# Clinical Study of Vitiligo

## – *Comparative Study of Type A and Type B Vitiligo* –

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***Background*: The clinical behavior of vitiligo has not been clearly understood and hypothesis concerning the pathogenesis of the disease has been confusing and contradictory though autoimmune mechanisms have been considered important by many authors.**

***Objective*: The purpose of this study was to develop a better understanding of the clinical features and pathogenesis of vitiligo.**

***Methods*:We investigated clinical features of vitiligo in 1315 patients, and also compared the clinical course and features of non-segmental type(type A) and segmental type(type B) vitiligo patients to see whether the two types of vitiligo have a different pathogenic mechanism.**

***Results*: Previously reported clinical patterns of the disease were reviewed and compared with our data, and the different clinical findings between the two types which supported the hypothesis of Koga et al. that type A and type B vitiligo had a different pathogenesis and autoimmune mechanisms played a role only in type A were shown.**

***Conclusion*: We investigated the clinical characteristics of vitiligo in Korea and showed that the type A vitiligo might have a different pathogenic mechanism with type B.**
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*Key Words*: Clinical study, Non-segmental type, Segmental type, Vitiligo

Vitiligo is an acquired, sometimes familial depigmentary disorder of skin and hair. Even though there have been many basic[1-4] and clinical studies on vitiligo in Korea, those studies so far have been limited to the generalized type[5], childhood group[6], treatments[7-9], or a smaller group of patients[10,11] in comparison with the prevalence of vitiligo in the general population(1-2%)[12]. This clinical study was carried out in a larger group of patients than any of the previous studies done in Korea.

For the etiology of vitiligo, though it still remains obscure[13], autoimmune mechanisms have been considered as an important factor by many authors[14-16]. In 1988, Koga et al[17]. proposed different pathogenic mechanisms according to different clinical types. By dividing vitiligo patients into non-segmental type(type A)and segmental type (type B) and comparing the clinical features between them, they concluded that vitiligo of non-segmental type(type A) showed a high tendency to be associated with autoimmune mechanisms compared to segmental type(type B). We also included comparative clinical study between the type A and type B vitiligo to confirm whether the two types of vitiligo have different pathogenesis or that autoimmune mechanisms are confined to type A vitiligo.

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## MATERIALS AND METHODS

One thousand and three hundred fifteen patients with clinical and/or histopathologic diagnosis of vitiligo were evaluated by individual inter-

viewing at the dermatologic clinics of Sinchon Severance Hospital, Wonju Christian Hospital and Capital Armed Forces General Hospital, We had investigated the patients from September 1988 to June 1991. Careful personal and family history taking and physical examination were done on each patient to obtain the detailed clinical data. The patients were classified into localized, generalized and universal type according to the clinical classification of vitiligo suggested by Ortonne et al[12]. The extent of vitiligo was calculated by the "rule of nine".

For comparative study, the patients were divided into two groups by examining the distribution pattern of the patches of depigmentation: segmental type(type B), were depigmented patches were confined to a definite dermatome in the same manner as herpes zoster, and non-segmental type (type A), which did not show any dermatomal distribution or spreading of depigmented patches in all lesion. In each type of patient, above clinical parameters and history of diseases with proven or suspected allergic or immunological etiology were compared.

The data were collected and processed with a data base system(DBASE, Ashton-Tate Co.) in a personal computer. Statistics were analyzed by using the SPSS(Statistical Package for Social Science, SPSS Inc.) program to compare type A and B groups.

## RESULTS

### Clinical classification

Of the one thousand and three hundred fifteen patients, 609 cases were males(46.3%) and 706 cases were females(53.7%). Among them, there were 660 cases(50.2%) of the generalized type, 654 cases(49.7%) of the localized type and one case of universal type vitiligo(Table 1). In etiologic classification, 186 cases(14.1%) and 902 cases (68.5%) were definitely classified into type B and type A vitiligo and others were excluded.

