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BRIEF REPORT

Impact of Biologic Therapy on Key Cardiovascular Risk Parameters in a Psoriatic Cohort—a Retrospective Review

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ABSTRACT

Background: Psoriasis is a risk factor for cardiovascular disease. Biologic agents have revolutionised psoriatic skin control. This study aims to assess the change in cardiovascular risk factors in a cohort of patients treated with 1 year of continuous biologic treatment.

Methods: A retrospective medical record review was conducted of consecutive patients receiving biologic therapy for chronic plaque psoriasis in a single dermatology centre at a major tertiary hospital in Australia. The effect of biologic therapy on psoriasis was assessed using a psoriasis area severity index (PASI). Cardiovascular risk factors included systolic blood pressure (SBP), diastolic BP (DBP), heart rate (HR) and body mass index (BMI). Measurements at baseline and 1-year follow-up were compared using paired *t*-tests.

Results: A total of 106 patients were reviewed with a median age of 44 years, and 63% of the patients were male. At baseline, mean BMI was 30 (SD 7), mean SBP was 129 (SD 17), mean DBP was 81 (SD 9) and mean HR was 82 (SD 14). Over 12 months, the PASI was reduced from 17.4 (SD 8.5) to 1.4 (SD 1.7, $p < 0.001$) indicating skin improvement. There was no significant difference from baseline in SBP (difference 2.3 mmHg, 95% CI – 1.4–5.9), DBP (0.6 mmHg, 95% CI – 1.2–2.5), BMI (difference – 0.1 kg/m^2, 95% CI – 0.9–0.7) or HR (difference 1.3, 95% CI – 3.9–6.4).

Conclusion: In patients with psoriasis, markers of cardiovascular disease risk did not improve after 1 year of biologic therapy despite significant improvements in psoriasis skin severity.

Keywords: Cardiovascular risk factors; Biologic therapy; Psoriasis; Cardiovascular disease; Retrospective review

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Key Summary Points

Why carry out this study?

Psoriasis is a major cardiovascular risk factor.

Biologics dramatically improve the skin manifestations of psoriasis but their effect on other cardiovascular risk factors is unclear

Very brief background leading to the study, including for example disease population, economic burden and/or unmet need.

Patients with psoriasis have increased cardiovascular morbidity leading to economic burden. It is unknown whether biologics help improve cardiovascular risk factors to reduce this morbidity

What did the study ask?

Do biologics improve cardiovascular risk parameters?

What was learned from the study?

Biologics alone are not enough to reduce cardiovascular risk parameters

What were the study outcomes/conclusions?

In addition to biologics, which clear the skin, there needs to be a holistic approach to cardiovascular risk management in psoriasis patients incorporating weight loss and blood pressure management

INTRODUCTION

Psoriasis is a chronic systemic inflammatory disease occurring in 2–4% of the population and has a significant impact on patient quality of life. The systemic inflammation inherent in severe psoriasis is recognised as an independent and significant risk factor for cardiovascular disease (CVD) [1]. Studies report that the presence of psoriasis increases 10-year cardiovascular risk scores by an estimated 6.2%, which brings this to a rate similar to that of people with previous coronary artery disease or type 2 diabetes [2, 3]. This increased risk of developing CVD has concordantly been associated with decreased life expectancy, and CVD related death remains the leading cause of death in people with psoriasis [1, 4–6]. Aggressive cardiovascular risk factor management, including blood pressure, lipids, obesity and smoking control, is effective in primary prevention of CVD [7] and likely best achieved in a multidisciplinary care setting in the psoriatic cohort [8].

There is emerging evidence suggesting biologic therapy for moderate to severe psoriasis decreases the risk of subclinical coronary artery disease progression and non-calcified plaque burden independent of traditional cardiovascular risk factors. The purported mechanism for this relates to overlapping inflammatory and immunologic pathways in both coronary artery disease and psoriasis [9–11]. The observational evidence supporting this potential relationship is mostly limited to first-generation biologics including TNF-a inhibitors, with emerging data for newer generation biologics such as IL-12/23 inhibitors and IL-17A inhibitors [12]. Additionally, there is some evidence that canakinumab, an IL-1B inhibitor, used in rheumatological disorders, reduces the rate of non-fatal myocardial infarctions and cardiovascular death [13]. This retrospective study aims to determine whether, in a cohort of patients with psoriasis, biologic therapy is associated with positive effects on traditional cardiovascular risk factors such as blood pressure, heart rate and body mass index.

