The effect of rooibos tea on the type I allergic reaction

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Summary

The hypothesis that Aspalathus linearis (rooibos tea) has anti-allergic properties was tested. The type I skin reaction was employed as a measurable criterion of allergic disease. Local application and the ingestion of rooibos tea failed to inhibit these reactions, and it is highly unlikely that this substance has any value in the treatment of this type of allergic disease.

Rooibos tea, which is brewed from the dried stems and leaves of the indigenous plant Aspalathus linearis, contains the flavonol substances quercetin and luteolin. These substances have antihistaminic properties, as demonstrated by their ability to inhibit histamine-induced contraction of a guinea-pig ileum preparation.1,2 Rooibos tea is widely employed as a substitute for cow’s milk in allergic children, and the popular opinion is that rooibos tea has anti-allergic properties. The purpose of this study was to determine the influence of rooibos tea on a common manifestation of the atopic status, the type I skin response.

Materials and methods

Skin-prick tests with 16 commonly inhaled allergens were conducted on both forearms of 7 adult volunteers who had symptoms of allergic disease (asthma or hay fever). Antihistaminic drugs were discontinued 1 week before the study.

Total serum IgE levels (Phadebas radio-immunosorbent test) and specific IgE (Phadebas radio-allergosorbent test) for cat and dog epithelium and grass pollens were determined on the sera of all participants before the study. An experienced nursing sister conducted the skin-prick tests. After 10 minutes two independent observers assessed the size of the induration of the positive reactions on both arms with the aid of a Bencard skin-test gauge. Readings of induration size were increased by a value of 0.5 mm when pseudopodia was present. A mean value was obtained for each allergen after comparison of the reactions on the right and the left arm.

In order to determine the repeatability of skin reactions to the same allergen on different days, and to compare the size of skin reactions on different arms, this procedure was repeated by the same team 4 - 7 days later.

The effect of rooibos tea on the type I skin reaction was assessed 1 week later in the following manner. Each volunteer drank 500 ml of black rooibos tea with sugar at 09h00, 12h00 and 15h00. No other food or drink was permitted during this period. Tea was prepared by adding 25 grams of rooibos tea to 1 litre of boiling water. Five minutes later the leaves were removed by pouring the tea through a sieve. A rooibos tea poultice was prepared by soaking 500 ml of tealeaves in 500 ml of cold water for 30 minutes. This was applied to one of the forearms of each volunteer for a period of 15 minutes. The leaves were removed with running tap-water and the arm was dried with an absorbent paper towel without rubbing the skin.

The series of skin-prick tests was then repeated on both arms and the reactions recorded 10 minutes later by the same two independent observers.

Results

Elevated total serum IgE values in 4 subjects and elevated specific IgE for 1 or more of the 3 antigens in every participant supported the clinical diagnosis of allergic disease. Before the ingestion of rooibos tea the skin reactions to 14 of the 16 antigens were of similar size on the right and left arms of the patients. Small differences in the size of the induration of the positive reaction (P<0.05) were found for 2 antigens. A multiple comparison test was employed for the statistical analysis of the skin induration as recorded on the 2 control days and on the day on which the influence of rooibos tea was studied. The size of the reactions recorded on the 2 control days was reproducible for all the antigens in the 7 participants.

The size of skin induration to 12 of the antigens remained unchanged after the ingestion and local application of rooibos tea. The skin reaction to 4 antigens (house dust, grass pollen, dog epithelia and Aspergillus fumigatus) was larger on the day of treatment with tea than on the control days (P<0.01 to <0.05).

Discussion

The results of this study do not support the popular view that rooibos tea has potent anti-allergic properties. Histamine, an important product of immediate-type allergic reactions, contributes to the clinical signs and symptoms of nasal, skin, lung and gastro-intestinal tract allergic disease.3 Type I skin reactions are extremely sensitive to the inhibitory effects of antihistamines.4 No such effect could be demonstrated after subjects had drunk large quantities of rooibos tea and after local application of tea-leaves to the skin.

