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Pulmonary Arterial Hypertension: Epidemiological, Clinical Aspects and Prognoses in the Cardiology Department and Internal Medicine of CHU Brazzaville

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Abstract: Pulmonary Arterial Hypertension PAH is a medical condition serious and severe. In Congo, its epidemiology and its etiologies are poorly understood. This study, aimed at improving the care of patients with PAH, was interested to epidemio-clinical aspects, and prognosis of this entity. This was a retrospective, cross-sectional study carried out from the 1st January 2021 to December 31, 2022 (2 years) in Brazzaville University Hospital. Were included 148 patients, the diagnostic criteria having permit to retain a PAH were compliant to that of the PAPUCO study (PAH if PAPS ≥ 35 mmHg, severe PAH if PAPS ≥ 60 mmHg). The heart failure syndrome was present in 140 cases (94.6%), it was right exclusively in 36 cases (24.3%). The etiologies of the group, they were present in 97 cases (66%) followed group III in 24 cases (16.2%). PAH was said to be severe in 39 cases (26.3%). The evolution has been towards complications in 29 cases (19.6%), lethality in 17 cases (11.5%). Poor prognosis factors were the existence of underlying heart disease underlying (AOR =5.6; p =0.02), male sex (AOR=4.8; p=0.02); hyperkalemia (AOR= 9.4; p=0.00). High blood pressure pulmonary is an increasingly common condition encountered in clinical practice in our context.

Keywords: PAH, Left Heart Disease, Brazzaville, Congo

1. Introduction

PAH is a serious and severe condition. Its prevalence in France is estimated at 1.5 cases/100,000 inhabitants; this prevalence is undoubtedly underestimated [1, 2].

The prevalence of primary pulmonary arterial hypertension is well known; it is estimated at 1 or 2 cases per million inhabitants. In sub-Saharan Africa, few studies on this disease have been published; the PAPUCO multicenter study made it possible to list the etiologies of PAH [3].

The recommendations 2022 propose new hemodynamic definitions, as well as a clinical classification of pulmonary hypertension (PH). A mean pulmonary artery pressure (mPAP) threshold of 20mmHg now defines PH, and a pulmonary vascular resistance (PVR) threshold of 2WU integrates the definition of precapillary PH, with the aim of early diagnosis of incipient PH. Pre-capillary PH is defined by normal PAPO ≤ 15 mmHg and elevated PVRs > 2WU, while post-capillary PH is defined by elevated PAPO > 15 mmHg. The latter may be isolated, with normal PVRs ≤ 2WU, or associated with precapillary involvement ("combined PH") in case of elevated PVRs > 2 WU. Exercise-induced PH, which was not defined in previous

guidelines, has been reinstated in the new guidelines and is defined by a PAPm/cardiac output [4, 5].

In Cameroon, in 2011, the frequency of 25.3% of patients with PAH was noted in a cardiology center. In Abidjan in a pulmonology department there was a frequency of 0.36% including chronic respiratory problems [6, 7].

In the Democratic Republic of Congo, a hospital study in the South Kivu region reported a frequency of 3.7% [8, 9].

In Congo, its epidemiology and etiologies are poorly understood. This study, aimed at improving the management of patients with PAH, focused on the épidemio-clinical and prognostic aspects of this entity.

2. Materials and Methods

This was a retrospective, cross-sectional study carried out from January 1, 2021 to December 31, 2022 (2 years) at the Brazzaville University Hospital in the Department of Cardiology and Internal Medicine.

The study included patients hospitalized during the study period. The sampling was exhaustive. Were included 148 patients aged over 18 years meeting the inclusion criteria.

Data collection was carried out using a pre-established survey form containing epidemiological, clinical, paraclinical and evolutionary data contained in the medical files.

Hospitalization time was defined as the time interval from the onset of the first symptoms to admission to the cardiology department. The diagnostic criteria for pulmonary arterial hypertension are ultrasound, they were established based on the PAPUCO study.

