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Oxygenation of blood in varicose veins

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Summary
The aim of this prospective, controlled study is to compare the oxygen tension of blood from varicose veins with that from both the antecubital vein of the same patients and the normal long saphenous vein of normal controls at high altitude in Abha (8,000 feet above sea level), Saudi Arabia. Forty-two subjects (21 normal controls and 21 with uncomplicated primary varicose veins) had blood samples taken from 41 normal long saphenous veins and 35 varicose veins near the ankle, respectively (a total of 76 lower limbs). Samples were also taken simultaneously from the right antecubital vein in all the subjects. There was no difference in either oxygen tension (pO_2) or saturation (sO_2) between blood from varicose veins and blood from normal long saphenous veins. However, in patients with varicose veins, both pO_2 and sO_2 of varicose veins blood were significantly higher than those of arm venous blood (P = 0.009 and P = 0.018, respectively). In normal subjects, blood from normal long saphenous veins had also significantly higher sO_2 (P = 0.001) than that from arm veins, but pO_2 was not higher. In conclusion, the theory of arteriovenous communication is poorly founded. There must be other more important explanations for the pathogenesis of varicose veins.

Keywords: *Varicose veins, long saphenous veins, antecubital veins, oxygen tension, oxygen saturation.*

Résumé
Le but de cette etude prospective controlle est de comparer lu tension d'oxygene des sang dans les varices avec celui des veins anticubitales du meme patient et les reins normales sapheneuses des controles normaux a altitude elevee a Abha (8,000 pieds au dessus du nivenu de la mer), en Arabie Saouditesrubie Saondite. 42 sujets (21 controles et 21 controles et 21 ayant des varices non-compliques) ont fourni du sang 41 longues veins saphineuses et 35 varices pres de la cheville, respectivement (untotal de 76 members inferieurs). Des echantillons etaitnt aussi recenillis simultarienment de la veines anticubital droite sur tons les sujets. Il n'yarant pas de difference de pression d'oxygene (PO_2) ou de suturation d'pxygene (sO_2) entre le sang des varices et celui des longues veins sapheneuses. Cependant, chez les patients avec des varices, PO_2 et sO_2 du sang etaient signficantivement plus eleves que ceux du sang veineux des bras (P = 0,009 et P = 0,018) respectivement). Chez les sujets normaux, le sang des longues veines sapheneuses avait aussi une saturation plus elevee de O_2 ($s0_2$); P = 0,001 que celui des veines du bras, mair $P0_2$ etait pas plus eleve. En conclusion, ta theorie, des communications artero-veineuses est paurement fondee. Il doit y avaoir d'autres importatants explication pour la pathogenese des varices.

Introduction

There are several conflicting theories of the etiology of primary varicose veins (VVs). Incompetence of the saphenous vein valves, an inherent weakness of the walls of the veins, and the presence of arteriovenous (AV) fistulas are popular examples. The exact cause of the phenomenon remains unclear. Currently, controversy surrounds the contribution of AV communications to the cause of varicose veins. Some investigators postulate the presence of AV communications based on studies reporting increased pO_2 in blood within VVs compared with that in normal veins [1]. In 1843, Pigeaux observed that blood from varicose veins was sometimes as red as that from arteries [2]. This observation was pursued by Blalock who reported a higher oxygen content in varicose veins than in normal veins [3]. These findings were corroborated by later authors [4,5]. If AV anastomoses are present, localized turbulence and increased pressure may lead to wall dilatation and valvular incompetence. The aim of this study was to compare the oxygen tension of blood from varicose veins with that from both the antecubital veins of the patients and from normal long saphenous veins. This study was conducted at Asir Central Hospital (ACH) in the city of Abha (Saudi Arabia), which is located 8,000 feet above sea level [6]. ACH is the main referral hospital for the whole Asir region of southern Saudi Arabia. It is also the teaching hospital of the College of Medicine, King Khalid University.

