Numerical changes in blood monocytes in bronchial asthma.

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Abstract

Numerical changes in peripheral blood monocytes were examined in 125 patients with bronchial asthma using a new direct method of counting blood monocytes. The number of monocytes in non-attack stages of bronchial asthma was similar to that of healthy controls. The monocyte count observed in overall cases showed a significantly higher value both in pre-attack and attack stages than in non-attack stages. Changes in the number of monocytes in an individual spontaneous asthmatic cycle tended to increase in pre-attack stages, increase more markedly during asthma attacks, then to decrease after the attack was alleviated. Monocytes in cases with a positive test for bronchial challenge to house dust extract changed in almost the same manner as for spontaneous asthma attacks. The number of monocytes did not change during bronchospasm provoked by inhalation of acetylcholine. Exercise-induced asthma patients exhibited indefinite changes of monocytes; that is, some cases showed a significant increase in the number of monocytes related to the asthma cycle, but other cases did not show any appreciable change. These findings suggest that the number of monocytes in the peripheral blood may change in close relation to asthma attacks elicited by allergic reactions.

KEYWORDS: blood monocytes, nonspecific esterase staining, bronchial asthma

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NUMERICAL CHANGES IN BLOOD MONOCYTES IN BRONCHIAL ASTHMA

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Abstract. Numerical changes in peripheral blood monocytes were examined in 125 patients with bronchial asthma using a new direct method of counting blood monocytes. The number of monocytes in non-attack stages of bronchial asthma was similar to that of healthy controls. The monocyte count observed in overall cases showed a significantly higher value both in pre-attack and attack stages than in non-attack stages. Changes in the number of monocytes in an individual spontaneous asthmatic cycle tended to increase in pre-attack stages, increase more markedly during asthma attacks, then to decrease after the attack was alleviated. Monocytes in cases with a positive test for bronchial challenge to house dust extract changed in almost the same manner as for spontaneous asthma attacks. The number of monocytes did not change during bronchospasm provoked by inhalation of acetylcholine. Exercise-induced asthma patients exhibited indefinite changes of monocytes, that is, some cases showed a significant increase in the number of monocytes related to the asthma cycle, but other cases did not show any appreciable change. These findings suggest that the number of monocytes in the peripheral blood may change in close relation to asthma attacks elicited by allergic reactions.

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In recent years, the blood monocyte-vascular endothelial system has been under study in relation to immunological reactions (1-3). There are, however, many unclear mechanisms in this system, and only a few trials have studied the relationship between blood monocytes and allergic diseases (4-6). Snyderman et al. (5) reported that monocyte chemotaxis was depressed in patients with atopic eczema. Furuikawa et al. (6) described the chemotactic dysfunction of monocytes in respiratory allergies. Monocytes and macrophages are thought to be necessary for the initiation and control of immunological reactions. However, the functions of monocytes in allergic diseases are still unclear.

Methods for nonspecific esterase staining of monocytes have been developed (7-9).

We previously reported a simple method for direct count of blood monocytes using a nonspecific esterase staining method (10). In the present study, we observed numerical changes of blood monocytes in bronchial asthma using
this direct counting method.

MATERIALS AND METHODS

The subjects were 125 asthmatic patients treated at the Asthma Clinic of Okayama University (60 males, 65 females, Age 12-82 years, mean 43.2 ± 15.1 years). Forty healthy subjects without allergic disorders (20 males, 20 females, age 20-65 years, mean 35.8 ± 14.4 years) were selected as control subjects.

Direct counting of blood monocytes was performed according to the method previously reported (10). The numbers of monocytes in all cases were calculated in relation to the asthmatic cycle, the types of asthma and the use of corticosteroids. The number of monocytes during asthma attacks was observed in 4 patients every three hours for 24 h.

Bronchial provocation tests for house dust extract and acetylcholine were carried out and the number of monocytes were observed before, 20-30, 60, 90 and 120 min after the provocation tests.

The numerical changes in monocytes during exercise-induced asthma were also examined in the same way as in the provocation tests. Statistical analyses were performed using student’s t test.

RESULTS

The mean value for the monocyte counts in 40 healthy subjects was 245 ± 91/cmm. The mean value in 125 asthma patients was 215 ± 92/cmm in non-attack stages, 249 ± 86/cmm in pre-attacks (pre-attack: within 18 h before attack) and 336 ± 93/cmm in attacks. Patients free of attacks did not show a

Fig. 1. Numbers of blood monocytes in different stages of bronchial asthma (pre-attack: within 18 h before attack).
significant increase of monocytes compared with the healthy subjects. The difference between non-attack and pre-attack (P < 0.05), and between pre-attack and attack (P < 0.001) stages, was significant (Fig. 1).