Pulmonary arterial hypertension was considered based on a PAPS ≥ 35mmg on transthoracic ultrasound, obtained from IT flow and POD. It was said to be severe when the PAPS was ≥ 60mmHg.

The etiologies of PAH were divided into five groups, in accordance with the Nice 2018 pulmonary hypertension classification.

The data were entered and processed by Microsoft EXCEL 2017 and Epi info 3.5.2 software. The calculation of the Odds ratio with their 95% confidence interval was carried out to search for poor prognostic factors.

3. Definitions and Classification of PAH

a) Hemodynamic definition of pulmonary arterial hypertension [9]

1. Precapillary pulmonary hypertension:

It reflects the presence of pulmonary vascular disease associating a mPAP > 20 mmHg, a PCP ≤ 15 mmHg and a pulmonary vascular resistance (PVR) [ratio (mPAP-PCP)/cardiac output] ≥ 3 Wood units (mmHg/l/ min).

2. Isolated postcapillary pulmonary hypertension:

It is defined by a PAPm > 20 mmHg, a PCP > 15 mmHg and an RVP < 3 Wood units.

3. Combined pre- and postcapillary pulmonary hypertension associates a mPAP > 20 mmHg, a PCP > 15 mmHg and a PVR ≥ 3 Wood units.

b) Ultrasound definition [10]:

Pulmonary hypertension is defined as an elevation in pulmonary systolic blood pressure greater than 35mmHg in the absence of pulmonary stenosis. The PAPS is estimated by Doppler ultrasound from the maximum speed of the regurgitation flow of the tricuspid insufficiency, the right atrial pressure, according to the Bernoulli equation below: PAPS= 4VmaxIT2 +POD.

c) Classification [9-12]:

The classification of pulmonary arterial hypertension (PAH) according to the 2018 Nice consensus (Table 1).

Table 1. *Classification of pulmonary arterial hypertension.*

1. Pulmonary arterial hypertension
1.1. Idiopathic
1.2. Hereditary
1.3. Induced by drugs or toxicants
1.4. Associated with:
1.5.1. Connectivity
1.5.2. HIV infection
1.5.3. Portal hypertension
1.5.4. Congenital heart diseases
1.5.5. Bilharzia
1.5. Long-term responders to calcium antagonists
1.6. Pulmonary veno-occlusive disease and/or pulmonary capillary hemangiomatosis
1.7. Persistent HTP of the newborn
2. PH in left heart disease
2.1. Heart failure with preserved LVEF
2.2. Heart failure with reduced LVEF
2.3. Valvulopathy
2.4. Congenital obstructive postcapillary lesions
3. PH from respiratory diseases and/or chronic hypoxemia
3.1. COPD
3.2. Interstitial pneumonias
3.3. Other restrictive and/or obstructive respiratory diseases
3.4. Hypoxia without lung disease
3.5. Developmental anomalies
4. PH linked to pulmonary artery obstruction
4.1. Chronic thromboembolic PH
4.2. Other pulmonary artery obstructions
5. HTP of multifactorial or uncertain mechanism
5.1. Hematological diseases
5.2. Systemic diseases
5.3. Complex congenital heart diseases

4. Results

During the study period, 2036 patients were hospitalized, 148 of whom had pulmonary arterial hypertension (7.3%).

We noted a female predominance of 62.8% with a sex ratio (M/F) of 0.6. The average age was 59.2 years with extremes ranging from 18 to 97 years.

The average hospitalization time was 43.5 days; dyspnea was present in 98% of cases, of which 85.5% were in functional stage III-IV followed by edema in 96% of cases then cough in 90% of cases.

The heart failure syndrome was present in 140 cases (94.6%), it was said to be global in 96 cases (64.8%), left in 8 cases (5.4%), right exclusive in 36 cases (24, 3%).

Group II etiologies were present in 97 cases (66%) followed by group III in 24 cases (16.2%) then group I, IV, V

which respectively represented 19 cases (12.8%), 7 cases (4.7%) and 1 case.