METHODS

A retrospective review was conducted of all patients prescribed biologic therapy for whole-body chronic plaque psoriasis (CPP) in the Department of Dermatology at Westmead Hospital between January 2012 and July 2021. Patients were excluded if they did not have data for the cardiovascular risk parameters of interest or did not receive continuous biologic therapy for 1 year. This study was approved by the

Ethics Committee at Westmead Hospital, NSW, Australia, reference number 2105-07, and was performed in accordance with the Helsinki Declaration of 1964 and its later amendments.

Data were collected using written and electronic records of patients eligible for biologic therapy as per Australian Medicare criteria, which include a psoriasis area severity index (PASI) > 15 and having failed two treatments, including phototherapy and oral therapies (acitretin, methotrexate, ciclosporin, apremilast), or having incurred toxicity to these treatments or contraindications existing to their use. Data extraction was performed at two timepoints: commencement of therapy (baseline) and 1-year post therapy. Data included demographic information (gender and age), biologic agent, previous biologic treatment, systolic blood pressure (SBP in mmHg), diastolic blood pressure (DBP in mmHg), weight (kg), height (cm) and PASI. Body mass index (BMI) was calculated from weight and height. Interruption to therapy and use of medications known to potentially effect parameters of interest were collected, including antihypertensives, diabetic medications, lipid-lowering medications and psychotropic medications. Patients' characteristics and biologic agent were described using mean and standard deviation (SD) for continuous variables and frequency (proportion) for categorical variables. Continuous measurements collected at baseline and 1 year were summarised with their mean and changes between the two periods assessed. The change in cardiovascular risk factors after 1 year with biologic use was evaluated by calculating the mean difference between pairs of measurements with the associated 95% confidence interval (CI) and *p*-value based on a paired *t*-test. The analysis was based on complete records at both baseline and 1 year; pattern of missing data at baseline was provided. Subgroup analysis was performed to determine whether there were differences in cardiovascular risk factors based on gender (male versus female), BMI (≤ 25 versus > 25) and history of biologic therapy [biologic naïve versus biologic experienced (BE)]. All statistical analyses were performed using R version 4.0.3 (R Core Team, Vienna, Austria). Two-sided *p*-values < 0.05 were considered significant.

RESULTS

A total of 147 CPP patients were treated with continuous biologic therapy for 12 months; 40 patients had incomplete data sets at baseline and 12 months and were excluded, leaving 106 in the analysis.

Patients were predominantly male (63%, $n = 67$), mean age was 44 (SD 16) years, and mean BMI on biologic therapy commencement was 30 (SD 7). Ustekinumab (IL-12/23 inhibitor, 35.8%), ixekizumab (IL-17 inhibitor, 21.7%) and secukinumab (IL-17 inhibitor, 19.8%) were the most frequently used biologic agents (Table 1). Missing data were analysed and data completeness was generally high with the exception of heart rate. The missing data rate for heart rate was 38% ($n = 40$), whereas for all other parameters, the missing data rate was < 6% (Fig. 1). At baseline, 17 (16%) patients were on antihypertensives, 11 were on antidiabetic agents, and 16 were on both an antihypertensive and antidiabetic medication.

For patients included in the analysis, it was established that medications with the potential to impact CV parameters of interest were not commenced during the 12-month period of biologic therapy assessed based on documentation within the patients' electronic medical record. Biologic-naïve (BN) patients were initiated on biologic agents with an average PASI of 20.6, which was reduced by 19.2 (95% CI: 17.9–20.5), leading to a final average PASI of 1.4 at 12 months (Table 2), compared to biologic-experienced (BE) patients, who at the time of transitioning biologic agent had an average PASI of 7.8 and improved their PASI by 6.2 (95% CI: 2.9–9.6), leading to a final average PASI of 1.6 at 12 months.

