Polycythaemia - Another Lifestyle Disorder

Surumi AR[a], Sasidharan PK[b]

a. Junior Resient Medicine, PVS Hospital Kozhikode;
b. Former HOD General Medicine and Division of Haematology, Govt Medical College, Kozhikode*

Published on 27th June 2025

ABSTRACT

Introduction: Polycythaemia is an increase in the absolute red blood cell (RBC) mass with increase in haemoglobin levels and haematocrit. It can be primary and secondary. Management depends on the causes and can be pharmacological and non-pharmacological. This original study has documented polycythaemia as yet another lifestyle disease along with hypertension, diabetes, dyslipidaemia fatty liver disease

Objectives of the study: To determine the clinical profile of patients with all types polycythaemia and to study the impact of weight reduction with diet and lifestyle changes on behaviour of polycythaemias.

Methods: We enrolled 92 consecutive subjects in a tertiary care centre with confirmed polycythaemia during a period of one year, subjected them to diet and lifestyle changes with an intention to achieve weight reduction and they were followed up for six months to one year. It was a hospital based prospective study between January 2020 to June 2021. Patients who were confirmed to have polycythaemia by symptoms, signs and laboratory features were included. They were classified into primary or secondary polycythaemia based on clinical judgement and laboratory parameters. Polycythaemia vera was diagnosed using WHO 2016 criteria.[1] The enrolled patients in both the groups were given clear advice on proper balanced diet with less carbohydrates and lifestyle modification aimed at weight reduction, if they were overweight, cessation of smoking and alcohol along with the standard care. They were then followed up by monthly reviews for re-counselling for lifestyle changes and weight recording at each visit with follow up for a minimum period of six months to maximum of one year. Only those who were compliant with the lifestyle changes and or achieved weight reduction were taken for the final evaluation in the study. Variables were compared between the two groups.

Results: The majority were males (88%), and the mean (SD) age was 46.5 (13.3) years. All subjects, irrespective of the causes of polycythaemia, had change in wellbeing, symptom relief, reduced frequency of venesections, haematocrit levels and other blood parameters (p value <0.05). There was statistically significant differences between the primary and the secondary polycythaemia groups, with respect to the clinical features, comorbidities, addictions and haematological profiles (p-value <0.05). The most striking observation of the study was that, there were 22 patients out of 92 (24%) with secondary polycythaemia due to overweight or obesity and they all had reversal of polycythaemia after weight reduction.

Conclusions: The study showed that intervention by diet and lifestyle modification leading to weight reduction, can reverse secondary polycythaemia due to weight gain and obesity. Besides that the same interventions, irrespective of the cause, gave statistically significant changes in haematological outcomes of all subjects with primary and secondary polycythaemia. The weight reduction needed to get benefit varied between patients but the mean weight reduction achieved was 9.13 Kg.[2]

Keywords: Polycythaemia, Reversal of polycythaemia, Lifestyle, Diet, Weight reduction

**See End Note for complete author details*

INTRODUCTION

Absolute polycythaemia or erythrocytosis is an increase in Hb in blood, due to overproduction of red cells and the resultant increased red cell numbers/red cell mass. Spurious polycythaemia reflects plasma volume contraction rather than increased red cell mass, which is obvious from clinical situations of severe dehydration or a decrease in plasma volume due to any cause.[1,3] Absolute polycythaemia may be primary with no physiological stimulus for erythrocytosis, the prototype of which is polycythaemia vera (PV), due to JAK2 (V617F) mutation.[4] Absolute polycythaemia may be secondary with a physiological stimulus like hypoxia for erythropoiesis or a pathological overproduction of erythropoietin/erythropoietin like substances. Differentiating

Cite this article as: Sasidharan PK, Surumi AR. Polycythaemia - Another Lifestyle Disorder. Kerala Medical Journal. 2025 Jun 27;18(2):47-53.