Ultrasonographically, the average PAPS was 52.7 mm Hg with a median of 50 mm Hg, pulmonary arterial hypertension was said to be severe in 39 cases (26.3%). LVEF was reduced in 77 cases (52%).

The progression was towards complications in 29 cases (19.6%), lethality in 17 cases (11.5%). Poor prognostic factors were the existence of underlying heart disease (AOR =5.6; p =0.02), male gender (AOR =4.8; p =0.02); hyperkalemia (AOR=9.4; p=0.00).

Table 2. *Patients repartition r according to functional signs.*

	N	%
Dyspnea	145	98
NYHA		
Stage 4	71	49
Stage 3	53	36,5
Stage 2	20	13,8
Stage 1	1	0,7
Edema	96	64,9
Cough	90	60,8
Hepatalgia	77	52
Chest pain	14	9,4
Palpitations	10	6,8
Hemoptysis	7	4,7
Asthenia	5	3,3
Others*	2	1,3

Others*: vertiges (n=1), syncope (n=1)

Table 3. *Etiologies of pulmonary arterial hypertension.*

	N	%
Group I	19	12.8
HIV	9	47.4
Idiopathic PAH	3	
Congenital heart disease (CIA)	4	
Others*		
Group II	97	65.5
Dilated cardiomyopathy	66	68
Left valvular disease	22	22.7
Hypertensive heart disease	5	
Ischemic heart disease	4	
Group III	24	16.2
CPC sequelae of tuberculosis	13 5	4.2
CPC post COPD	4	
Others**		
Group IV (Pulmonary embolism)	7	4.7
Group V (sickle cell anemia)	1	0.7

Others*: Liver cirrhosis (n=1), hepatitis B (n=1), hepatitis C (n=1)
Others**: Post-embolic CPC (n=3), Pulmonary fibrosis (n=4)

Table 4. *Echocardiographic parameters.*

	Means ± extreme	standard deviations
Left ventricle (mm)	27.7±7.6	14 and 55
Right ventricle (mm)	54.2±14.1	4.7 and 87.9
VD/VG	0.6±0.4	0.1 and 5.2
Left atrium (cm2)	25.1±10.7	5.50 and 78
Right atrium (cm2)	24.4 ±8	9.2 and 52.4
PAPS (mmHg)	52.7±13.3	35.2 and 106
LVEF (%)	50.8±19.4	20 and 90

Table 5. *Echocardiographic abnormalities.*

	N	%
Dilation of DO	122	82.4
Dilation of the LA	103	69.6
Dilated IVC	92	62.1
Kinetics disorder (Segmental and/or global)	80	54

	N	%
LV dilatation	77	52
EF (%)		
Lowered	61	41.2
Intermediate	16	10.8
Valvulopathy	45	30.4
RV dilatation	40	27
PAPS (mmHg) ≥ 60	39	26.3
Spontaneous contrast	13	8.8
Pericardial effusion	9	6.1
Others*	5	3.3

Others*: Calcification of the pericarde (n=1); défect du septum inter auriculaire (n=4).

Table 6. *Multivariate analysis of factors linked to deaths.*

	Death		AOR [IC-95%]	P-value
	yes (n=17)	No (131)		
Sex				
Male	10	45	4,8 (1,3-20,7)	0,02
Underlying heart disease				
yes	10	39	5,6 (1,5-26,2)	0,02
K+				
Hyperkaliémia	7	25	9,4 (2,3-47,2)	0,001

5. Discussion

Several limitations and biases punctuated our study; the first concerns the sample analyzed which obviously reduces the power of the observations, secondly the survey was carried out in a single medical training (CHUB) not allowing any extrapolation to the rest of the country, a selection bias was possible given the exclusive recruitment of medical files which was limited to the Cardiology department, the retrospective nature of the study not having allowed the taking into account of ultrasound parameters not evaluated and certain paraclinical examinations (EFR, lung scintigraphy, CT pulmonary angiography and sophisticated laboratory tests, etc.) for etiological guidance. Diagnoses of PAH were based on specialist opinion confirmed by cardiac ultrasound without confirmation by a right heart catheter. As discussed, the ETT only allows an estimation of the PAPS.