In our cohort, BE patients (98 kg) were heavier than BN patients (86 kg) at baseline, $p = 0.021$ (Table 1). The two groups had similar BMIs. No statistically significant change was found between baseline and 12 months in cardiovascular parameters of the psoriasis patients assessed (Table 2). There was no significant

Table 1 Baseline characteristics overall and stratified by biologic naïve versus biologic experienced

Variable	All (N = 106)	Biologic Naïve (N = 80)	Biologic experienced (N = 26)	p-value
Age (years)				
Mean (SD)	44 (16)	43 (16)	49 (15)	0.102
Median (IQR)	45 (30–56)	44 (30–56)	49 (36–59)	
Missing	0	0	0	
Gender				
Female	39 (36.8%)	33 (41.3%)	6 (23.1%)	0.095
Male	67 (63.2%)	47 (58.8%)	20 (76.9%)	
Biologic agent name (mechanism)				
Adalimumab (TNFa inhibitor)	5 (4.7%)	5 (6.3%)	0 (0.0%)	0.245
Etanercept (TNFa inhibitor)	2 (1.9%)	2 (2.5%)	0 (0.0%)	
Guselkumab (IL-23 inhibitor)	5 (4.7%)	2 (2.5%)	3 (11.5%)	
I nfliximab (TNFa inhibitor)	9 (8.5%)	7 (8.8%)	2 (7.7%)	
Ixekizumab (IL-17 inhibitor)	23 (21.7%)	18 (22.5%)	5 (19.2%)	
Risankizumab (IL-23 inhibitor)	3 (2.8%)	1 (1.3%)	2 (7.7%)	
Secukinumab (IL-17 inhibitor)	21 (19.8%)	15 (18.8%)	6 (23.1%)	
Ustekinumab (IL-12/23 inhibitor)	38 (35.8%)	30 (37.5%)	8 (30.8%)	
Weight (kg)				
Mean (SD)	89 (23)	86 (20)	98 (30)	0.021
median (IQR)	87 (73–100)	84 (70–99)	89 (80–105)	
Missing	1	0	1	
Height (cm)				
Mean (SD)	170 (9)	170 (9)	172 (11)	0.187
median (IQR)	171 (163–176)	171 (163–175)	171 (167–181)	
Missing	0	0	0	
BMI				
Mean (SD)	30 (7)	30 (6)	31 (10)	0.318
Median (IQR)	30 (26–35)	30 (25–34)	31 (27–36)	
Missing	1	0	0	
BMI categories				
$\leq$ 25	23 (21.7%)	20 (25.0%)	3 (11.5%)	0.346

Table 1 continued

Variable	All (N = 106)	Biologic Naïve (N = 80)	Biologic experienced (N = 26)	p-value
25–30	30 (28.3%)	22 (27.5%)	8 (30.8%)	
> 30	53 (50.0%)	38 (47.5%)	15 (57.7%)	
Systolic blood pressure (mmHg)				
Mean (SD)	129 (17)	128 (18)	133 (13)	0.209
Median (IQR)	128 (116–142)	127 (114–142)	130 (123–143)	
Missing	6	5	1	
High systolic blood pressure (> 130 mmHg)				
	43 (43.0%)	31 (41.3%)	12 (48.0%)	
Diastolic blood pressure (mmHg)				
Mean (SD)	81 (9)	80 (9)	83 (8)	0.131
Median (IQR)	82 (75–85)	81 (74–85)	82 (79–87)	
Missing	6	5	1	
Diastolic blood pressure (> 80 mmHg)				
	56 (56.0%)	40 (53.3%)	16 (64.0%)	0.353
Blood pressure (SBP > 130 mmHg or DBP > 80 mmHg)				
	61 (61.0%)	43 (57.3%)	18 (72.0%)	0.193