Corresponding Author:
Dr.Sasidharan PK, MD, FICP, FRCP
Former HOD, General Medicine and Division of Haematology, Govt Medical College, Kozhikode
Email: sasidharanpk@gmail.com

between primary and secondary causes is a challenge, but our experience is that it is easily achieved by good clinical evaluation and common laboratory tests, since erythropoietin levels are unreliable.[5,6] With hypoxia acting as a physiological stimulus, the commonest causes for polycythaemia are living at high altitude, smoker's polycythaemia, chronic lung disease like COPD and ILD, cyanotic heart disorders and obesity related. In response to the hypoxia, there is overproduction of erythropoietin from the renal interstitial peritubular capillary cells. But decreased perfusion of the renal interstitium due to local causes might also act as a hypoxic stimulus for erythropoietin release as in renal artery stenosis, polycystic kidney disease, hypernephroma, hydronephrosis, and sometimes even in nephrocalcinosis.[16] Pathological overproduction of erythropoietin like substances is seen rarely as in hepatoma, atrial myxoma, and cerebellar hemangioblastoma.

Irrespective of the cause, patients with polycythaemia presents with non-specific symptoms like fatigue, heaviness of head, headache, dizziness, transient blurring of vision or sometimes thrombotic events in venous or arterial circulation involving any part of the body. On examination, they may have ruddy complexion and cyanosis as a manifestation of polycythaemia. In addition to these, patients with PV may present also with erythromelalgia and symptoms due to hyperuricemia. Presence of splenomegaly, thrombocytosis or leucocytosis suggests PRV as the diagnosis.[14,16] But before diagnosing PV, it is mandatory to rule out secondary causes by good clinical evaluation, rather than doing JAK 2 mutation or serum erythropoietin upfront to make a diagnosis.[1,3,4,6-,9] It is mandatory to elicit history of weight gain in all patients, even if it is just two kilograms excess from the previous normal, to look for chronic hypoventilation as a possible cause, and history of chronic smoking to suggest smoker's polycythaemia.[14,16] Decreased chest expansion and clubbing of fingers could suggest interstitial lung disease as the cause. But clubbing could suggest congenital cyanotic heart diseases, or rarely chronic liver disease with hepatoma as a cause for polycythaemia. Hypertension, renal lumps and renal bruits could also be suggesting secondary causes for polycythaemia.[14,16]

Treatment of all polycythaemia may require phlebotomy, but it is the preferred method in PV where hydroxyurea, and low dose aspirin also may be needed. Ruxolitinib is giving promising results too in PV. The mainstay of treatment of secondary causes is to manage the underlying disease.[7,12,13] The effects of non-pharmacological measures like weight reduction and lifestyle modification in the management of primary and secondary polycythaemia is not studied well before. Literature search showed no studies that have ventured on lifestyle modification on polycythaemia, its clinical presentation, risk factors and management.

THE HYPOTHESIS FOR THE STUDY

Since weight gain is the commonest cause of decreased chest wall compliance and chronic hypoventilation, we had hypothesised that weight reduction would remove the commonest hypoxic stimulus and thereby removing that component and would cure polycythaemia in those with weight gain and obesity. This study was the outcome of our observation over the last two decades that unnoticed weight gain and the consequent chronic hypoventilation is the commonest cause of secondary polycythaemias.[5,7,14,16] We noted also that weight reduction by diet and lifestyle modification certainly had helped all types of polycythaemias and had resulted in reversal of polycythaemia in those with weight gain or obesity. This study was done to document our original observation and the hypothesis that weight reduction certainly helps in the management of all polycythaemias and in curing some of them. The study also aimed at evaluating the clinical and haematological profile, risk factor assessment and the effect of weight reduction, by diet and lifestyle modification in both primary and secondary polycythaemia. Weight reduction also improves several other comorbidities in those with polycythaemia, like hypertension, diabetes and non-alcoholic steatohepatitis (NASH). In those with overweight and obesity, the adipocyte size and mass expand and the white adipose tissue becomes hypoxic, leading to inflammation and cellular dysfunction. Thus it could be possible that weight reduction and lifestyle modifications would help to decrease the impact pro-inflammatory mediators and the improved outcomes.[9,10]