Despite these limitations, we obtained results that deserve to be discussed with data from the literature.

5.1. Epidemiological Data

In our study, PAH had a hospital frequency of 7.3%. Our result is close to that of Choudhary G et al in 2013 in the USA [13] who reported a hospital frequency of 6.8%, and of Enea I et al in 2010 in Italy [14] who reported a hospital frequency of 6. 8%, 6% on the other hand it differs from that of Ellenga Mbolla FB et al in 2017 in the Republic of Congo who reported a prevalence of 1.1% [15]. This difference in frequency is explained by the short period, the sample size of our study, but also by the fact that our study took into account all the etiological groups of pulmonary arterial hypertension.

The age distribution in our study shows that the average age was 59.2±17.5 years with a median of 59 years, extremes ranging from 18 to 97 years and the most represented age group was that from 70 to 79 years old with 37% of cases. Our results are close to those reported by the American registry (Reveal Registry) in 2010 [16], Jingi AM et al in 2017 in Cameroon [6], Ellenga Mbolla FB et al in 2017 in the Republic of Congo [15], which reports respectively an average age of 53 years, 63 years, 60.9 years.

The female predominance reported in our study is found in the literature and in various studies such as those of Ellenga Mbolla FB et al in 2016 in Congo BZV [13]; Choudhary G et al in 2013 in the USA [13]. This female predominance remains poorly understood based on literature data; certain hypotheses put forward the role of sex hormones (estrogens) and autoimmunity in predisposition to the disease. It is also reported that in sub-Saharan Africa women consult much more than men.

5.2. Clinical Data

The time between the appearance of the first symptoms and admission to the cardiology department varies greatly depending on the series. As a general rule it is long, this is the case in our study (43.5± 61 days); similarly Ellenga Mbolla FB et al in 2017 in Congo B, American register (Reveal Register) in 2010 [15], Humbert M et al in 2006 in France [1], report a long delay. This delay is justified by the non-specific and slowly evolving clinical symptoms and the use of traditional treatments. The majority consult at an advanced stage of the disease.

The clinical picture of PAH boils down to a syndrome of right heart failure or manifestations linked to the underlying disease. In our series, dyspnea was the majority symptom, it was present in 98% of cases. This observation is superimposable to that of the series Friedrich T et al (PAPUCO registry) in 2016 [2], Ellenga Mbolla FB et al in 2017 in Congo BZV [15], Pessinaba et al in Togo had placed their frequencies respectively at 100% and 98% from cases of CPC and pulmonary embolism.

Functional class III-IV of the NYHA was the most represented in 85.5% of cases, our results are superimposable to those of Dzudie A et al 2018 in Cameroon [18] and Ngunga M et al in 2020 in Kenya [19], who also report a predominance of this functional class. Edema constituted the second most frequent symptom in 64.9% of cases, this result is similar to that of Friedrich T et al (PAPUCO registry) in 2016 who reported 64% of cases. The significant presence of edematous syndrome is the clinical translation of the severity of the cardiac involvement and the late diagnosis of the disease. Global heart failure syndrome was present in 64.9% of cases, followed by exclusive right heart failure syndrome in 24.3% of cases.

5.3. Ultrasound Data

The mean PAPS was 52.7 mmHg with a median of 50mmHg, extremes ranging from 32.5 to 106 mmHg. These results are close to those of Strange et al in Australia [18], who found an average PAPS of 56 mmHg; the PAPUCO Cohort study reported a mean PAPS of 61.4 mmHg, Ngunga et al in Kenya [19], found a median PAPS of 56 mmHg. The severity of PAH was observed in 39 patients (26.3%) and was not linked to a risk of mortality, which differs from several studies [19, 20].