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Materials and methods

A total of 42 subjects were studied. Varicose veins (VVs) were present in 21 patients (5 females and 16 males) and 21 were normal controls (16 females and 5 males). The mean age of the VVs patients was 40.8 years ± 9.4 and that of the control group was 28.5 years ± 6.8. Patients with varicose veins were examined both clinically and by continuous wave Doppler (CWD) ultrasound probe to confirm the presence of superficial venous reflux; none of them had evidence of deep venous disease. Patients were chosen on the basis of the presence of well-developed, full-blown distal calf varicosities, as close to the ankle as possible. An informed consent was obtained from every patient who volunteered for the study. We explained the procedure of blood sampling to the patients and that it was for purely research purposes and not for diagnosis or treatment. Control subjects were young and healthy volunteer members of the Department of Surgery (nurses and doctors). Members of the control group did not undergo Doppler examination, but were examined clinically to exclude the presence of asymptomatic varicose veins. None of them had history of varicose veins in the past.

In patients with VVs, blood was taken from a varicosity (blowout) of the calf as close to the medial malleolus as possible using a 2 ml heparinized syringe and a 23 Fr gauge needle. Patients were asked to be standing up for a minimum of 5 minutes at the end of the clinical and Doppler examination before sampling. Patients were then asked to sit down and another sample was taken from the right antecubital vein. In normal subjects, blood was taken from both long saphenous veins, 1-2 cm proximal to the medial malleolus while standing up for at least 5 minutes by the same technique as previously described. Blood was then taken from the right antecubital vein after they sat down.

The syringes containing the blood samples were put on ice and sent immediately to the laboratory for blood gas analysis. The venous oxygen tension (pO_2) and saturation (sO_2) were then determined by means of a Radiometer ABL 510 gas analyzer (Radiometer, Copenhagen, Denmark), which was in constant use. We employed Mann-Whitney U and Wilcoxon Signed-Ranks Test to analyze variances and to calculate 2-Tailed P value, using SPSS for Windows version 6.0.

Results

The oxygen tension (pO_2) of varicose veins blood (31.0 mmHg ± 14.78) was similar to that of blood taken from normal long saphenous veins (31.0 mmHg ± 8.03) [Fig. 1]. Oxygen saturation (sO_2) was also similar at 52.3% ± 15.63 and 54.8 % ± 17.65, respectively. The mean pO_2 of arm venous blood in the control group was higher (29.06 mmHg ± 11.11) than that of arm venous blood in varicose vein patients (25.46 mmHg ± 5.53) ($P = 0.000$) [Fig. 2].

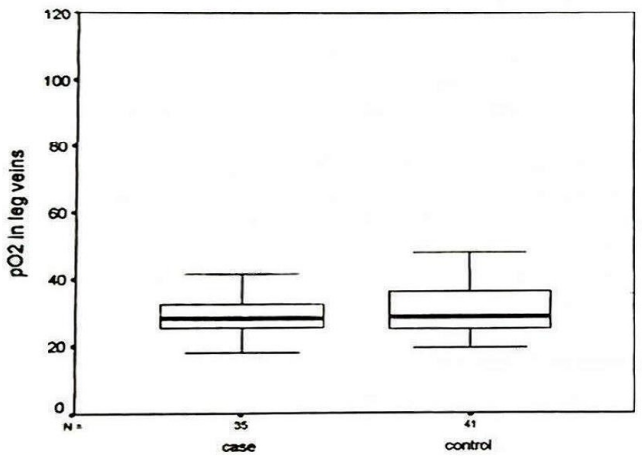

Fig. 1: Oxygen tension (pO_2) in leg veins of varicose vein patients and normal controls. $P = 0.570$ (Mann-Whitney U-Wilcoxon Test)

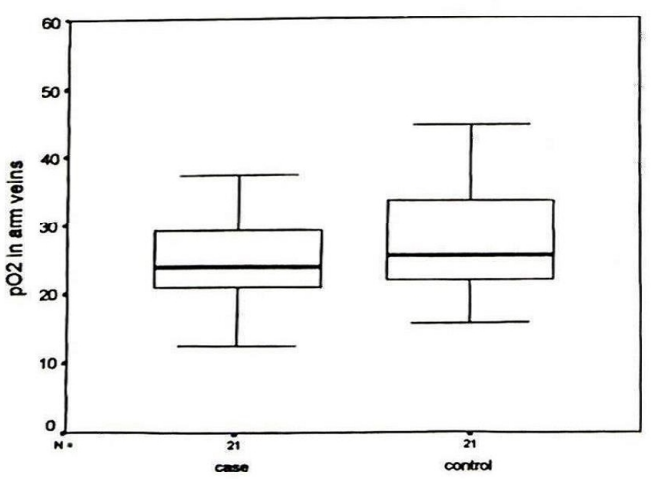

Fig. 2: Oxygen tension (pO2) in arm veins of the whole group P = 0.000 (Independent samples t-test)