STUDY METHODS AND INTERVENTIONS[2]

It was a prospective observational study, from January 2020 to June 2021. The cut-off values for inclusion was Hb >15 g/dl and haematocrit >45 % in women and Hb >17g/dl and haematocrit >50 % in men. Patients with relative polycythaemias, those on erythropoietin, anabolic steroids, hydroxyurea or aspirin and those who would not be compliant with the lifestyle modification, were excluded. We enrolled 92 consecutive cases of polycythaemia who satisfied the inclusion

criteria. Informed written consent was obtained from all participants. Clinical and laboratory evaluations for secondary causes were done in all subjects. Patients were divided into primary or secondary polycythaemia based on clinical and laboratory parameters. Polycythaemia Vera (PV) was diagnosed using WHO 2016 diagnostic criteria.[2] Initially we panned to do JAK2 mutation only where categorization into primary and secondary was difficult, but we had got that in most patients, as it was already been done before the patients were referred to us. Erythropoietin estimation was not done as the study was conducted in a low-resource setting and it was our experience that it almost never helped us. Clinical presentation, dietary habits, history of any weight gain from the previous normal, causes of weight gain, history of tobacco smoking and alcohol intake, height, weight and BMI were recorded in all subjects. The lowest weight when the subjects were healthy and active was also recorded. BMI more than 23 was taken as overweight and above 28 as obesity. Frequency of venesection before and after the intervention was recorded. The baseline blood pressure was recorded in all at enrolment and during follow up. For modifying dietary practices, food questionnaire was used to collect details regarding each item, using a 24-hour dietary recall method on an average for a week's period. Details regarding the frequency of fruits, vegetables, protein containing items, of junk food, salt intake, salty food and fried food etc per day were separately recorded. Physical activity was assessed by noting the type, duration and frequency and the sedentary nature of work. Weight, blood pressure, Hb, haematocrit, liver function tests and blood sugar were checked at each visit.

Diet plan: All the enrolled patients were given advice on balanced diet with emphasis on calorie restriction, lifestyle modification for weight reduction and cessation of smoking and alcohol, along with standard care. A comprehensive diet plan with a pictorial representation of the proportions between different components was used for easy understandin.[9,10,11] Each meal containing all five components with adequate intake of protein (pulses, yogurt, fish, egg or meat), adequate vegetables (preferably raw or steamed and never over cooked), fresh seasonal fruits, one source of calorie in least possible amounts and adequate water intake were explained in detail.[9,10] The subjects were advised to consume only half the capacity of their stomach and leave the rest empty at each meal. Calorie rich foods were reduced and replaced that portion with fibre rich vegetables constituting the major proportion of any individual meal. They were instructed to restrict eating to a maximum of three times a day and to avoid all snacks. Water consumption was ensured to be adequate to produce sufficient urine output (2-2.5L/day). Previous consumption of junk food, fried foods, snacks, salted pickles and nuts, all fast foods and processed food items were taken note of and was advised to avoid.[9,10,11]

Physical activity: Any activity requiring a moderate amount of physical exertion was advised to be carried out for at least 30 minutes a day. Any level of exercise that could be incorporated into daily routine like avoiding elevators and the use of staircases, walking instead of using automobiles for short commutes, avoiding electrical appliances for household chores etc were advised.

Standard care: In those with PV were advised phlebotomy, aspirin and hydroxyurea as per individual clinical profile. In subjects with secondary polycythaemia, phlebotomy was advised in the initial visits only, when haematocrit was high (>50%), or when they were symptomatic, to reduce hyper viscosity.