Seventy-seven patients (52%) had reduced LVEF. This is consistent with the significant proportion of left heart diseases in our study, this finding was also observed in the PAPUCO cohort study and by Dzudie A et al 2018 in Cameroon who reported a similar result of 50.5%.

5.4. Etiological Data of Pulmonary Arterial Hypertension

The predominance of group II etiologies in our study is similar to that of the Armadale and PAPUCO echocardiography cohort, which reported 67.9% and 69%, respectively [17, 22]. In a large Italian echocardiography cohort on PAH 52.6% had left heart disease; 7.5% pulmonary disease, 1.3% a thromboembolic cause while 10.5% had an unknown etiology of PAH [17].

Group II PAH is the result of an increase in pulmonary capillary pressure due to diastolic and/or systolic LV dysfunction, and/or severe left valvular disease. Thus, cardiovascular risk factors (hypertension, diabetes, obesity) and left-sided valve disease responsible for increased left atrial pressure were not associated with PAH.

The main etiologies of group II encountered in our study were dilated cardiomyopathy (DCM) in 68% of cases, followed by mitro-aortic valve disease in 22.7% and then hypertensive and ischemic heart disease in 9.3% of cases. CMD constitutes one of the main causes of heart failure (HF) with a hospital frequency in sub-Saharan Africa varying between 17 and 48% [21]. In the Republic of Congo, Kimbally Kaky et al in 2015 reported a hospital frequency of 32.1% and PAH was observed in 33 cases; CMD was the second cause of HF after arterial hypertension [20]. Pulmonary hypertension of group III constituted the second most frequent group in our study, the after-effects of tuberculosis represented the main cause of this group. Our results differ from literature data which incriminate COPD as the main chronic respiratory condition responsible for PAH [23]. In sub-Saharan Africa, tuberculosis remains a real public health problem. In the Republic of Congo, Bemba et al in 2016 reported that the management of tuberculosis in our context was done late, which causes parenchymal and/or bronchial damage responsible for pulmonary vascular remodeling of small arteries and arterioles with the consequence of increased pulmonary vascular resistance, hence pre-capillary PAH.

In our study, group I pulmonary hypertension accounted for 19 (12.8%) of cases; in high-income countries, most PAH studies focus on group I pulmonary hypertension. [1, 16], which differs from our study which explores all etiological groups of pulmonary arterial hypertension.

5.5. Poor Prognostic Factors

In our study, the risk factors identified were: the existence of an underlying heart disease (AOR =5.6; p =0.02), male gender (AOR =4.8; p =0.02); hyperkalemia (AOR=9.4; p=0.00).

The presence of pulmonary arterial hypertension is a predictive factor of death in patients with myocardiopathy according to Cappola et al who explain having demonstrated a significant correlation between mean pulmonary arterial pressure and myocarditis [24]. Observation already made by the WHO which reports that HF is one of the frequent causes of death in patients suffering from pulmonary arterial hypertension.

The identification of prognostic factors at the time of diagnosis has been the subject of numerous studies with sometimes discordant results depending on the methodology [24].

6. Conclusion

Pulmonary arterial hypertension is a condition increasingly encountered in clinical practice in our context. Dyspnea was found in most patients as well as a picture of heart failure.

Group II is the main cause of pulmonary hypertension dominated by CMD. Male gender, underlying heart disease, hyperkalemia were poor prognosis factors.

Targeted action to combat cardiovascular risk factors and regular monitoring of chronic pulmonary pathologies as well as systematic HIV screening is necessary.

Author Contributions

Solange Flore Mongo Ngamami: redactor
Ruddy Junior Taty: redactor
Kivie Mou-moue Ngolo Letomo*: redactor and translater
Fikahem Ellenga Mbolla: reviewer

Conflicts of Interest

The authors declare no conflicts of interest.

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