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Characteristics of hair loss in COVID-19 patients in Thailand

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Abstract

There is still a scarcity of data on hair loss caused by coronavirus disease 2019 (COVID-19) infection. This study aims to determine the characteristics of hair loss in Thai individuals after COVID-19 infection and to identify associated factors. From March to June 2022, a retrospective review of medical records and telephone interviews was conducted to determine the details of hair loss, the severity of infection, and the associated treatments of patients with an abrupt onset of hair loss after the diagnosis of COVID-19 infection at Siriraj Hospital in Bangkok, Thailand. This study included 43 patients who experienced hair loss within 4 months after COVID-19 infection. The mean age was 46.5 ± 14.5 years, predominantly women. Most had mild COVID-19 symptoms (59.3%), and 59.1% experienced weight loss, with a mean weight loss of 4.3 ± 2.0 kg per month. Preexisting hair loss was reported in 31.0% of participants, with approximately 3-quarters diagnosed with androgenetic alopecia. The median onset of hair loss after COVID-19 infection was 30 days (interquartile range 30–60). Telogen effluvium was the most common acute hair loss diagnosis, and topical minoxidil was the predominant treatment (95.3%). Female gender was correlated with a more severe shedding scale (adjusted odd ratio 24.76, 95% CI 1.67–168.86). Patients with a history of androgenetic alopecia tended to have a lower hair shedding scale (adjusted odd ratio 0.03, 95% CI 0.01–0.38). This study reviewed the characteristics of hair loss after COVID-19 infection during Omicron outbreaks in Thailand. The COVID-19-associated telogen effluvium, which is the primary cause in our patients, manifested with earlier onset at approximately 30 days.

Abbreviations: AA = alopecia areata, AGA = androgenetic alopecia, aOR = adjusted odd ratio, CATE = Coronavirus disease 2019 associated telogen effluvium, COVID-19 = coronavirus disease 2019, ICU = intensive care unit, IL = interleukin, IQR = interquartile range, TE = telogen effluvium.

Keywords: androgenetic alopecia, COVID-19, hair loss, telogen effluvium, vitamin D

1. Introduction

Coronavirus disease 2019 (COVID-19) is a worldwide pandemic disease. The disease was first found in Wuhan, Hubei Province, China.[1] Fever, cough, rhinorrhea, and dyspnea are the most common symptoms of COVID-19 infection; however, the patient may also present with pneumonia symptoms, which can cause respiratory failure and death.[2] In addition to respiratory symptoms, patients with COVID-19 can experience a variety of clinical manifestations during or after recovery from the disease. Dermatological involvement, including skin, hair, and nails, is one of the most prevalent presentations, which can be a new-onset dermatosis or an exacerbation of preexisting skin disease.[3]

The increased prevalence of hair loss during the COVID-19 pandemic was initially recognized by Dursun et al in 2020.[4] The first series of cases of telogen effluvium (TE) related to COVID-19 infection was published in November 2020.[5] In general, TE usually occurs approximately 3 months after an illness or any stressful event. It affects the hair cycle and causes the anagen, or growth phase, to become the telogen, or resting phase, resulting in diffuse synchronous hair shedding.

Alopecia areata (AA), both new-onset and aggravated preexisting AA, has also been reported following COVID-19 infection.[6] The disease has a significant impact not only on the image of the patient, but also on their confidence and social relationships. There is still a scarcity of information on hair loss after COVID-19 infection, particularly in Asians. More information about the disease can help us understand and treat it. The objective of this study was to determine the characteristics of hair loss in Thai individuals after COVID-19 infection and to identify any potential associated factors.

2. Methods

This is a retrospective cohort, single-center study approved by the Siriraj Institutional Review Board (IRB number

The authors have no conflicts of interest to disclose.

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Supplemental Digital Content is available for this article.

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317/2565(IRB3)). The inclusion criteria comprised: (1) patients aged 18 years or older who experienced increased hair shedding within 4 months of contracting COVID-19, and (2) confirmation of COVID-19 infection through a positive antigen test kit and/or polymerase chain reaction result. Medical records of patients who met the inclusion criteria between March 2022 and June 2022 at Siriraj Hospital, Bangkok, were reviewed and retrieved to collect demographic information, record clinical manifestations of hair loss, and obtain details about the COVID-19 infection. Furthermore, patients who had recently undergone major surgery, severe illnesses, or received chemotherapeutic agents were excluded. A telephone interview was conducted to collect missing variables, including COVID-19 vaccination history, COVID-19 symptoms, and stress levels (rated on a scale of 0–10). To minimize the potential for recall bias, each interview question was asked twice, using closed-ended questions with specific answer choices. Additionally, vaccination history was verified with documented information.