### Age of onset and first visit

The mean age of onset was 21.9 years(male:20.7, female:22.9) and the mean age of first visit was 27.8 years(male:26.7, female:28.8). Minimal and maximal age of onset was at birth and 68 years. Minimal and maximal age of first visit was 3 months and 80 years.

### Duration

The mean duration was 5.9 years(male:6.1, female:5.9). Minimal and maximal duration was 20 days and 60 years.

### Distribution of the initial lesion

The initial lesion was single in 76.8%(1010 cases: male:75.5%, female:77.8%). Among them, the most common site of initial involvement was the face(24.5%), especially the forehead(19.2%), and the neck was more commonly involved in females (24.45) than in males(12.1%). An exposed area including the head, neck and arms(63.5%) was more frequently involved than an unexposed area (36.5%)(Table 2).

**Table 1.** Clinical classification*

| Clinical classification | Male(%) | Female(%) | Total(%) |
|---|---|---|---|
| Localized | 284 (46.7) | 370 (52.4) | 654 (49.7) |
| focal | 194 (31.9) | 249 (35.3) | 443 (33.7) |
| segmental | 85 (14.0) | 118 (16.7) | 203 (15.4) |
| mucosal | 5 ( 0.8) | 3 (0.4) | 8 (0.6) |
| Generalized | 325 (53.3) | 336 (47.6) | 661 (50.3) |
| acrofacial | 107 (17.6) | 83 (11.8) | 190 (14.4) |
| vulgaris | 49 ( 8.0) | 87 (12.3) | 136 (10.4) |
| mixed | 169 (27.7) | 165 (23.4) | 334 (25.4) |
| Universal | 0 (0.) | 1 (0.1) | 1 (0.1) |
| Total | 609 | 706 | 1315 |

*Methods of Ortonne et al.[12]

Table 2. Distribution of the initial lesion

| Distribution | Male(%) | Female(%) | Total(%) |
|---|---|---|---|
| Face | 99 (21.5) | 148 (27.0) | 247 (24.5) |
| Neck | 56 (12.1) | 134 (24.4) | 190 (18.8) |
| Scalp | 48 (10.4) | 65 (11.8) | 113 (11.2) |
| Arm | 15 (3.3) | 8 (1.5) | 23 (2.3) |
| Hand | 45 (9.8) | 23 (4,2) | 68 (6.7) |
| Leg | 38 (8.2) | 19 (3.5) | 57 (5.6) |
| Foot | 7 (1.5) | 13 (2.4) | 20 (2.0) |
| Abdomen | 34 (7.4) | 14 (2.5) | 48 (4.7) |
| Chest | 26 (5.6) | 33 (6.0) | 59 (5.8) |
| Back | 43 (9.3) | 31 (5.5) | 74 (7.3) |
| Axilla | 7 (1.5) | 4 (0.7) | 11 (1.1) |
| Genitalia | 15 (3.3) | 14 (2.6) | 29 (2.9) |
| Buttock | 3 (0.7) | 2 (0.4) | 5 (0.5) |
| Inguinal | 14 (3.0) | 19 (3.5) | 33 (3.3) |
| Flank | 11 (2.4) | 22 (4.0) | 33 (3.3) |
| Known | 461(100.0) | 549 (100.0) | 1010(100.0) |
| Unknown* | 148 | 157 | 305 |
| Total | 609 | 706 | 1315 |

*The number of patients who did not remember the initial lesion of disease

Table 3. Precipitating factors

| Factors | Male(%) | Female(%) | Total(%) |
|---|---|---|---|
| Trauma | 91 (54.5) | 52 (33.1) | 143 (44.1) |
| Emotional tension | 35 (20.9) | 50 (31.8) | 85 (26.2) |
| Sun burn | 15 (9.0) | 23 (14.6) | 38 (11.7) |
| Tanning | 13 (7.8) | 13 (8.3) | 26 (8.1) |
| Inflammation | 13 (7.8) | 5 (3.2) | 18 (5.6) |
| Pregnancy and delivery | 0 (0.) | 9 (5.8) | 9 (2.8) |
| Oral pill | 0 (0.) | 5 (3.2) | 5 (1.5) |
| Total | 167 | 157 | 324 |

## Progression of the disease at the time of visiting and symptoms

The disease was progressive in 73.6% of patients (male:74.2, female:73.1) at the time of visiting and only 1.3% showed regression of the disease. Some patients complained of pruritus(9.6%) or burning sensation(0.8%) as a symptom.