All the study participants were reviewed every month for re-counselling and weight recording. The weight was monitored with the same electronic weighing machine for easy comparison, and adherence to diet and physical activity was re-emphasized at each visit. Appropriate modifications in lifestyle were advised accordingly. All study subjects were periodically assessed for a period of 6 months at least and final weight, smoking status, physical activity status, clinical status, and haematological parameters with focus on Hb, haematocrit, SGPT, and frequency of venesection were reviewed and recorded

Statistical methods: Data was entered in Excel and analysed using Stata 14.2. Variables were summarised appropriately. The percentage of individuals with different forms of polycythaemia and the presence of JAK2 mutation were summarized as frequency and proportions. Association between weight status, JAK2 mutation, smoking status, alcohol status, clinical symptoms, haematological profile and comorbidity status and the type of polycythaemia were done using Chi square test / Fischer's exact test. Dietary status (carbohydrate, protein, fast food intake etc.), and physical activity levels (mild, moderate, and severe) along with the different patterns of polycythaemia were also checked using the Chi-square test / Fischer's exact test. A P-value of < 0.05 was considered statistically significant.

Table 1. Key differences between biologic agents and Tofacitinib

Characteristics	Primary Polycythaemia n = 37 No. (%)	Secondary Polycythaemia n = 55 No. (%)	P value
Mean Age (SD)	59.4 (7.8)	37.8 (8.1)	<0.001
Female	7 (19)	3 (5)	0.08
Male	30 (81)	52 (95)	
JAK 2 Positive	24 (83)	0 (0)	<0.001
JAK 2 Negative	5 (17)	44 (100)	
Current Smokers	11 (30)	31 (56)	<0.001
Stopped Smoking	12 (32)	2 (4)	
Never Smoked	14 (38)	22 (40)	
Type 2 DM	18 (49)	20 (36)	0.24
Hypertension	21 (57)	19 (35)	0.04
COPD	0 (0)	2 (3.6)	0.51
OSAS	0 (0)	1 (1.8)	1.0
ILD	0 (0)	1 (1.8)	1.0

Table 2: Distribution of clinical presentation among the types of polycythaemia, n=92[2]

	Primary Polycythaemia n = 37 No. (%)	Secondary Polycythaemia n = 55 No. (%)	p value (chi square)
Asymptomatic	9 (24)	11 (20)	.62
Tiredness	11 (29.7)	24 (44)	.18
Arterial Thrombosis	5 (13.5)	1 (1.8)	.04
Venous Thrombosis	3 (8)	4 (7)	1.0
Headache	22 (60)	23 (42)	.10
Dizziness	14 (38)	12 (22)	.09
Erythromelalgia	3 (8)	0 (0)	.06
Aquagenic Pruritus	6 (16)	0 (0)	.003
Plethoric Face	9 (24)	7 (13)	.15
Eye Congestion	27 (73)	24 (44)	.006
Palmar Erythema	14 (38)	14 (25)	.21
Splenomegaly	32 (87)	0 (0)	<.001
Hepatomegaly	5 (13)	34 (62)	<.001

RESULTS

There were 37 cases of primary polycythaemia and 55 cases of secondary polycythaemia **(Table 2,3 and 4)**. The majority were males (88%) between 20 and 74 years with a mean age of 46.5 years. The most common symptoms were tiredness, fatigue, heaviness of head and or headache in both groups. Arterial thrombosis, tinnitus and blurring vision, cardiac symptoms, and aquagenic pruritus were found to be more common in primary polycythaemia group. Eye congestion, palmar erythema and plethoric face was seen in both, but splenomegaly was seen only in primary polycythaemia group. Hepatomegaly was significantly more common in secondary group. JAK 2 mutation was done in 73 cases, none of the secondary group had JAK 2 positivity, while 83% of the primary polycythaemia had JAK 2 positivity. The distribution of clinical presentation among the main types of polycythaemias are described in **table1.**

Table 3 shows the BMI pattern in different types of polycythaemia. Among the 22 patients with polycythaemia due to weight gain one patient had normal BMI but had a history of weight gain from his previous normal weight. On reducing that excess weight in that subject the haematocrit normalised without venesection.

