

Asian Journal of Cardiology Research

Volume 6, Issue 1, Page 300-304, 2023; Article no.AJCR.102008

Bidirectional Relationship between Severe Resistant Hypertension and Extensive Psoriasis at Young Man: A Case Study

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/102008

Case Study

Received: 20/04/2023 Accepted: 29/06/2023 Published: 29/07/2023

ABSTRACT

Introduction: Hypertension is a common manifestation in patients with psoriasis. The bidirectional relationship between psoriasis and hypertension, by inflammatory mechanism and by the effect of democratic therapy, is proven in several studies. Our clinical case study illustrates this relationship and its adequate management.

Case Presentation: We report the case of a 42-year-old man suivi for unbalanced diabetes (HbA1c: 8.5%) and psoriasis treated with corticosteroids for the last 11 years. Presented to our cardiology department for severe hypertension and extensive psoriasis lesions. On examination, he was clinically dyspneic NYHA-II, severe hypertension at 170/112 mmHg, tachycardia up to 105 beats per minute. His skin examination noted the presence of well- defined, symmetrical,

Asian J. Cardiol. Res., vol. 6, no. 1, pp. 300-304, 2023

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erythematous patches covered with silvery scales affecting almost the entire body surface, including face and scalp. The echocardiography showed important cardiac hypertrophy with a normal contractility, his left ventricle ejection fraction estimated at 58%. In view of his young age, we lanced a secondary hypertension testicular objective the presence of secondary hyperaldosteronism, the cortisol, metanephrine and normetanephrine were all in normal levels. On imaging, the Doppler ultrasound of the renal arteries showed no renal arteries stenosis, CT scan of the adrenal glands demonstrate bilateral adrenal hypertrophy without detectable mass. The evolution was marked by the control of his hypertension after the psoriasis relapse, with a blood pressure of 129/65 mmHg under quadritherapy based on calcium channel blocker 10mg, ACEI 10mg, indapamide 5mg and carvedilol 6.25mg twice daily, aldactone 50mg/d.

Discussion: The relationship between psoriasis and hypertension is bidirectional, hence the need for comprehensive and adequate management, especially in terms of the use of dermocorticoids in psoriasis, which can unbalance hypertension control. There is evidence that the severity of psoriasis, as determined objectively by body surface area, has a significant impact on the control of hypertension. especially those with more extensive skin involvement.

Conclusion: There is evidence that the severity of psoriasis, as determined objectively by body surface area, has a significant impact on the control of hypertension. especially those with more extensive skin involvement.

Keywords: Resistant hypertension; psoriasis; erythematous patches.

1. INTRODUCTION

Hypertension is a common manifestation in patients with psoriasis. The bidirectional relationship between psoriasis and hypertension, by inflammatory mechanism and by the effect of democratic therapy, is proven in several studies. Our clinical case study illustrates this relationship and its adequate management.

2. CASE PRESENTATION

We report the case of a 42-year-old diabetic patient (HBA1c: 8.5%). He has been treated with

corticosteroids for psoriasis for the last 11 years under topical corticosteroids, admitted to our hospital to assess and control his resistant hypertension.

On admission, the patient was presented with severe hypertension levels and extensive psoriasis lesions, clinically dyspneic NYHA-II, High→high blood pressure (BP) was 170/112 mmHg, tachycardia up to 105 bpm. His skin examination noted the presence of well-defined, symmetrical, erythematous patches covered with silvery scales affecting almost the entire body surface, including his face and scalp (Fig. 1).



Fig. 1. Typical appearance of a psoriasis flare-up, with symmetrical erythematous patches covered with silvery scales, affecting almost the entire body surface, including his face and scalp reflecting the sever form

A: A close up of small chronic extensive plaques with thick scaling on the lower extremities. Note the scattered areas of post-inflammatory hypopigmentation resulting from previously treated plaques Band; B, E: Close-up, Thick and red patches with sharply defined edges and adherent silvery scales covering the entire upper arms indicate severe psoriasis; C: Extensive thin plaques with scales on the dorsal face of his tronc; D: Thin plaques with bran-like scales on his face, affecting his quality of life

After his clinical examination, we performed a cardiac assessment started with electrocardiogram (ECG) showed a regular sinus rhythm, fixed PR interval at 160ms, narrow QRS complex, aspect of electrical left ventricular hypertrophy with secondary repolarisation disorder (Fig. 2).

The transthoracic echocardiography (TTE) showed important cardiac hypertrophy with a normal global and segmental contractility, LVEF estimated a 52% with biplane Simpson

(Fig. 3). In view of his young age, a secondary hypertension tests was performed, which biologically objectifies the presence of secondary hyperaldosteronism, the cortisol, metanephrine and normetanephrine were all in normal levels after complying with the preparation protocol for each hormone (Table 1). On imaging, the doppler ultrasound of the renal arteries showed no renal artery stenosis, CT scan of the adrenal glands demonstrated bilateral adrenal hypertrophy without detectable mass.