SD standard deviation, *IQR* interquartile range

difference in SBP (mean difference 2.3, 95% CI – 1.4–5.9, p = 0.222), DBP (mean difference 0.6, 95% CI – 1.2–2.5, p = 0.492), HR (mean difference 1.3, p = 0.63) or BMI (mean difference – 0.1, 95% CI – 0.9–0.7, p = 0.827) or HR (mean difference 1.3, 95% CI – 3.9–6.4, p = 0.630). However, some differences were found in the subgroup analysis. BE patients at follow-up had lower SBP and DBP by 5.7 mmHg (p = 0.042, 95% CI: 0.2–11.2) and 3.6 mmHg (p = 0.023, 95% CI: 0.5–6.6), respectively. Similar findings were also found in the male patients treated with biologic agents, with an improvement in SBP and DBP of 5.5 mmHg (p = 0.011, 95% CI: 1.3–9.6) and 2.2 mmHg (p = 0.036, 95% CI: 0.1–4.2), respectively. Patients with a BMI < 25 gained 2.2 kg (p = 0.02, 95% CI: 0.4–4.1) of weight over a year while on biologic therapy.

DISCUSSION

Psoriasis is an underrecognised cardiovascular risk factor. In this cohort of biologically treated patients on treatment for 1 year of continuous therapy, psoriasis severity significantly improved but there was no improvement in the cardiovascular risk factors including blood pressure, heart rate or BMI.

Psoriasis is associated with an increased risk of CVD, including premature myocardial infarction and stroke, not reflected by traditional cardiovascular risk factors [3, 14]. While psoriasis management has been revolutionised by biologic therapies that have the capacity to achieve complete skin clearance in most cases, the next most pressing therapeutic target is addressing the systemic inflammatory sequelae

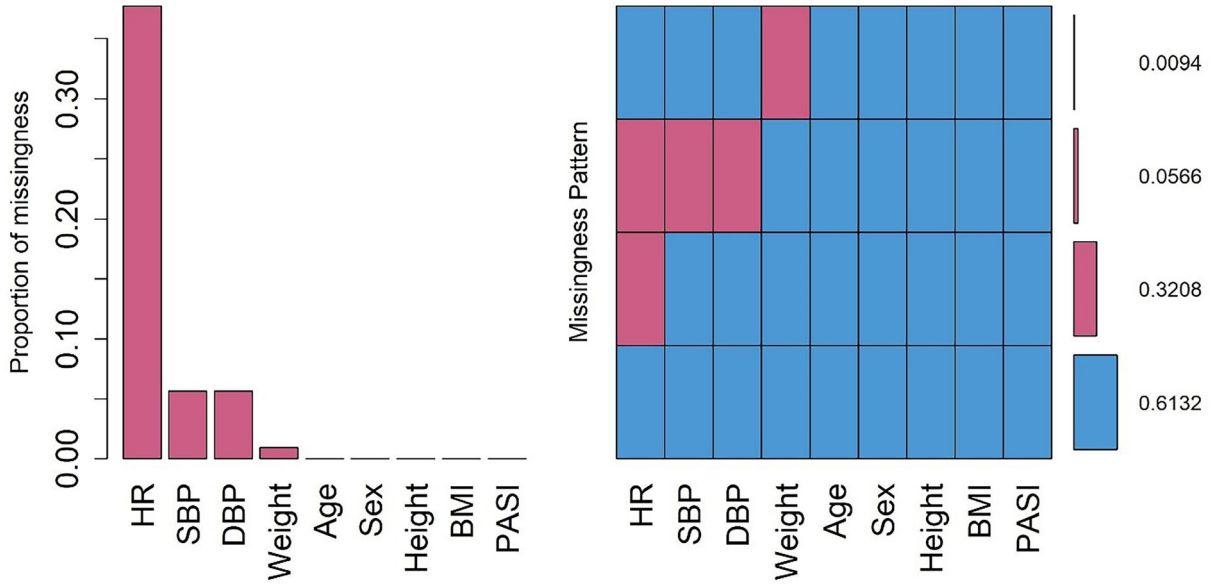

Fig. 1 Missing pattern of recorded parameters at baseline

of this disease. The systemic inflammation inherent in psoriatic disease, in part, explains the incumbent cardiovascular risk and premature CVD seen in patients with this disease [15, 16]. Psoriasis is thought to be linked with CVD through common immunological pathways involving pro-inflammatory and pro-atherogenic cytokines, including IL-1B, IL-17, IL-22, IL-23, IL-6 and TNF-a [17]. These may contribute to explaining the risk of accelerated atherogenesis and noncalcified coronary plaque burden observed in patients with psoriasis [9]. This has been evidenced through serial coronary computed tomography angiography (CCTA), where psoriasis patients had increased prevalence of high-risk plaque and greater noncalcified coronary plaque burden compared to controls [18]. While biologic therapy may improve the inflammatory phenotype of the coronary circulation [9], our retrospective data analysis revealed no impact on cardiovascular parameters after 1 year of therapy.