The patients were divided into two groups, atopic and non-atopic type, and the difference in the monocyte count between the two types was discussed. In atopic asthma, the mean value of the monocyte count was 215 ± 88/cmm in non-attack stages and 225 ± 83/cmm in pre-attacks. The difference between the two groups was not significant. In attack stages, the number of monocytes increased to 343 ± 116/cmm, which was significantly higher than in pre-attack stages (P < 0.001). In non-atopic asthma, the mean value of monocytes was 215 ± 100/cmm in non-attack stages, 265 ± 86/cmm in pre-attack, and 329 ± 116/cmm in attacks. The difference between non-attack and pre-attack stages was significant (P < 0.05). The monocyte count in attack stages also showed a significant increase compared with pre-attack stages (P < 0.05). Non-atopic asthma showed a marked increase in the number of monocytes in pre-attack stages over atopic asthma (Fig. 2).

The influence of corticosteroids on the monocyte count was examined. In cases being treated without corticosteroids, the mean value of monocytes was 221 ± 96/cmm in non-attack stages and 243 ± 80/cmm in pre-attacks. There was no significant difference between the two groups. The mean value in attack stages, however, was 305 ± 78/cmm, which was significantly higher than during pre-attacks (P < 0.02). In steroid-dependent asthma patients in whom it was not possible to cease steroid hormone for more than 1 week each month over at least the proceeding 2 years, the mean value of the monocyte count.

Fig. 2. Number of monocytes in cases classified by asthma types.
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Fig. 3. Numerical changes in monocytes in relation to a spontaneous asthmatic cycle.

Fig. 4. Numerical changes in monocytes during positive bronchial provocation tests for house dust extract.

was 181 ± 61/cmm in non-attack stages, 263 ± 99/cmm in pre-attacks and 388 ± 98/cmm in attacks. The differences were statistically significant between non-attack and pre-attack stages (P < 0.02) and between pre-attack and attack stages (P < 0.005).
The monocyte count in each case showed a close relation to asthma attacks. Monocytes increased in pre-attack stages, increased more during attack stages and decreased after the asthma attack subsided (Fig. 3). Monocytes in patients with positive bronchial provocation tests for house dust extract showed the same tendency as those in spontaneous attacks. Monocytes, however, did not change.
when attacks were not provoked (Fig. 4).

The changes in monocytes during exercise-induced asthma were different from those in spontaneous or house dust-induced attacks. Two types of change in monocyte count were observed, and monocytes did not always increase either in pre-attack or in attack stages. Some cases showed the same tendency as the cases provoked by inhaled house dust extract. Other cases, however, showed no changes. These phenomena are very interesting in regard to mechanisms of exercise-induced asthma (Fig. 5).

Monocytes in bronchospasm elicited by inhalation of acetylcholine did not show any change. The results were in part similar to those for exercise-induced asthma (Fig. 6).

**DISCUSSION**

Blood monocytes are white blood cells and develop in the bone marrow. They migrate from the peripheral blood into tissues (mononuclear phagocyte system) (11). The main functions of monocytes in immunological reactions are phagocytosis and cytotoxic actions. Monocytes possess membrane receptors for IgG and C₃ which are closely related to the phagocytosis for antigen-antibody complexes or antigen-antibody-complement complexes (12).

Furthermore, monocytes also have histamine receptors (13), although the mechanisms of action are still unclear. The cytotoxic action of monocytes in ADCC (antibody dependent cell-mediated cytotoxicity) have been noticed in immunological reactions (14-16). These functions of monocytes are, however, controversial and remain unclear in allergic diseases.

Whether monocytes participate in asthma attacks is an interesting problem. The present study demonstrated that the number of monocytes increases in pre-attack and attack stages of spontaneous and specific allergen-induced attacks, although no increase occurs in bronchospasm induced by inhalation of acetylcholine. The results suggest that blood monocytes play a role in asthma attacks elicited by allergic reactions. On the other hand, the changes in monocytes during exercise-induced asthma were indefinite. An increased number of monocytes was observed in some cases related to the asthmatic cycle, but not in other cases.

It has been reported that anti-cholinergic drugs (17) and agents inhibiting release of chemical mediators are effective in exercise-induced asthma. These reports suggest the participation of the parasympathetic system and chemical mediators such as histamine and SRS-A (slow reacting substance A) in the initiation of exercise-induced bronchospasm.

The release of chemical mediators in exercise-induced asthma might be triggered by a nonimmunologic mechanism, but not by IgE-mediated reactions (18). Further investigation is necessary to clarify the relationship between the
participation of monocytes and mediator release in exercise-induced asthma.

We previously reported that basophils, target cells of IgE, change in close relation to the asthmatic cycle (19). The changes in monocytes during the asthmatic cycle are similar to those in basophils, which increase in pre-attack stages and decrease after the initiation of attacks. However, blood monocytes increase in pre-attack stages and begin to decrease following improvement.

This suggests that basophils participate in the initiation of attacks by the reaction of IgE to an inhaled allergen and monocytes exert their roles following antigen-antibody reactions. Although the functions of monocytes in bronchial asthma remain unclear, the results that non-atopic asthma shows an increase in monocytes in pre-attack stages more marked than in atopic asthma imply the participation of monocytes in reactions other than those mediated by IgE.

REFERENCES


