the mother has a previous child with the condition).[4] In addition to damaging the conductive tissue antibodies can cause diffuse cardiac damage in the form of endocardial fibroelastosis and dilated cardiomyopathy,[9] which result in considerably worse outcomes. Different treatment approaches have been used in immune-mediated AVB, including fluorinated steroids, beta-agonists, and plasmapheresis, aiming to prevent myocardial inflammation, increase the foetal heart rate, and reverse heart failure,[10–13] but their efficacy is controversial.[6,14,15] The lack of consensus on the best strategy to manage these patients accounts for the heterogeneous, even divergent therapeutic approaches of different hospitals.[15] This study describes the diagnosis and clinical approach to the management of foetal bradycardia in 9 Spanish hospitals, and aims for starting point for a future prospective multicentre study with a broader, nationwide scope.