Table 5 shows the subgroup with polycythaemia of overweight separately with the change in haemoglobin and haematocrit values post weight reduction. It is noteworthy that there was complete normalisation of haemoglobin and haematocrit levels after weight reduction in those with overweight related polycythaemia. They were managed initially with venesection and following intervention their mean haemoglobin, haematocrit and liver enzymes became normal over time. The difference was statistically significant with all p values <0.001. Out of 22 patients with polycythaemia of overweight / obesity, 13 were diabetic and 8 were having hypertension and 19 were having raised SGPT levels. Out of 11 patients with mixed cause for polycythaemia, 3 were diabetic, 4 were having hypertension and 7 were having raised SGPT levels. Hb, PCV, SGPT became normal in all of them and did not require any more venesection in the subgroup of polycythaemia due to weight gain. In addition there was a significant drop in all these parameters after weight reduction among PV and among polycythaemia due to mixed causes too. The frequency of venesections became much less in PV after weight reduction. In the smokers' polycythaemia group, Hb

Table 3. BMI and the categories of polycythaemia, n=92[2]

	Normal N (%)	Overweight N (%)	Obese N (%)
Polycythaemia Vera (n = 37)	21 (57)	12 (32)	4 (11)
Smokers' polycythaemia (n = 18)	16 (89)	2 (11.)	0 (0)
Polycythaemia of overweight (n = 22)	1 (4.6)	4 (18.2)	17 (77)
Polycythaemia of mixed Causes (n = 11)	0 (0)	1 (9)	10 (91)

Table 4. Pre and post-intervention, subgroup analysis of change in haematological parameters[2]

	Primary Polycythaemia n = 37 No. (%)	Secondary Polycythaemia n = 55 No. (%)	p value (chi square)
Polycythaemia Rubra Vera patients (n = 37)			
Hb (gm/dl)	20.5 (1.9)	19.8 (1.4)	0.003
PCV (%)	55.9 (4.1)	53.0 (2.9)	<0.001
SGPT (IU/ml)	42.1 (8.7)	40.0 (8.7)	0.02
Weight (kg)	57.44(6.7)	54.35 (5.4)	0.03
Smokers' polycythaemia managed without Venesection (n = 7)			
Hb (gm/dl)	18.4 (0.3)	15.8 (1.7)	0.004
PCV (%)	51.8 (1.1)	44.6 (4.4)	0.005
SGPT (IU/ml)	36.4 (3.0)	34.7 (1.9)	0.21
Weight (kg)	58.5(3.9)	57.5(2.9)	0.53
Smokers' polycythaemia managed initially with Venesection (n = 11)			
Hb (gm/dl)	20.1 (1.0)	17.6 (2.1)	0.002
PCV (%)	55.4 (2.2)	48.7 (6.0)	0.002
SGPT (IU/ml)	33.4 (12.1)	32.7 (5.4)	0.80
Weight (kg)	56.8(5.8)	55.6(5.1)	0.62
Overweight related polycythaemia managed initially with venesection (n = 22)			
Hb (gm/dl)	19.9 (2.0)	16.7 (1.6)	<0.001
PCV (%)	54.4 (4.5)	46.8 (4.3)	<0.001
SGPT (IU/ml)	69.3 (22.9)	52.5 (12.6)	<0.001
Weight (kg)	71.9(12.3)	62.5(11.1)	0.01
Polycythaemia due to mixed causes managed initially with venesection (n = 11)			
Hb (gm/dl)	19.9 (1.3)	16.2 (1.4)	<0.001
PCV (%)	53.9 (2.8)	45.1 (2.0)	<0.001
SGPT (IU/ml)	50.4 (17.3)	40.0 (11.9)	0.001
Weight (kg)	66.9 (8.7)	57.2(7.3)	0.01

and PCV dropped significantly after cessation of smoking.[2]

DISCUSSION[2]