All data were collected using PASW Statistics, version 18.0 (SPSS Inc., Chicago, IL). Qualitative data were presented as numbers and percentages, while quantitative data were summarized as mean ± standard deviation, median and interquartile range (IQR). The Kruskal–Wallis test was used to compare the median ranks of non-normally distributed quantitative variables between the categories of qualitative multinomial variables. A P value < 0.05 was considered statistically significant.

3. Results

Forty-three individuals with a mean age of 46.5 ± 14.5 years were enrolled in this investigation. Seven patients (16.3%) were men, and 36 (83.7%) were women. Demographic information is shown in Table 1. All patients denied concurrent smoking.

Details of COVID-19 infection were collected from 27 patients, and this information is displayed in Table S1, Supplemental Digital Content, http://links.lww.com/MD/L29. The median duration of symptoms before confirmation by the test was 3 days. Most of the patients detected COVID-19 infection by polymerase chain reaction (77.8%). More than 90.0% of the patients had symptoms, with cough being the most commonly reported (70.4%). Only one patient developed cutaneous lesions during the illness, which presented as a maculopapular rash. We obtained weight loss data from 14 patients (51.9%) with mean weight loss of 4.3 ± 2.0 kg per month. Most of the patients in our study (59.3%) were rated as having mild symptoms. Only 1 participant was asymptomatic for COVID. He has the only risk of contact with a positive patient and was later tested a positive with an antigen test kit in our study. Following diagnosis, most of the patients in our investigation (48.1%) were hospitalized with the median length of stay was 10 days (IQR 7–14). The median stress level during COVID-19 infection was 8 (IQR 5–10). The most widely used drug among our patients was favipiravir (85.2%), followed by systemic steroids (25.9%). Before developing COVID-19, 21 subjects had received at least 1 dose of the vaccine. Of these, 16 patients (76.2%) received 2 doses of the COVID-19 vaccine, which was mainly adenovirus vector vaccines.

As demonstrated in Table S2, Supplemental Digital Content, http://links.lww.com/MD/L30, 30.2% of the patients in our research reported having preexisting hair loss prior to contracting COVID-19. Of these, androgenetic alopecia (AGA) was the most frequently identified condition (76.9%) and topical minoxidil was the most prescribed medication (69.2%) among them.

Table 2 shows the characteristics of post-COVID-19 hair loss. The median onset of hair loss after COVID-19 diagnosis was 30 days (IQR 30–60). The mean hair count was 160 ± 82.4 hairs per day, while the mean hair shedding scale was 3.4 ± 0.8. Diffuse hair loss made up 95.3% of the reported hair loss patterns. Additionally, 37.2% of the patients had a positive hair pull test found during the first outpatient department visit. A normal scalp condition was present in 93.0% of study subjects. Clinical signs of seborrheic dermatitis were seen in only 3 cases. Further laboratory tests were conducted in some patients and most of the results were normal. These tests included complete blood counts, ferritin levels, thyroid function tests, antinuclear antibody tests, and vitamin D levels. It is interesting to note that all patients who underwent a vitamin D level blood test were found to have vitamin D deficiency or insufficiency. Based on the clinical presentation as patchy alopecia and dermoscopic findings, 2 individuals were diagnosed with AA, which was less prevalent than TE (95.3%) in terms of acute hair loss associated with COVID-19 infection (Fig. 1). Topical minoxidil was the most frequently used treatment for hair loss after COVID-19 (95.3%) (Fig. 2).

We discovered that patients who were women and those with a history of weight reduction had higher proportions of shedding scales (adjusted odd ratio, aOR 24.76, 95% CI 1.67–168.86 and aOR 2.28, 95% CI 0.25–20.72, respectively) (Table 3). Patients who had preexisting AGA had a lower hair shedding scale than other types of preexisting hair loss (aOR 0.03, 95% CI 0.01–0.38). Other variables, including underlying diseases, stress levels, the severity of COVID-19 infection, and abnormal blood tests results, did not show a significant correlation with the hair shedding scale.

Table 1

Demographic data of the patients (N = 43).

	N (%)
Sex	
Female	36 (83.7)
Male	7 (16.3)
Age at diagnosis (yr), mean ± SD	46.5 ± 14.5
Underlying disease	
No	24 (55.8)
Hypertension	12 (27.9)
Diabetes mellitus	11 (25.6)
Dyslipidemia	9 (20.9)
Depression	2 (4.7)
Allergy	2 (4.7)
Anemia	2 (4.7)
Current medications	
No	28 (65.1)
Anti-hypertensive drug	10 (23.3)
Diabetes medication	10 (23.3)
Lipid lowering agents	8 (18.6)
Antidepressants	2 (4.7)
NSAIDs	1 (2.3)

NSAIDs = non-steroidal anti-inflammatory drugs, SD = standard deviation.