## Precipitating factors and previous treatments

324 patients(24.6%) had one of the seven types of precipitating factors in order of frequency:trauma history of the lesion site(44.1%), emotional tension(26.2%), sunburn, tanning, inflammation, and pregnancy, delivery and oral pill in women (Table 3). Also 21 patients had experience of improvement of the lesion with light exposure.

Most of the patients(1327 cases) had a history of previous treatment. Among them, about half of the patients had a history of topical steroid therapy(46.8%) and 21.3% had a history of sunlight exposure with topical oxsoralene(Table 4).

## Extent of depigmentation

71.6% of patients showed depigmented lesion of 5-10% and the mean percentage of depigmented

Table 4. Previous modalities of treatments

| Treatments | Male(%) | Female(%) | Total(%) |
|---|---|---|---|
| Topical steroid | 276 (50.0) | 303 (44.2) | 579 (46.8) |
| Sunlight + Topical oxoralene | 106 (19.2) | 158 (23.1) | 264 (21.3) |
| Oral Steroid | 80 (14.5) | 96 (14.0) | 176 (14.3) |
| Herb medicine | 32 ( 5.8) | 41 ( 6.0) | 73 ( 5.9) |
| UVR + Topical oxoralene | 29 ( 5.3) | 39 ( 5.7) | 68 ( 5.5) |
| UVR + Systemic oxoralene | 15 ( 2.7) | 33 ( 4.8) | 48 ( 3.9) |
| Sunlight + Systemic oxoralene | 8 ( 1.4) | 12 ( 1.8) | 20 ( 1.6) |
| Acupuncture | 6 ( 1.1) | 3 ( 0.4) | 9 ( 0.7) |
| Total | 552 | 685 | 1237 |

Table 5. Involvement of depigmented area

| Involvement(% of BSA) | Male (%) | Female(%) | Total(%) |
|---|---|---|---|
| < 5 | 419 (68.8) | 522 (73.9) | 941 (71.6) |
| 5 - 10 | 402 (16.7) | 97 (13.7) | 199 (15.1) |
| 10 - 20 | 55 (9.0) | 48 (6.8) | 103 (7.8) |
| 20 - 30 | 18 (3.0) | 18 (2.6) | 36 (2.7) |
| 30 - 50 | 9 (1.5) | 13 (1.9) | 22 (1.7) |
| 50 - 70 | 3 (0.5) | 3 (0.4) | 6 (0.5) |
| 70 < | 3 (0.5) | 5 (0.7) | 8 (0.6) |
| Total | 609 | 706 | 1315 |

BSA = body surface area

area was 7.3%(male:7.6, female:7.0)(Table 5). Poliosis was observed in 27.0%.

## Others

37.7% of male patients were students and 49.9% of female patients were housewives. Family history was present in 12.2% of patients:brother, parents, and grandparents in the order of frequency. 203 patients(15.4%) answered that their diseases were aggravated when the season changed: 154 patients (male:68 cases, female:86 cases)in summer, 33 patients in spring, 9 patients in fall, and 4 patients in winter. Koebner's phenomenon was observed in 237 patients(18.0%).

## Comparative clinical study of type A and type B vitiligo

We compared the above clinical parameters of 1,088 patients(902 type A and 186 type B) who could be definitely classified into two groups from 1315 patients. Among them the following different clinical features were found. The onset of type A vitiligo occurred over a wide age range, while most patients with type B became affected while they were young(Fig. 1). 67% of type B vitiligo noticed the first depigmented patches before the age of 25, whereas only 40% of type A vitiligo were suffering from the disease by that age. There was a statistically significant difference between age distributions in the two types($P<0.05$, $\chi^2$ test). The duration of type B vitiligo occurred over a relatively short period as shown in Fig. 2. The median period of duration of vitiligo was 1.3 years in type B, which was significantly shorter than type A(4.5 years)($P<0.001$, Wilcoxon rank sum test). As shown in Table 6, history of diseases with a proven or suggested allergic or immunological etiology which could affect the immune system were

found more frequently in type A vitiligo. Although not age matched, 142 of the 902 patients with type A vitiligo(15.7%) had a disease history which was a significantly higher frequency than that in type B, where 18 out of the 186 patients(9.7%) had a history of the disease($P<0.05$:$\chi^2$ test).

