New Oral Anticoagulants In Complex Adult Congenital Heart Disease Patients: A Single Centre Experience

B. Sarubbi, G. Scognamiglio, A. Merola, A. Correra, M. D’Alto, E. Romeo, D. Colonna, G. Di Nardo, M.G. Russo

Adult Congenital Heart Disease UNIT (GUCH Unit). Monaldi Hospital, Naples, Italy

Abstract

Introduction & Background: New oral anticoagulants (NOA), including apixaban, dabigatran and rivaroxaban have been developed as alternatives to warfarin, the standard oral anticoagulation therapy for patients with atrial fibrillation. Adult congenital heart disease (ACHD) patients are usually at high risk for thromboembolism due to the presence of atrial arrhythmias, impaired ventricular function and pro-thrombotic conditions. Usually in this complex patients, warfarin use is limited by medication adherence, necessity of laboratory monitoring, pro-hemorrhagic conditions.

Aim of the study: was to assess the efficacy and safety of NOA in ACHD patients at high risk for thromboembolism.

Methods: 22 ACHD (5 male - 22.7%) patients affected by complex lesions had been randomized treated with NOA. Mean age at inclusion was 50±14.6 years. 1 patients had Ebstein anomaly, 5 had an atrial septal defect (1 uncorrected), 4 tetralogy of Fallot after surgical correction, 2 pulmonary stenosis (1 uncorrected), 4 tricuspid atresia (3 with Fontan circulation, 1 with Glenn palliation), 1 double inlet left ventricle with a Fontan circulation, 2 ventricular septal defect (1 uncorrected with Eisenmenger syndrome), 1 double outlet right ventricle with a Fontan circulation, 1 transposition of the great arteries after Mustard procedure, 1 uncorrected partial atrio-ventricular septal defect. 17 (77.3%) patients were taking vitamin K antagonists at the time of the inclusion, 3 (13.6%) antiplatelet drugs and 2 (9.1%) were native. Only 1 patient had pulmonary arterial hypertension (Eisenmenger syndrome). 12 (54.5%) patients had permanent atrial fibrillation, 6 (27.3%) had history of persistent atrial fibrillation, 1 (4.5%) of parossistic atrial fibrillation and 3 (13.6%) of intra-atrial reentry tachycardia. 15 (68.2%) patients had heart failure (9 with right heart failure).

Results: Mean follow-up was 8.5±4 months. 10 (45.4%) patients were treated with Apixaban 5 mg bid, 2 (9.2%) with Apixaban 2.5 bid (low calculated glomerular filtration), 10 (45.4.7%) started Dabigatran 150 mg bid.

One patients with Fontan circulation had to discontinue the treatment with NOAC (namely Apixaban 2.5 mg bid) for major gastrointestinal bleeding and severe anemization. She had a previous history of minor bleeding (epistaxis, menorrhagia) and impaired renal function (creatinine 0.123 mmol/l). She was the only one with previous history of bleeding.

There were no thromboembolic events during the follow-up. 3 patients with atrio-pulmonary Fontan circulation and previous history of atrial thrombus remained thrombus free during the follow-up.

Conclusions: The NOA can be safety used even in a high risk population such as complex ACHD patients, as they showed to be efficacious in the prevention of stroke and systemic embolism with a slow risk for major bleeding. They appear to have a favourable safety profile in a long-term use population with a great number of extracardiac possible diseases, making them promising alternatives to warfarin.