However, in both varicose vein patients and in normal controls, both the oxygen tension (pO_2) and saturation (sO_2) tended to be higher in the lower limbs than in the upper limbs. In varicose veins patients, pO_2 of varicose blood (31.0 mmHg ± 14.78) was significantly higher than that of arm venous blood (25.5 mmHg ± 5.53) ($P = 0.009$). Oxygen saturation (sO_2) was also significantly higher, at 52.3% ± 15.63 compared with 43.6% ± 14.41 (P = 0.018) [Fig. 3 and 4]. In normal controls, the sO_2 of blood from long saphenous veins (54.8% ± 17.65) was also significantly higher than that from arm veins (42.9% ± 17.87) ($P = 0.001$), but the difference in pO_2 between leg and arm venous blood was not significant (31.0 mmHg ± 8.03 and 29.1 mmHg ± 11.11, respectively) [Fig. 3 and 4].

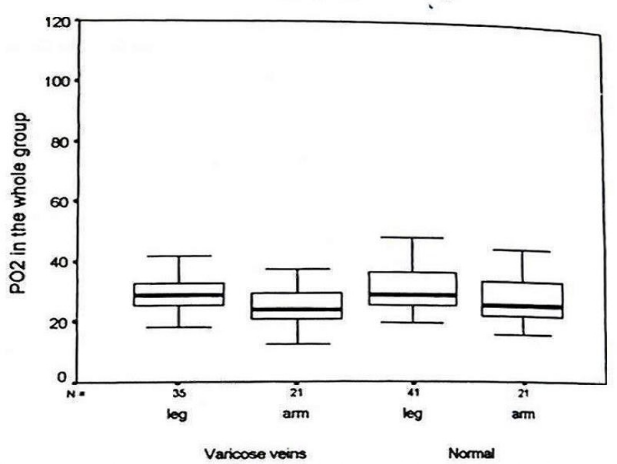

Fig. 3: Oxygen tension (pO2) in arm and leg veins of varicose veins patients and normal controls. $P = 0.009$ and $P = 0.095$ respectively (Wilcoxon signed ranks test)

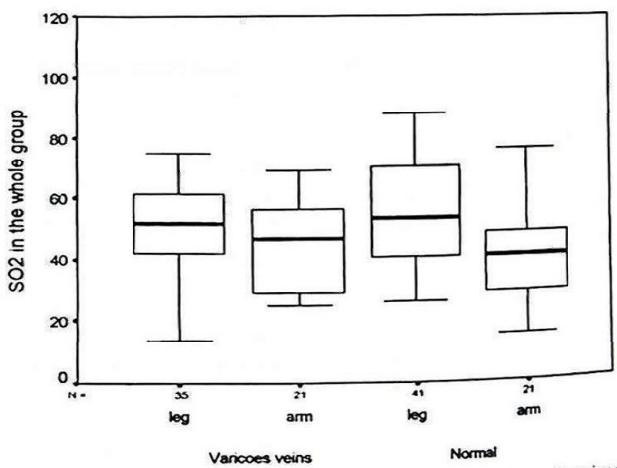

Fig. 4: Oxygen saturation (sO2) in arm and leg veins of varicose veins patients and normal controls. $P = 0.018$ and $P = 0.001$ respectively. (Wilcoxon signed ranks test)

Discussion

The incidence of varicose veins (VVs) increases with age; the peak incidence occurs in the 6th decade of life. Less than 1-2% of adults report varicose veins disease in their 20s, compared with up to 72% of women over 60. VVs are not only more common in women, but also occur earlier in life, leading to a female:male ratio of 5-6:1 in the 3rd and 4th decade of life. However, this ratio decreases as the population ages to a ratio of 1-2:1 after the 6th decade [1].