After the intervention with weight reduction, there was significant relief of symptoms in both primary and secondary polycythaemia (p value 0.08) and significant reduction in haemoglobin and PCV in both groups compared to pre-intervention levels (p values <.05). Though all had benefit on weight reduction, the main focus of our study was on the secondary polycythaemia due to weight gain. There was a strikingly high prevalence of secondary polycythaemia in the subjects with overweight or obesity, which was independent of smoking, heart, lung or kidney diseases, and this appears due to chronic hypoventilation probably due to reduced chest wall compliance. There were 22 of them and all of them normalised their Hb and PCV after achieving weight reduction. The weight reduction in this subgroup ranged from 2 to 15 kg with a mean of 9.13kg. The percentage of weight reduction achieved ranged from 2.7 to 25 percent, with an average of 12.3 percentage . The mean weight reduction achieved in the whole study group was 9.13kg. The study concluded, that weight gain and overweight as the commonest reason for secondary polycythaemia in the subjects. Lifestyle changes, change in food habits and decreased physical activity have led to the obesity epidemic across the globe. Out of 22 patients with polycythaemia of overweight / obesity, 19 were having raised SGPT levels suggesting fatty liver disease (NASH) as the commonest association of secondary polycythaemia. There were 13 were diabetic and 8 were having hypertension which are the other common association of being overweight or obese. In those subjects with overweight or obesity, there was not only reversal of polycythaemia, the elevated liver enzymes, diabetes and hypertension too became better after weight reduction. The difference was statistically significant with all p values less than 0.001. The observation that 86% of those with secondary polycythaemia due to weight gain, also had NASH, and both normalised on weight reduction, permits us to conclude that both are lifestyle disorders. Three other studies before, which was the basis of the hypotheses for this study, showed reversal of diabetes, NASH and hypertension on weight reduction and it was then observed that some of the polycythaemia too reversed.[20-22] Out of 11 patients with mixed cause for polycythaemia, 3 were diabetic, 4 were having hypertension and 7 were

Table 5. Overweight related polycythaemia who were managed initially with Venesection (n = 22)[2]

	Pre intervention	Post intervention	p value
Hb gm/dl (SD)	19.9 (2.0)	16.7 (1.6)	<0.001
PCV % (SD)	54.4 (4.5)	46.8 (4.3)	<0.001
SGPT IU/ml (SD)	69.3 (22.9)	52.5 (12.6)	<0.001

having raised SGPT levels. These findings underscore the importance of looking at all the comorbidities with equal concern while managing patients irrespective of the nature of the disease. Becoming overweight, even without being frankly obese, as the commonest cause for secondary polycythaemia is thus a conclusion from this study. The very same patients were having either NASH, hypertension or diabetes or some of them had all of these, and the findings make us consider secondary polycythaemia of weight gain as yet another lifestyle disorder.

Previously there were only case reports of polycythaemia with hypertension and erythrocytosis without splenomegaly or leucocytosis, which were being described as Gaisbock syndrome.[3] This study asserts that what was described originally as Gaisbock syndrome was in fact secondary polycythaemia due to weight gain. Interestingly one subject, in the overweight category had a BMI less than 23 only, but he had 5Kg weight gain from his previous normal, and after weight reduction by 4 Kg, had normalised his Hb and PCV too. This points to another hypothesis that the assessment of desired normal weight based on some arbitrary or imaginary normal range has to be given up and the desired weight and BMI has to be individualised.[2,15]