It is recognised that patients with psoriasis have a higher likelihood of obesity. The precise mechanism underlying this association is not clear. Endocrinological, genetic and immune-mediated processes all play a role in this association [25]. Behavioural factors also play a key role as the combination of a highly visible skin disease with psychological comorbidities significantly impacts motivation to partake in healthy lifestyle behaviours. Furthermore, obesity correlates with more severe psoriatic disease and may impact therapeutic response to biologics; hence, weight reduction in this cohort is a desired outcome [26]. The findings in our study show that the biologically treated psoriasis cohort were predominantly obese (BMI $>$ 30). It also demonstrated that regardless of significant improvement in skin psoriasis (with near or complete skin clearance achieved in most, reflected by reduction in PASI from 20.6 to 1.4 ($p < 0.001$), there was no significant improvement in obesity. Interestingly, patients with a BMI $\leq$ 25 experienced a 2.2 kg weight gain over a year. The lack of significant change in BMI overall may be a product of the length of observation and timepoints of capture; another possibility includes the notion of the indelible 'psychological scar' of psoriasis which may persist well beyond skin clearance and hinder a patient's capacity to positively engage in healthy lifestyle behaviours [27]. It is important to address weight loss in obese patients with psoriasis not only to reduce the development of obesity-associated comorbidities which may contribute to CV risk, but more so, it has the

Table 2 Cardiovascular parameters at baseline and 1 year in BE and BN patients on biologic therapy for severe psoriasis

Patients group	Outcome	*N*	Baseline (SD)	1 Year (SD)	Mean difference (95%C)	*p*-value
All						
	Heart rate (BPM)	66	82.3 (14.1)	82.0 (16.8)	1.3 (– 3.9, 6.4)	0.630
	Systolic blood pressure (mmHg)	100	129.5 (16.9)	127.4 (16.7)	2.3 (– 1.4, 5.9)	0.222
	Diastolic blood pressure (mmHg)	100	80.9 (8.6)	80.4 (9.8)	0.6 (– 1.2, 2.5)	0.492
	Weight (kg)	105	89.1 (23.4)	89.4 (23.9)	– 0.1 (– 1.5, 1.3)	0.895
	BMI	106	30.3 (7.1)	30.4 (7.2)	– 0.1 (– 0.9, 0.7)	0.827
	PASI score	106	17.4 (8.5)	1.4 (1.7)	16.0 (14.4, 17.7)	< **.001**
Biologic naive						
	Heart rate (BPM)	51	81.9 (14.2)	82.4 (18.4)	– 0.1 (– 6.1, 6.0)	0.986
	Systolic blood pressure (mmHg)	75	128.2 (17.8)	127.4 (17.8)	1.0 (– 3.5, 5.6)	0.651
	Diastolic blood pressure (mmHg)	75	80.2 (8.8)	80.7 (10.2)	– 0.4 (– 2.6, 1.8)	0.724
	Weight (kg)	80	86.2 (20.4)	86.6 (21.4)	– 0.2 (– 1.9, 1.5)	0.831
	BMI	80	29.9 (6.0)	29.7 (7.1)	0.2 (– 0.6, 1.0)	0.608
	PASI	80	20.6 (5.9)	1.4 (1.7)	19.2 (17.9, 20.5)	< **.001**
Biologic experienced						
	Heart rate (BPM)	15	83.7 (14.0)	80.3 (8.6)	6.2 (– 4.5, 16.9)	0.222
	Systolic blood pressure (mmHg)	25	133.2 (13.5)	127.4 (13.3)	5.7 (0.2, 11.2)	**0.042**
	Diastolic blood pressure (mmHg)	25	83.2 (7.7)	79.6 (8.8)	3.6 (0.5, 6.6)	**0.023**
	Weight (kg)	25	98.5 (29.6)	98.3 (29.4)	0.2 (– 2.1, 2.5)	0.863
	BMI	26	31.5 (9.7)	32.5 (7.1)	– 1.0 (– 3.3, 1.3)	0.393
	PASI	26	7.8 (8.2)	1.6 (1.9)	6.2 (2.9, 9.6)	< **.001**
Male						
	Heart rate (BPM)	39	82.8 (14.4)	82.4 (17.3)	4.0 (– 3.1, 11.2)	0.257
	Systolic blood pressure (mmHg)	61	134.1 (16.8)	129.3 (13.1)	5.5 (1.3, 9.6)	**0.011**
	Diastolic blood pressure (mmHg)	61	83.6 (8.3)	81.8 (8.5)	2.2 (0.1, 4.2)	**0.036**
	Weight (kg)	66	91.7 (23.6)	92.0 (24.4)	0.1 (– 2.0, 2.2)	0.927
	BMI	67	29.6 (7.1)	29.7 (7.2)	– 0.1 (– 1.3, 1.2)	0.918
	PASI score	67	17.7 (9.1)	1.7 (1.8)	15.9 (13.7, 18.2)	< **.001**

