

INFLAMMATORY PROFILE IN CHD PEDIATRIC PATIENTS

RS Leal^a, MO Cunha^b, SF Oliveira^b,
MMP Luciano^b, ACS Castro^c, IPC Tavares^c,
RMM Imorib Gonçalves MS^d, JPM Neto^{b,c,e}

^a Pós-graduação em Farmácia, Universidade Federal da Bahia (PPGFAR/UFBA), Salvador, BA, Brazil

^b Pós-graduação em Ciências Farmacêuticas, Universidade Federal do Amazonas (PPGCF/UFAM), Manaus, AM, Brazil

^c Pós-graduação em Imunologia Básica e Aplicada, Universidade Federal do Amazonas (PPGIBA/UFAM), Manaus, AM, Brazil

^d Instituto Oswaldo Cruz Salvador (CPqGM), Salvador, BA, Brazil

^e Pós-graduação em Ciências Aplicadas à Hematologia, Universidade Estadual do Amazonas (PPGH-UEA/HEMOAM), Manaus, AM, Brazil

Background and aims: Congenital heart defects (CHD) are a group of alterations that have an impact on the cardiocirculatory structure and function, which can lead to death in the first years of life or remain asymptomatic until adulthood. The CHD development is associated with multifactorial causes such as: genetic, epigenetic, immunological, environmental, nutritional deficiency and prenatal care. Our aim was to investigate the profile of inflammatory cytokines (IL6, IL10, IL18, and TNF- α) in pediatric patients with congenital heart disease followed at the Hospital Universitário Francisca Mendes, Manaus, Amazonas. **Materials and methods:** The epidemiological design was of the cross-sectional type, with samples collected in the period between 08/2022 to 02/2023 at the Hospital Universitário Francisca Mendes – HUFM. Participated in the study all patients aged < 1-year-old, diagnosed and followed up at the HUFM, after signing the free and informed consent form (ICF) from the mother of the minor's legal guardian. **Results:** A collection of 110 children aged between 1 week and 6 months was carried out. All those who underwent cardiac surgery had elevated serum complications of cytokines IL6, IL8, IL10, and TNF- α before and after surgery, when compared to patients who did not require surgery. **Discussion:** The inflammatory syndrome seen in CC is usually described as innate factors, which can determine the magnitude of its systemic inflammatory state and the individual inflammatory response to stressors. Faced with the lack of data in the literature regarding the prevalence of these CCs both in the state of Amazonas and in the city of Manaus, studies that seek to contribute with epidemiological data in this population are extremely necessary, since the late diagnosis of CC contributes to the high risk of morbidity and mortality from carrier patients. **Conclusion:** According to our preliminary data, we believe that the continuation of this work will contribute with relevant information regarding cardiovascular diseases, bringing direct benefits to both the patient and the medical staff. In addition, the characterization of genetic alterations and the profile of inflammatory cytokines in patients with CHD can contribute to the discovery of new risk factors, better understanding of the genesis of cardiovascular problems and who knows how to elucidate the presence or

absence of certain genetic variants with its biochemical, hematological and inflammatory impact

<https://doi.org/10.1016/j.htct.2023.09.1595>

6-NITRODOPAMINE AND 6-CYANODOPAMINE ARE RELEASED BY HUMAN WASHED PLATELETS

FC Mathias-Netto^a, R Campos^a, CES Nash^a,
MO Moraes^b, MEA Moraes^b, G Nucci^{a,c}

^a Universidade Estadual de Campinas (Unicamp), Campinas, SP, Brazil

^b Universidade Federal do Ceará (UFC), Fortaleza, CE, Brazil

^c Universidade de São Paulo (USP), São Paulo, SP, Brazil

Background and aims: 6-nitrodopamine is a novel catecholamine that is released from vascular tissues, such as human umbilical cord vessels, aortic rings from *Chelonoidis carbonarius*, *Pantherophis guttatus* and from *Callithrix* spp. 6-nitrodopamine (6-ND) has been previously identified as an endogenous substance with dopamine antagonistic properties. This study aims to identify its role in platelet aggregation. **Methods:** Liquid chromatography couple to mass spectrometry was used to quantify catecholamines from washed platelet samples. Optical aggregometry was used to identify the role of 6-ND, 6-cyanodopamine, dopamine, known receptor subtype selective dopaminergic agonists and antagonists have in ADP, collagen and thrombin stimulated aggregation to identify any inhibitory or potentiator role of 6-ND. Aggregations were carried out in human plasma rich platelets (PRP) and washed platelets. All subjects gave written informed consent, and the Universidade Estadual de Campinas (Unicamp) ethics committee approved the clinical protocol (Approval Number 00812918.0.0000.5404). **Results:** Platelets released 6-nitrodopamine and 6-cyanodopamine upon stimulation by thrombin (0.1 U; n = 15), collagen (1 μ g/m; n = 15), U-46619 (3 μ M; n = 13), ADP (30 μ M; n = 13), dopamine (10 μ M; n = 12), adrenaline (10 μ M; n = 12). 6-nitroadrenaline, 6-bromodopamine and 6-nitrodopa were not detected (n = 12 for each group). 6-ND does not cause or inhibit platelet aggregation and does not affect ADP, collagen or epinephrine induced PRP aggregation or thrombin-induced washed platelet aggregation. Curiously, 6-ND potentiates the aggregation induced by U-46619. 10 μ M dopamine is sufficient to potentiate ADP and collagen induced aggregation at subthreshold concentrations. 6-ND reduces dopamine potentiation of ADP and collagen induced aggregation. Haloperidol and the D3 receptor antagonists PG01037 and SB277011A reduce the potentiation induced by dopamine but not basal ADP and collagen potentiation. Sumanriole, a selective D2 agonist, potentiated aggregation which was blocked by 6-ND. **Conclusion:** The data demonstrates that platelets released 6-nitrodopamine and a new catecholamine, 6-cyanodopamine. 6-ND acts as a D2/D3 receptor antagonist. Given that both dopamine and 6-ND are released by the vascular endothelium, 6-ND modulates platelet reactivity and may play a