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AORTIC ARCH INTERRUPTION WITHOUT DUCTUS ARTERIOSUS AND NO VENTRICULAR SEPTAL DEFECT

To the Editor:

We read the interesting case report by Cazavet and associates¹ on “an infant with a previously unrecorded type of aortic arch interruption.” However, similar types of aortic arch interruption without ductus arteriosus and without ventricular septal defect, although rare, have been previously described since 1964.²⁻⁶ The common features of these unusual patients were the longevity up to adolescence with no severe symptoms and the presence of extensive collateral circulation supplying the descending aorta.²⁻⁶ Surgical treatment was also reported in relation to the various anatomic patterns.²⁻⁶ In the field of pediatric cardiology, sometimes some old scientific papers can represent important sources of knowledge.

The interesting new aspect described by Cazavet and associates¹ is the association with del 22q11 and the relative embryogenetic implications that expand the spectrum of cardiovascular defects of this syndrome.

Carolina Putotto, MD
Marta Unolt, MD
Dario Marino, MD
Department of Pediatrics
“La Sapienza” University
Rome, Italy

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RECOGNITION AND MANAGEMENT OF HEPARIN-INDUCED THROMBOCYTOPENIA IN PATIENTS AFTER CARDIAC SURGERY

To the Editor:

In a recent publication, Demma, Paciullo, and Levy¹ evaluated the recognition and treatment of heparin-induced thrombocytopenia (HIT) in patients after cardiothoracic surgery using the direct thrombin inhibitor (DTI) argatroban. We agree that the detection and management of HIT in patients who have had cardiac surgery is of clinical importance, and we have

used a similar method of HIT identification and treatment with the DTI bivalirudin.

Our familiarity with the use of bivalirudin was heightened after our participation in the CHOOSE-ON trial published in 2007, which evaluated the use of this DTI during cardiopulmonary bypass in patients having cardiac surgery.² Since then, we have standardized our method of identifying HIT patients postoperatively and treating them with bivalirudin. To detect HIT, we use a method similar to that of Demma, Paciullo, and Levy¹ in which patients who score in the moderate range on the 4-Ts schema³ are subjected to a platelet factor 4/heparin immunoassay. Occasionally, we will also confirm the diagnosis with a serotonin release assay. To then distinguish HIT from hemodilution or other causes of thrombocytopenia, we also categorize the patterns of platelet counts as outlined by Poupard and colleagues.⁴

Our standardization of detection methods did not alter the number of patients who received a diagnosis of HIT (14.5% compared with 15.1% before 2007). However, we did see an increased rate in the use of bivalirudin to treat thrombocytopenia (23.1% compared with 17.5%) (Table 1). This suggests that our standardization procedures may have resulted in earlier identification and medical management of HIT.

TABLE 1. Characteristics of patients treated with bivalirudin after cardiac surgery

	Before (2004-2006)	After (2007-2010)
Thrombocytopenia	15.1% 463/3065	14.5% 476/3288
DTI use in patients with thrombocytopenia	17.5% 81/463	23.1% 110/476
HITS (per decreased platelets)	4.3% 20/463	5.5% 26/476
HITS (per total adult patients)	0.7% 20/3065	0.8% 26/3288
HITS mortality	60.0% 12/20	23.0% 6/26

Thrombocytopenia is defined as a 50% decrease in platelets from preoperative baseline by 24 hours after cardiopulmonary bypass. Mortality is defined as death within index admission. DTI, Direct thrombin inhibitor; HITS, heparin-induced thrombocytopenia and thrombosis syndrome.