In the present study, most of our VVs patients were in their 5th decade. The Female:Male ratio was 1:3 which is almost the opposite of the natural incidence in this age group. However, apart from the sex of the patient, we tried to sample the blood of the full-blown picture of the distal varicosities and

apparently, male patients usually present with a more severe form of the disease. The other point is that male patients were more agreeable in volunteering for blood sampling of their varicosities, while most of our female patients did not agree. With the preconception that VVs get more common with the increasing age of the individual, as previously explained, we tried to recruit a younger and healthy group of controls to minimize the exclusion of those with any evidence of the disease. The great majority of the nurses in our hospital are young females in their 3rd decade and they readily volunteered for this study. Furthermore, we are not aware of previous evidence that there is a difference in either blood oxygen tension or saturation between the two sexes.

In his original work, Blalock compared blood from varicose veins with that from normal veins in the unaffected limb, using a Van Slyke-Neil manometric blood gas analyzer (which measures blood gas volume, not oxygen tension) [3], Scott *et al.* used a group of normal controls and found that the oxygen tension were higher in blood from varicose veins than in normal veins in the supine position. They also showed, for the first time, that while supine, superficial venous blood in the leg had higher oxygen content than that in the arm, whether varicose veins were present or not. They also confirmed that venous oxygen tension in the leg falls on standing, in which position there is no significant difference between the two groups [7]. In the present study, we have shown that, also on standing, superficial venous blood in the leg has higher oxygen saturation than that in the arm, whether varicose veins are present or not. We have also confirmed that on standing, there is no significant difference in either oxygen pressure or saturation between the two groups. With patients in the standing position, Reikers and Scorlie even found that the oxygen tension in blood from varicose veins was actually lower than that in blood from normal veins [8]. The other interesting new finding in our study, is that oxygen pressure of arm venous blood in normal controls was higher than that in varicose veins group.

Haimovici reviewed the evidence for the presence of arteriovenous communications in patients with varicose veins [9,10]. This included the early filling of varicose veins on arteriograms, suggestive Doppler flow measurements [11] and direct visualization at operation [9,12,13]. Piulachs and Vidal-Barraquer in 1953, attributed their findings of higher pO_2 in varicose veins to the presence of these abnormal vessels and described their presence at operation [14]. However, examination of the microcirculation surrounding VVs with macro-aggregates revealed no evidence of increased AV shunting in the area of VVs [15]. Other investigators suggest that VVs are associated with peripheral vasodilatation including the venous capillaries, which may result in the opening of the capillary bed leading to a functional AV shunt as a result - not a cause - of VVs [1].

Our finding that the oxygen saturation (sO_2) of the venous blood of the lower limbs is higher than that of the arms in both groups, suggests that a physiological rather than a pathological mechanism is at work. Different metabolic rates in the foot and the hand may explain this phenomenon. The ratio of red "slow twitch" fibers to white "fast twitch" is greater in the postural muscles of the leg [16] and as the two types of fibers have different metabolic rates [17], it is reasonable to expect different oxygen saturation of the venous blood draining them. Another explanation for the difference in venous oxygen tensions in the leg and arm could be that the specific blood flow to the leg is higher than that to the arm [7]. All of these avoids the need to suggest the presence of abnormal arteriovenous communications.

The other interesting finding of significantly higher oxygen tension in arm veins of control people compared with varicose veins group, could be related to the younger age of the control group in this study. It may also be related to the hemodynamic disturbance and venous insufficiency in varicose veins patients, especially on standing.

However, this study was conducted in Abha, southern Saudi Arabia, which is 8,000 feet high above sea level. Our findings therefore may or may no hold for an area of low altitude or at sea level. It may therefore be important in a future study to compare these findings with those obtained from patients living at sea level.

Conclusion

We have confirmed in this study, that there is no difference in either oxygen tension or saturation between varicose veins blood and blood from normal long saphenous veins. We have also demonstrated that the oxygen saturation of venous blood in the lower limbs is higher than that in the upper limbs, irrespective of the presence of varicose veins. These can be explained based on the physiological and hemodynamic mechanisms that control the circulation. There is no evidence in the present study to support the theory of arteriovenous communications as a contributory factor in the pathogenesis of varicose veins.

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