Another useful observation of the study was that secondary polycythaemias were all seen in the age group between 30 and 39 years, while PV was seen in the 5th decade of life. In other words we should not evaluate those below 30 years for PV, unless there are sufficient justification to do that. Males were more commonly affected with all types of polycythaemias in the study, similar was the findings in other studies.[13-15] We found JAK2 positivity in 83% of those with primary polycythaemia.[3,15] Though JAK 2 mutation is highly sensitive for picking up PV, and is being used as a diagnostic criteria, we observed that it is most often not needed, as its cost-effectiveness and usefulness in diagnosis are questionable, unless we have a plan to start JAK 2 inhibitors upfront.[3,4,15] In addition JAK 2 negative does not rule out primary polycythaemia, since JAK2 exon 12 have been described in JAK2V617F negative patients with PV.[16] It goes without saying that we need to diagnose PV with clinical features and hemogram alone.[14,15] Another point noted in the study is that, just because JAK 2 is positive one should not make a conclusion that the polycythaemia is entirely due to PV. In other words the causes of secondary polycythaemia should be evaluated even if the patient is JAK2 positive. Even after the study we continue to see increasing numbers of polycythaemia due to weight gain and their definitive reversal after weight reduction. We had one patient who was originally labelled as PV since JAK 2 was positive, and was getting hydroxyurea. But after weight reduction he had normalised his Hb, PCV and the liver enzymes and is no longer on hydroxyurea. In this patient there was a need for weight reduction for the NASH and to our surprise all these reversed and for the last one year he has normal haematocrit and no longer needs any venesection.[2]

There were only very few studies evaluating the effect of lifestyle interventions, with special focus on dietary modifications. We found that excess carbohydrate intake was the primary reason for polycythaemia of weight gain and they contributed to adverse outcomes with PV as well. One previous study which evaluated the effect of fruits, vegetables, and coffee intake with MPNs have shown that those taking more carbohydrate had a higher risk of PV compared to those with lower carbohydrate intake.[17] Even patients with PV had improved well-being and needed less frequent venesections without increasing hydroxyurea or JAK 2 inhibitors.[18,19] The common comorbidities were non-alcoholic fatty liver disease, diabetes and hypertension in the study population, especially so in the overweight polycythaemia. They were consuming excess carbohydrates with decreased intake of vegetables, became overweight and developed polycythaemia of overweight and systemic diseases associated polycythaemia (p value <.001). It could be even possible that JAK 2 mutation and the subsequent development of PV itself could be the end result of this prolonged presence of inflammatory meditators due to weight gain.

We noted that majority of patients from all category had fast food intake. Majority of patients with polycythaemia vera and polycythaemias of mixed causes consumed vegetables less than once per week. Research works exploring diet and physical inactivity as risk factors of haematological neoplasms are many, but only a few interventional studies have established the protective effect of diet and physical activity in delaying disease progression and improving clinical and haematological outcomes. After extensive literature review, our study is the first of its kind in India, to evaluate the effect of dietary and physical activity in patients with primary and secondary polycythaemias.[1]

CONCLUSIONS AND RECOMMENDATIONS[2]

Weight gain associated polycythaemia is the commonest secondary polycythaemia in the study group. All those with secondary polycythaemia due

to weight gain had complete reversal of polycythaemia after weight reduction. In many patients there was reversal of NASH, diabetes, and hypertension too. The study observed that the originally described Gaisbock syndrome is secondary polycythaemia due to weight gain. We should not be looking for PV in patients below 30 years, unless there are compelling clinical pointers. Diet and lifestyle interventions led to statistically significant improvements in the clinical and haematological outcomes of both primary and secondary polycythaemias. Findings stress on the importance of advocating diet and lifestyle modification for all polycythaemias along with usual care, to improve the management outcomes. Future research is warranted in the same line to evaluate the effect of lifestyle modifications of polycythaemias in varied study settings

END NOTE

Author Information

1. Surumi AR, Junior Resient Medicine, PVS Hospital Kozhikode
2. Sasidharan PK, MD, FICP, FRCP Former HOD General Medicine and Division of Haematology, Govt Medical College, Kozhikode