The slightly larger size of reactions recorded with 4 antigens on the day of treatment was interpreted as representing increased local sensitivity due to repeated skin testing.

However, the fact that the quercetin and luteolin in rooibos tea inhibit histamine- and acetylcholine-induced contractions in guinea-pig ileum has to be further considered.1,3 This antispasmodic effect may relate to the inhibition of neurogenic factors specific to the smooth muscle of the gut. The results of this study do not exclude the possibility that rooibos tea has antispasmodic properties in children who react to ingested allergens with gut wall spasm. The therapeutic value of rooibos tea in allergic diseases of the skin, lungs and nose is questionable.

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Lymph node enlargement in rheumatoid arthritis

A case of angio-immunoblastic lymphadenopathy

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Summary

A patient with rheumatoid arthritis and angio-immunoblastic lymphadenopathy is presented and discussed in relation to other forms of lymphadenopathy which are seen in this disease. The angio-immunoblastic lymphadenopathy seen in this patient appears to have been shortlived, and while it was active there was an associated increase in the levels of serum immunoglobulin G.


Lymphadenopathy is found in 29-82% of patients with rheumatoid arthritis, and it is generally regarded as part of the rheumatoid process. Lymphadenopathy is frequent in the axillae, groins and epitrochlear regions and less common in the neck. The degree of lymph node enlargement is greater in males, and in patients with seropositive disease. It may be generalized, but it is usually most marked proximal to areas of active synovitis. The development of a generalized lymphadenopathy in a patient with rheumatoid arthritis should always raise the possibility of a lymphoma because there is a slightly increased frequency in rheumatoid arthritis with features of Sjögren's syndrome. The following case report describes the development of an angio-immunoblastic lymphadenopathy in a patient with rheumatoid arthritis.

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Case report

A woman, aged 44 years, in January 1975 developed a polyarthritis involving her hands, shoulders, elbows, knees and feet which progressed rapidly. Two months later she was admitted for treatment. Examination showed a pale individual with no evidence of lymphadenopathy or hepatosplenomegaly. There was active polyarthritis involving wrists, elbows, shoulders, knees, ankles and feet, and the Ritchie articular index was 33. There were striking fixed hyperextension (swan-neck) deformities at the proximal interphalangeal joints of the hands and fixed flexion deformities of the metacarpophalangeal joints, with mild synovitis. No subcutaneous nodules were found. Radiographs of the hands and feet showed the features of stage 3 rheumatoid arthritis, which was complicated by a venous thrombosis with pulmonary embolism requiring warfarin.

Following discharge the patient continued to use diclofenac sodium 150 mg and indomethacin 100 mg/d as an outpatient until she was readmitted in March 1977 for control of arthritis. In May 1977 corrective surgery of the left hand was undertaken and this was complicated by a further pulmonary embolus. Warfarin therapy was again instituted and continued until 1980. In October 1977 the patient developed three ulcers on her left foot, diagnosed as pyoderma gangrenosum, which responded to conservative treatment after 3-4 months. In June 1978, following a meal of fresh fish, she developed a tachycardia and an urticarial rash with superficial buttock ulcers. This acute allergic reaction responded rapidly and well to antihistamines. Three months later the patient required admission to hospital for treatment of dehydration following a week of profuse diarrhoea. Examination revealed an ill, pyrexial patient with a morbilliform maculopapular rash on the arms and upper trunk and a generalized lymphadenopathy in the neck, axillae and groins. The glands, which measured 2 cm on average were discrete, firm and non-tender. The spleen was not palpable and the arthritis was quiescent. Stool culture was negative. Results of blood tests are outlined in Table 1.

A 99mTc scan showed foci of colloid uptake in the left lung and in the bones of the spine. Bone marrow aspiration showed moderate megaloblastic maturation and dyserythropoiesis, lymphocytes constituting 35% of the cells, and increased plasma cells. A