Fig. 2. Showed a regular sinus rhythm at 88 bpm, fixed PR interval at 160ms, fine QRS complex, aspect of electrical left ventricular hypertrophy with secondary repolarisation disorder



Fig. 3. Echocardiography showed the aspect of hypertensive heart disease with normal contractility

A: Strict PLAX section showing symmetric and significant left ventricular wall hypertrophy; posterior wall and septum at 14mm; B: Symmetrical and significant hypertrophy of the left ventricular walls is seen in the strict PSAX; C: T mode shows left ventricular hypertrophy; D: In the Simpson biplane, the left ventricular ejection fraction is 52%.

	Test	value	normal value
Complete Blood Count	Leukocytes	17600/mm3	4500 à 11000/ mm³
(CBC)	Neutrophils	13600/mm3	1800 à 7800/ mm³
	Lymphocytes	14600/mm3	1000 à 4800/ mm³
	Hemoglobin	13g/dL.	14-16,5 g /dL
	Platelets	341000/mm3	150000 à 400000 / mm³
	Prothrombin rate	88%	70 - 100%
	TCA :	33s	26 – 39 s
Urinary Hydro-electrolytic	Natrurie	25mmol/L	40–220 mmol/L
	Kaliurie	17 mmol/L	25,0–125,0 mmol/L
	Uree:	8.42 mmol/24h	250 - 580 mmol /24 h
	Creatinine	857 mmol/24h	7,1–17,7 mmol/24 h
	24h Proteinuria	0.52 g/l	0.5-1.3 g/24h
Blood Hydro-electrolytic	Natremia	141mEq/L	135-145 mEq/L
	kalemia	3.7mEq/L	3,5 - 4,5 mEq/L
	Urée:	0,42g/L.	0,15 - 0,45 g/L
	Creatinine:	24.6mg/L.=) MDRD:	7 - 13 mg/L
		36mL/min	
	Albumine	39g/L	0.5-1.3 g/24h
	CRP:	136mg/L	< 6 mg / L
	procalcitonin	0.10 µg/l	< 0,05 µg/l.
	Erythrocyte	98 mm	< 20 mm
	sedimentation rate		
	TOUL	0.75	
		0.75 mUl/l	0,5 - 5.0 mUl/l
Hormonal tests	Cortisolemia 8H	20.3 µg/ai	5-23 µg/di
	Aldosterone	57 pg/mi	97-626 pg/ml
	Renin	203 mui/l	3.3-41.8 mui/l
	Aldosterone-Renin	= 2.1	<3
	Ratio (ARR)	00 / I	0.0500 / 1
	ivietanephrine	9∠ pg/mi	0 - 2500 pg/mL
	Normetanephrine	69 pg/ml	0 - 3000 pg/mL
Urine test	Culture	Sterile	

Table 1. Biological assessment

The evolution was marked by the control of his hypertension under quadritherapy based on 10 mg of amlodipine, 10mg of perindopril, 5mg of indapamide, carvedilol 6.25mg twice daily and aldactone 50mg/d. And after the regression of the relapse of his psoriasis with an appropriate protocol in collaboration with the dermatology department based on methotrexate and several sessions of phototherapy. The monitoring is based on self-measurement at home and on 24h blood pressure holter was carried out on admission, during course of treatment and at the end of treatment, during which the average of his blood pressure was found to be well controlled at 118/60 with low variability. We believe that the control of the blood pressure is done after the regression of his skin lesions, confirming the relationship between hypertension and psoriasis.

3. DISCUSSION

The relationship between psoriasis and hypertension is bidirectional, hence the need for comprehensive and adequate management,

especially in terms of the use of dermocorticoids in psoriasis, which can unbalance hypertension control [1]. Psoriasis patients have an increased risk of hypertension, via pathogenic immune cells (Th17 and CD T cells) in psoriasis, and cytokines related to the TNF/IL23/IL-17 pathway have been shown to participate in the development of hypertension [2]. The risk of psoriasis incidence was increased in hypertensive patients, a history of hypertension in subjects aged < 65 years and the use of thiazides were associated with an increased risk of psoriasis incidence [3]. There is evidence that the severity of psoriasis, as determined objectively by body surface area, has significant impact on the control а of hypertension. especially those with more extensive skin involvement [4].

In view of this clinical situation, various studies suggest that the moderate-to-severe psoriasis population has a high prevalence of undiagnosed and untreated cardiovascular risk factors, emphasising the importance of screening patients with psoriasis for cardiovascular risk factors [5,6]. Therapeutically; psoriasis patients with hypertension were 5 times more likely to be on antihypertensive monotherapy, 9.5 times more likely to be on dual antihypertensive therapy, 16.5 times more likely to be on triple antihypertensive therapy, and 19.9 times more likely to be on quadruple therapy or centrally acting agents [7].

4. CONCLUSIONS

The mechanism of psoriasis on the control of hypertension is poorly understood, particularly during flares. There is a need for multicentre studies that can explain the common pathophysiology between these two conditions and recommend comprehensive and appropriate management.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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Peer-review history: The peer review history for this paper can be accessed here: https://www.sdiarticle5.com/review-history/102008