Conflict of Interest: None declared

REFERENCES

1. Pearson TC. Apparent polycythaemia. Blood Rev. 1991 Dec;5(4):205-13.
2. Rahim SA, Sasidharan PK (2023) Polycythaemia- Clinical Profile and Impact of Lifestyle Change with Weight Reduction. J Thrombo Cir.9:241.
3. Krishnamoorthy P, Gopalakrishnan A, Mittal V, Kalla A, Slipczuk L, Rangaswami J, et al. Gaisböck syndrome (polycythaemia and hypertension) revisited: results from the national inpatient sample database. J Hypertension. 2018 Dec 1;36(12):2420-4.
4. Levine RL, Pardanani A, Tefferi A, Gilliland DG. Role of JAK2 in the pathogenesis and therapy of myeloproliferative disorders. Nat Rev Cancer. 2007; 7:673–683.
5. Sasidharan PK. Polycythaemia–How and How Much to Investigate? Medicine Update. Association Physicians India. 2008;18(46):358-67.
6. Tefferi A. Mutations galore in myeloproliferative neoplasms: would the real Spartacus please stand up? Leukemia. 2011; 25:1059–1063.
7. Weil MH, Prasad AS. Polycythemia of obesity: further studies of its mechanism and a report of two additional cases. Annals Internal Med. 1957 Jan 1;46(1):60-7.
8. Alkhedaide AQ. Tobacco smoking causes secondary polycythemia and a mild leukocytosis among heavy smokers in Taif City in Saudi Arabia. Saudi J Biological Sciences. 2020 Jan 1;27(1):407-11.
9. Sever M, Newberry KJ, Verstovsek S. Therapeutic options for patients with polycythemia vera and essential thrombocythemia refractory/resistant to hydroxyurea. Leuk Lymphoma. 2014; 55:2685–2690.
10. Sasidharan PK, Nutrition, balanced diet and good lifestyle, chapter 20, Guide to Clinical Practice in Post Covid World. KSG Publications; 2020; 371-407.
11. Sasidharan PK, Good lifestyle and practical tips on balanced diet, chapter 21, Guide to Clinical Practice in Post Covid World. KSG Publications; 2020; 408-423.
12. Tefferi A, Vardiman JW. Classification and diagnosis of myeloproliferative neoplasms: the 2008 World Health Organization criteria and point-of-care diagnostic algorithms. Leukemia. 2008 Jan;22(1):14-22.
13. Passamonti F. How I treat polycythemia vera. Blood.2012;120: 275–284.
14. Augustine T, Sasidharan PK, Shaan M, Polycythaemia Rubra vera-clinical profile & predictors of thrombotic complications. Blood Res Transfusion J. 2017;1(2):19-23.
15. Stuart BJ, Viera AJ. Polycythemia vera. Am Fam Physician. 2004; 69:2139–2144.
16. Sasidharan PK, Polycythaemia evaluation and management made easier
17. Chapter 19, Guide to Clinical Practice in Post Covid World. KSG Publications; 2020; 347-370
18. Broséus J, Park JH, Carillo S, Hermouet S, Girodon F. Presence of calreticulin mutations in JAK2-negative polycythemia vera. Blood, Journal American Society Hematol. 2014 Dec 18;124(26):3964-6.
19. Podoltsev NA, Wang X, Wang R, Hofmann JN, Liao LM, Zeidan AM, et al. Diet and risk of myeloproliferative neoplasms in older individuals from the NIH-AARP Cohort. Cancer Epidemiol Prev Biomarkers. 2020 Nov 1;29(11):2343-50.
20. Tibes R, Mesa RA. Emerging drugs for polycythemia vera. Expert Opin Emerg Drugs 2013; 18:393–404.
21. Tefferi A, Rumi E, Finazzi G. Survival and prognosis among 1545 patients with contemporary polycythemia vera: an international study. Leukemia. 2013; 27:1874–1881.
22. Afsheen Ali, Sasidharan PK, "Impact of Diet and Lifestyle Modification and Weight Reduction on Essential Hypertension." Clinical Case Reports and Clinical Study, 2022; 6(1).
23. Jamsheena P, Sasidharan PK. Management of Non-Alcoholic Fatty Liver Disease with Diet and Lifestyle Modification. Ann Clin Med Res. 2020; 1(2): 1009
24. Divakaran S, Sasidharan PK. Diet and Lifestyle to Prevent, Control or Reverse Type 2 Diabetes. Ann Clin Med Res. 2020; 1(3): 1013