**Table 6.** History of diseases with proven or suggested allergic or immunological etiology

| Diseases | Type A (n=902) | Type B (n=186) | Total |
|---|---|---|---|
| Premature graying of hair | 49 | 6 | 55 |
| Halo nevi | 34 | 4 | 38 |
| Atopic dermatitis | 14 | 3 | 17 |
| Thyroid disease | 13 | 1 | 14 |
| Diabetes | 9 | 1 | 10 |
| Alopecia areata | 8 | 2 | 10 |
| Asthma | 6 | 0 | 6 |
| Tuberculosis | 3 | 0 | 3 |
| Urticaria | 2 | 1 | 3 |
| Joint pain | 1 | 0 | 1 |
| Psoriasis | 1 | 0 | 1 |
| Drug eruption | 2 | 0 | 2 |
| Total | 142 | 18 | 160 |

## DISCUSSIONS

Concerning the clinical classification of vitiligo, Ortonne et al[12]. reported that 90% of vitiligo patients had a generalized type, and the universal type which had many accompanying diseases was rare. In the present study, the incidence of vitiligo with generalized type was 50.2%. This was about the same with the results of 51.2% by Park et al[11]. Therefore, we think that the lower incidence of vitiligo with generalized type is a characteristic of vitiligo in Korea. In etiologic classification, El Mofty et al[19]. reported that the percentage of type B patients was only 5% of vitiligo patients, whereas Koga et al[17]. reported it as 27.9%. Park et al[11]. reported it as 9.5%. In the present study it was 15.4%.

Most of the vitiligo is acquired in relatively early age and the average age of onset is around the twenties. Lerner[20] reported that 50% of patients in the clinic had disease onset prior to the age of 20 years. Seghal[21] found that most patients in the clinics presented for evaluation by the age of 19 and Levai[22] found the average age of onset to be in the second decade. However, Howitz et al.[23] re-