Table 2 continued

Patients group	Outcome	*N*	Baseline (SD)	1 Year (SD)	Mean difference (95%C)	*p*-value
Female						
	Heart rate (BPM)	27	81.6 (13.8)	81.5 (16.5)	− 1.8 (− 9.7, 6.1)	0.645
	Systolic blood pressure (mmHg)	39	122.3 (14.5)	124.7 (20.8)	− 2.4 (− 9.0, 4.1)	0.458
	Diastolic blood pressure (mmHg)	39	76.7 (7.3)	78.3 (11.3)	− 1.6 (− 5.0, 1.8)	0.338
	Weight (kg)	39	84.7 (22.5)	85.1 (22.8)	− 0.4 (− 1.9, 1.1)	0.574
	BMI	39	31.4 (7.0)	31.6 (7.1)	− 0.1 (− 0.7, 0.4)	0.633
	PASI score	39	17.0 (7.4)	0.8 (1.5)	16.2 (13.7, 18.7)	**< .001**
BMI ≤ 25						
	Heart rate (BPM)	14	82.9 (13.3)	91.5 (25.0)	− 6.3 (− 16.6, 4.0)	0.199
	Systolic blood pressure (mmHg)	21	125.2 (17.7)	124.7 (17.5)	1.9 (− 4.1, 8.0)	0.507
	Diastolic blood pressure (mmHg)	21	78.1 (9.6)	79.9 (12.6)	− 1.3 (− 6.7, 4.1)	0.614
	Weight (kg)	22	65.0 (6.0)	67.2 (6.0)	− 2.2 (− 4.1, − 0.4)	**0.020**
	BMI	23	21.5 (5.0)	22.5 (5.3)	− 1.0 (− 4.2, 2.3)	0.538
	PASI score	23	18.7 (7.7)	1.3 (1.9)	17.4 (14.0, 20.8)	**< .001**
BMI > 25						
	Heart rate (BPM)	52	82.2 (14.4)	79.5 (13.2)	3.2 (− 2.8, 9.3)	0.284
	Systolic blood pressure (mmHg)	79	130.6 (16.6)	128.1 (16.6)	2.3 (− 2.0, 6.7)	0.288
	Diastolic blood pressure (mmHg)	79	81.7 (8.2)	80.5 (9.1)	1.1 (− 0.8, 3.0)	0.248
	Weight (kg)	83	95.5 (22.1)	95.1 (23.5)	0.4 (− 1.3, 2.1)	0.602
	BMI	83	32.7 (5.5)	32.5 (6.0)	0.2 (− 0.4, 0.7)	0.570
	PASI score	83	17.1 (8.8)	1.5 (1.7)	15.7 (13.7, 17.6)	**< .001**

Bold indicates the significant *P* value

potential to positively impact psoriasis severity and response to treatment [25, 26].

