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CLINICAL RESEARCH

Left atrial volume is not an index of left ventricular diastolic dysfunction in patients with sickle cell anaemia



Le volume atrial gauche ne constitue pas un paramètre de dysfonction diastolique chez les patients atteints de drépanocytose homozygote

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Abbreviations: A, Late peak diastolic velocity of the mitral inflow; DD, Diastolic dysfunction; E, Early peak diastolic velocity of the mitral inflow; e', Early diastolic tissue velocity at the septal mitral annulus level; LA, Left atrial/atrium; LAVi, Left atrial volume index; LV, Left ventricle/ventricular; LVMi, Left ventricular mass index; LVEF, Left ventricular ejection fraction; ROC, Receiver operating characteristic; SCA, Sickle cell anaemia.

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MOTS CLÉS

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Summary

Background. — Left ventricular diastolic dysfunction (LVDD) is common in sickle cell anaemia (SCA). Left atrial (LA) size is widely used as an index of LVDD; however, LA enlargement in SCA might also be due to chronic volume overload.

Aim. — To investigate whether LA size can be used to diagnose LVDD in SCA.

Methods. — One hundred and twenty-seven adults with stable SCA underwent echocardiographic assessment. LA volume was measured by the area-length method and indexed to body surface area (LAVi). Left ventricular (LV) filling pressures were assessed using the ratio of early peak diastolic velocities of mitral inflow and septal annular mitral plane (E/e'). Using mitral inflow profile and E/e', LV diastolic function was classified as normal or abnormal. LAVi > 28 mL/m² was used as the threshold to define LA enlargement.

Results. — The mean age was 28.6 ± 8.5 years; there were 83 women. Mean LAVi was 48.3 ± 11.1 mL/m² and 124 (98%) patients had LA dilatation. In multivariable analysis, age, haemoglobin concentration and LV end-diastolic volume index were independent determinants of LAVi ($R^2 = 0.51$; $P < 0.0001$). E/e' was not linked to LAVi ($P = 0.43$). Twenty patients had LVDD; when compared with patients without LVDD, they had a similar LAVi (52.2 ± 14.7 and 47.5 ± 10.2 mL/m², respectively; $P = 0.29$). Receiver operating characteristics curve analysis showed that LAVi could not be used to diagnose LVDD (area under curve = 0.58; $P = 0.36$).

Conclusion. — LA enlargement is common in SCA but appears not to be linked to LVDD. LAVi in this population is related to age, haemoglobin concentration and LV morphology.

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Résumé

Contexte. — Chez les patients drépanocytaires homozygotes (DH), la dysfonction diastolique (DD) ventriculaire gauche (VG) est fréquente. La taille de l'oreillette gauche (OG) est couramment utilisée pour le diagnostic de DDVG, cependant, chez les DH la dilatation OG peut également être liée à la surcharge volumique secondaire à l'anémie.

Objectif. — Évaluer le volume OG indexé (VOGi) comme indice de DDVG chez les DH.

Méthodes. — Cent vingt-sept DH en état stable (28,6 ± 8,5 années, 83 femmes) ont bénéficié d'une échocardiographie. Le VOGi a été mesuré par la méthode de surface-longueur. Les pressions de remplissages VG ont été évaluées par le ratio des pics des vélocités proto-diastoliques du flux transmитral et de la portion septale de l'anneau mitral (E/e'). La fonction diastolique VG a été catégorisée comme normale ou anormale en utilisant le profil transmитral et E/e'. Un VOGi > 28 mL/m² définissait une OG dilatée.

Résultats. — Le VOGi moyen était de 48,3 ± 11,1 mL/m² ; 124 (98%) patients avaient une dilatation de l'OG. En analyse multivariée, l'âge, le taux d'hémoglobine et le volume VG téldiastolique indexé étaient les déterminants du VOGi ($R^2 = 0,51$; $p < 0,0001$) ; E/e' n'était pas corrélé au VOGi ($p = 0,43$). Le VOGi des patients avec DDVG ($n = 20$) était comparable à celui des patients sans DDVG (respectivement, 52,2 ± 14,7 et 47,5 ± 10,2 mL/m² ; $p = 0,29$). Le VOGi n'avait pas de valeur pour le diagnostic de DDVG (aire sous la courbe = 0,58 ; $p = 0,36$).

Conclusion. — La dilatation OG observée chez les DH ne semble pas être un indice diagnostique de DDVG. Dans cette population, le VOGi est lié à l'âge, au taux d'hémoglobine et à la morphologie VG.

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Background

Sickle cell disease is one of the most common inherited blood disorders worldwide [1]. Besides chronic anaemia, many pathophysiological processes contribute to the complexity of the disease, including haemolysis and repeated vaso-occlusive events, with ischaemia-reperfusion injury leading to endothelial cell dysfunction [1,2].

In patients with homozygous sickle cell disease – also called sickle cell anaemia (SCA) – cardiac remodelling

includes left heart chamber enlargement due to volume overload induced by anaemia [3]. In addition to morphological remodelling, left ventricular (LV) functional impairment is common in these patients. Two recent studies in invasive right heart catheterization have shown that post-capillary pulmonary hypertension is the most frequent cause of pulmonary hypertension in SCA [4,5]. LV diastolic dysfunction (LVDD) diagnosed by echocardiography is common and is an independent risk factor for mortality [6,7]. In addition, concomitant to the improvement in life expectancy

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