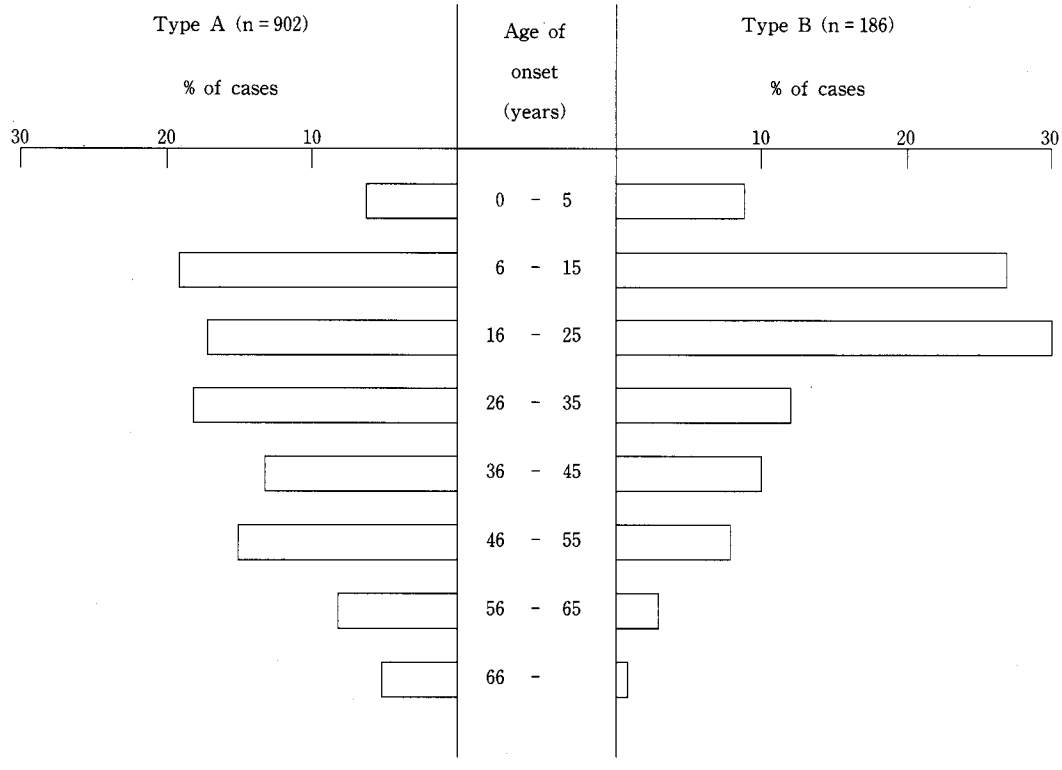


**Fig. 1.** Age of onset of type A and type B vitiligo.

ported atypical result that more than 50% of patients develop vitiligo after the age of forties. In our study based on data from out patient clinics more than 70% of patients had experienced vitiligo before the age of 35 years, and 57.5% before the age of 25 years, the peak onset was between the age of 6 and 15 years and the mean age of onset was 21.9% years. It could have resulted from the characteristics of the younger generation who are much concerned with their skin.

It has been known that the mean age of onset is younger in females than in males though Howitz et al.[23] reported no real difference between gender. Other reports[12,22] suggested that the earlier mean age of onset of vitiligo in females was due to their heightened concern at the cosmetic appearance. Slight female preponderence of the disease was observed in our study. However, our study and several other reports in Korea[5,10,11] showed contrary results that the age of onset was two years earlier in males.

The initial lesion of vitiligo has been known to occur mostly on the exposed area such as the face, neck and dorsum of hand, and appears as a single lesion[24]. In our study, 76.8% of them occurred as a single lesion and 71.1% occurred on the exposed area. 54.5% of the initial lesions occurred on the head, especially the forehead in detail. The neck was more fequently involve in female(24.4%) than in male(12.1%). Females may have more initial lesions of vitiligo at the neck because the neck of women is more frequently exposed to sunlight than in men. Considering the above data, sunlight may take a significant role in inducing the disease.