Psoriasis has been associated with an increased risk of hypertension in observational studies and the magnitude of hypertension correlates with psoriasis severity [28]. Hypertension is attributable to 47% of ischaemic heart disease worldwide [29]. Meta-analysis data of 24 observational studies of psoriasis, capturing 2.7 million study participants, confirm the increased prevalence of hypertension in psoriasis patients with an odds ratio of 1.49 (95% CI 1.20–1.86) in severe psoriasis patients compared with controls [30]. In the current study cohort, blood pressure did not significantly change after 1 year of continuous biologic therapy. The patients in this study had elevated blood pressures with a mean systolic blood pressure of 130 mmHg and diastolic blood pressure of 91 mmHg at baseline. In subgroup analyses of

BE and male patients, both groups experienced an improvement in their systolic and diastolic blood pressure. Hypertension in psoriasis is thought to be in part triggered by the effects of sustained inflammation [30], which may lead to endothelial dysfunction and arterial stiffness [31–33]. Given this contributory inflammatory aetiology, it is plausible that biologic therapy may improve hypertension in psoriatic patients.

Heart rate was the final cardiovascular parameter assessed in this retrospective analysis. Some studies have demonstrated that heart rates increased by 10 beats per minute can increase the risk of cardiac death by 20% and that this parameter should be considered an independent cardiovascular risk factor [35]. There is limited evidence as to the effect of psoriasis on resting heart rate; however, one study has shown that heart rate recovery may be adversely affected in patients with psoriasis and therefore improvement in psoriasis outcomes may lead to improvements in heart rate [36]. Furthermore, Holter monitor results of a cohort of patients with chronic psoriasis and no prior cardiac conditions demonstrated heart rates were significantly higher both during the day and at night in psoriatic patients and revealed positive correlation between increased heart rate and severity of psoriatic disease, as expressed by PASI [38]. The patients in this analysis had a normal resting heart rate with a mean of 82 beats per minute; this did not change after 1 year of treatment with biologic therapy. Missing data may have impacted this result (Fig. 1). Furthermore, a relatively normal baseline heart rate in this cohort may have reduced the ability to detect change and the two timepoints assessed may not have been an accurate reflector of average heart rate.

Our study is limited by its observational, retrospective design with the inability to adjust for all potential confounders and the lack of a control cohort. We only extracted data that were routinely assessed and recorded in this psoriasis cohort, and this did not include lipid profile or glycaemic index. In addition, while confounders, such as concurrent use of relevant medications such as anti-hypertensive therapy, were obtained from hospital medical records, medications started outside of the hospital setting may not have been accurately recorded in all cases. The study is also limited by the exclusion of a small number of patients who did not have cardiovascular risk factors measured at baseline and 1-year follow-up. Further limitations include the relatively small sample size, including the subgroup analyses with small numbers within each group. Finally, it was unknown how many patients also suffered from psoriatic arthritis. Future studies may benefit from prospective design as well as further exploration of the mechanism underpinning lack of improvement in CV risk factor status despite psoriatic skin clearance, including a more detailed assessment of systemic inflammatory markers.

CONCLUSIONS

Biologic therapy is overwhelmingly effective for psoriatic skin disease, with newer agents having the capacity to achieve clear or near clear skin in most cases. Although there are emerging data to suggest that biologic therapy may exert independent effects on the coronary circulation [9–11], this study did not demonstrate significant improvement in key cardiovascular parameters after 1 year of continuous biologic therapy. A particular issue identified was the high prevalence of obesity within this cohort, which did not improve with biologic therapy, despite skin clearance. This study highlights the need for optimal cardiovascular preventative care and focused weight loss strategies in the psoriatic cohort, which can positively impact disease severity and response to biologic therapy [26, 39]. It is undeniable that biologic therapy can achieve excellent cutaneous control in psoriatic disease; further research however is needed to determine the impact of these therapies on systemic inflammation.

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Data Availability. The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of Interest. The authors have no conflict of interests for this article.

Ethical Approval. This study was approved by the Ethics Committee at Westmead Hospital, NSW, Australia, reference number 2105–07, and was performed in accordance with the Helsinki Declaration of 1964 and its later amendments.

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