Various factors[12,20,25,27] such as physical trauma,

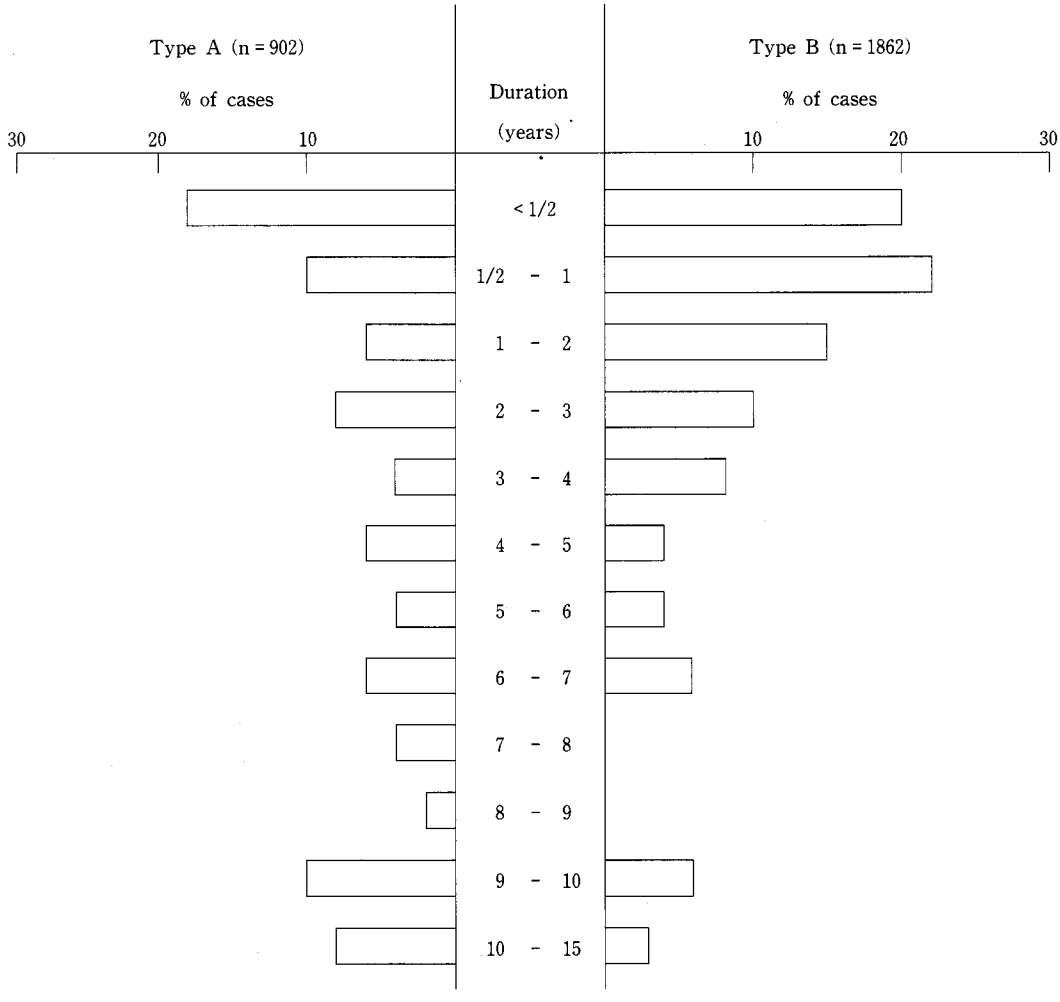


Fig. 2. Durations of the disease in type A and type B vitiligo

sunburn, emotional tension, inflammation, pregnancy and contraceptives are known to induce vitiligo. In the present study, 24.6% of patients had precipitating factors for vitiligo before its onset. This percentage was similar to Lerner's report[24]. However, this was higher than 5.2% in the report of Park et al.[11] and it is almost similar with 23.9% in the report of Park et al.[10] or 25% by Hann et al.[5] Physical trauma(44.1%), severe emotional tension or shock(26.2%), inflammatory disease such as folliculitis(5.6%), pregnancy and delivery(2.8%), and contraceptives(1.5%) acted as precipitating factors. Lerner[20] and Ortonne et al.[12] reported that vitiligo would occur after ultraviolet light exposure, and also in the present study, the lesions were induced by sunlight as well as sunburn.

The most favored treatment before the patient's visiting in our clinic was application of topical steroid, whereas Hann et al.[5] reported that application of topical oxsoralen with sunlight exposure was most common as a previous therapy. The difference may come from the fact that Hann et al.'s study[5] did not include localized vitiligo.

Poliosis is a disease frequently associated with vitiligo. Seghal[21] observed poliosis in 18 out of 202 cases, and Dutta et al.[25] reported that it occurred in 45% of vitiligo patients. In the present study, 27.0% of patients had poliosis. As for occupation, most male patients were students and most female patients were housewives. It seemed to be due to the fact that the peak age of onset and age of first visit of vitiligo were between 6 and 15 years and nearly all female patients older than this group were housewives in Korean society.

It was intended to find out whether farmers, fishermen, and laborers who are chronically exposed to sunlight had more incidence of vitiligo or not. However, as these people occupied only 2.4% of all cases, it seemed to be that chronic exposure to sunlight had nothing to do with the development or progression of vitiligo. But the fact that these kinds of people can not afford to visit the clinic because of their low income should be considered.

The prevalence of a positive family history of vitiligo varies from 6.25% to 38% in the literatures. In the present study, a family history was found in 12.2% of patients. This result was much less than 38% reported by Lerner[20], and similar to 7.4% by Park et al.[11] and 12% by Hann et al.[5] The low incidence of family history in Korea may result from the racial difference in inheritance pattern or a cultural tendency to hide the disease.

For the pathogenic mechanism of vitiligo, some hypotheses such as the autoimmune theory[14-16,26,28,29] neurohumoral theory[24] and self-destruction of melanocyte[30,31] have been discussed. The self-destruction theory is a hypothesis that vitiligo occurs when cells are destroyed by the excessive production or accumulation of phenol radicals in the process of forming melanin pigments in it, and the theory is supported by the phenomenon of discoloration similar to vitiligo on the exposed area of the employees in rubber or plastic factories who are apt to be exposed to phenol and catechol[30].

The neurohumoral theory is a hypothesis that the formation of melanocytes is restrained by a neuromediator secreted from the end of the nerve[24]. Koga[18] had reported that local injection of physostigmine revealed that dermatomally distributed vitiligo was associated with a dysfunction of the sympathetic nerves in the affected skin and that non-dermatomally distributed vitiligo was not. He also reported that type B improved with the monoamine oxidase inhibitor therapy such as oral nialamide.

The autoimmune theory has recently been discussed. About one third of vitiligo patients had discoloration of the retina and choroid membrane of eye[28] and many patients with autoimmune disease also had vitiligo.[15,16] The rate of accompanying autoimmune diseases such as the thyroid disease, diabetes in vitiligo was 10-15%, which was higher than in general prevalence[31], and also the frequency of antibodies such as antimelanocyte[4,16], antithyroid, antismooth muscle cell, antiparietal cell and antinuclear antibodies[29] was higher in vitiligo patients. Though vitiligo is likely to be an autoimmune disease, it is not certain whether vitiligo is a disease having one specific pathogenic mechanism or appears in the process of various other diseases[12].

Koga et al.[17] under their hypotheses that type A vitiligo is caused by an autoimmune mechanism and type B by the dysfunction of the sympathetic nerves, executed a clinical study with 481 patients with vitiligo. The results showed that the type A appeared in all age groups whereas the type B appeared mostly in young people before their thirties. In addition, type B was characterized by the arrest

of spreading after a rapid progression and the pattern of spread in type A was similar to that in autoimmune diseases, progressing continuously with alternate periods of remission and exacerbation. Also, they reported the presence of halo nevi[33] and Koebner's phenomenon which suggested the autoimmune mechanism appeared only in type A, and the association with diseases of proven or suggested allergic or immunological etiology such as tuberculosis, urticaria, drug eruption, asthma, and etc. appeared more often in type A. They explained that these findings supported their hypothesis that type A and B vitiligo had a different pathogenesis and autoimmune mechanism played a role only in type A.

In our study, a statistically significant difference in age distribution and duration between two groups was found. Onset was predominantly at an early age in type B, while type A could occur at any age. Type B vitiligo occurred over a relatively short period, whereas type A had a long disease duration like an autoimmune disease(Fig. 1 and 2). The investigated diseases which were proven or suggested to have an allergic or immunological etiology included premature graying of hair[32], halo nevi[33], atopic dermatitis, thyroid disease, diabetes, and etc.. These diseases were found more frequently in type A(Table 6). So the results of this present study supported the hypothese of Koga et al.[17] that the two types of vitiligo had different pathogenesis and autoimmune mechanisms were restricted to type A vitiligo.

As a conclusion, we investigated the clinical characteristics of vitiligo in Korea and showed that the type A vitiligo might have a different pathogenic mechanism with type B. However, there were some patients who had been affected by type A vitiligo and later transited to type B. They were disregarded in this study. Therefore, the possibility of the existence of some other pathogenic mechanism that could not be clarified by clinical researches could not be excluded. We think studies on such a possibility are required, and further research into treatment according to the differences of etiology, for example, systemic steroid or photochemotherapy for type A vitiligo and treatment affecting the nervous system such as nialamide for type B vitiligo, should also be considered in the future.

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