4. Discussion

Fever and respiratory symptoms are the most typical clinical manifestations of COVID-19 infection. However, post-COVID-19 syndrome can be characterized by several clinical manifestations that persist or emerge several months after the infection. Regarding dermatologic involvement, including skin, hair, and nail abnormalities, acute hair loss is one of the most common complaints of patients related to COVID-19 infection.[7] In our study, acute hair loss after COVID-19 infection was predominantly presented in women, consistent with data on the incidence of COVID-19 infection. Similar to the review by Hussain et al,[8] approximately half of COVID-19-associated TE (CATE) patients had comorbid conditions, including hypertension, diabetes mellitus, and dyslipidemia. Those with metabolic syndrome, cardiovascular disease, and obesity are more likely to develop COVID-19 than healthy individuals, as is well known. In our study, even with the higher rate of hospitalized patients, most of them (59.3%) had only mild symptoms. Due to strict measures and regulations in Thailand at the time of the pandemic, all confirmed patients were required to be confined to hospitals or COVID-19 treatment facilities. The most commonly prescribed medication was favipiravir.

Ten participants had previously been diagnosed with AGA, and most of them had mild symptoms of COVID-19. Previous studies have reported that AGA was not correlated with the severity of COVID-19.[9,10] Surprisingly, individuals in this group had a lower hair shedding scale than those with another type of preexisting hair loss (aOR 0.03, 95% CI 0.01–0.38). This could be explained by the fact that most people with AGA have already undergone medical therapy for hair loss. Omer Kutlu et al reported an association between the severity of AGA and recurrent COVID-19 infection. The recurrence rate of COVID-19 in men with AGA classified as grade 3 or higher according to the Hamilton classification was higher compared to those with AGA grades 1 to 3 or no AGA (7.3% vs 2.6%, respectively) with an odds ratio of 2.93 (95% CI 1.22–7.03).[10] Unfortunately we did not add this information in our study.

Table 2

Characteristics of acute hair loss after COVID-19 infection (N = 43).

	N (%)
Onset of hair loss after COVID-19 diagnosis (d), median (IQR) (N = 41)	30 (30–60)
Hair count (per day), mean ± SD (N = 30)	160 ± 82.4
Hair shedding scale (1–6), mean ± SD (N = 30)	3.4 ± 0.8
Pattern of hair loss	
Diffuse hair loss	41 (95.3)
Patchy hair loss	2 (4.7)
Hair pull test	
Negative	27 (62.8)
Positive	16 (37.2)
Scalp condition	
Normal	40 (93.0)
Scaling	2 (4.7)
Erythema	1 (2.3)
Abnormal laboratory investigation	
Anemia (N = 10)	1 (10.0)
Abnormal TSH (N = 10)	0 (0.0)
Positive ANA (N = 2)	0 (0.0)
Low ferritin level (N = 13)	1 (7.7)
Vitamin D deficiency/insufficiency (N = 10)	10 (100.0)

ANA = antinuclear antibody, COVID-19 = coronavirus disease 2019, IQR = interquartile range, SD = standard deviation, TSH = thyroid stimulating hormone.

The median onset of hair shedding after recovery from COVID-19 infection in our study was 30 days (IQR 30–60). Similarly, Omer Kutlu et al[10] reported mean onset of hair loss of 5.43 ± 4.10 weeks. Compared to previous studies, which showed an average onset of 53 to 67 days, patients with CATE in our study had an earlier clinical presentation of hair shedding[6,8,9,11] and earlier than the classic acute telogen effluvium that usually manifested approximately 2 to 3 months after stressful events.[9] In this study, the mean hair shedding scale was 3.4 ± 0.8, which was lower than those of the previous multicentric study, which revealed an average scale of 4.3 ± 1.5.[12] This could be explained by the length of time we spent collecting data on the Omicron variant, discovered in Thailand in early January 2022, and is known to be less severe than the Delta variant.[13] Even there have been reports of medications causing hair loss during COVID-19 pandemic.[14] In this study, patients' current medications were also reviewed but the prescription period was more than 4 months before the onset of hair loss. It suggested that those medications were unlikely to be the concomitant cause of hair loss in our patients.

Moreover, all patients revealed low vitamin D levels, while thyroid stimulating hormone, ferritin, antinuclear antibody, and complete blood count were normal in most of the cases. Similarly, a study from Iran demonstrated that vitamin D deficiency was the most common abnormal laboratory finding in patients diagnosed with CATE.[15] However, that study also found iron deficiency anemia and hypothyroidism in 21.0% and 13.8% of the patients, respectively.

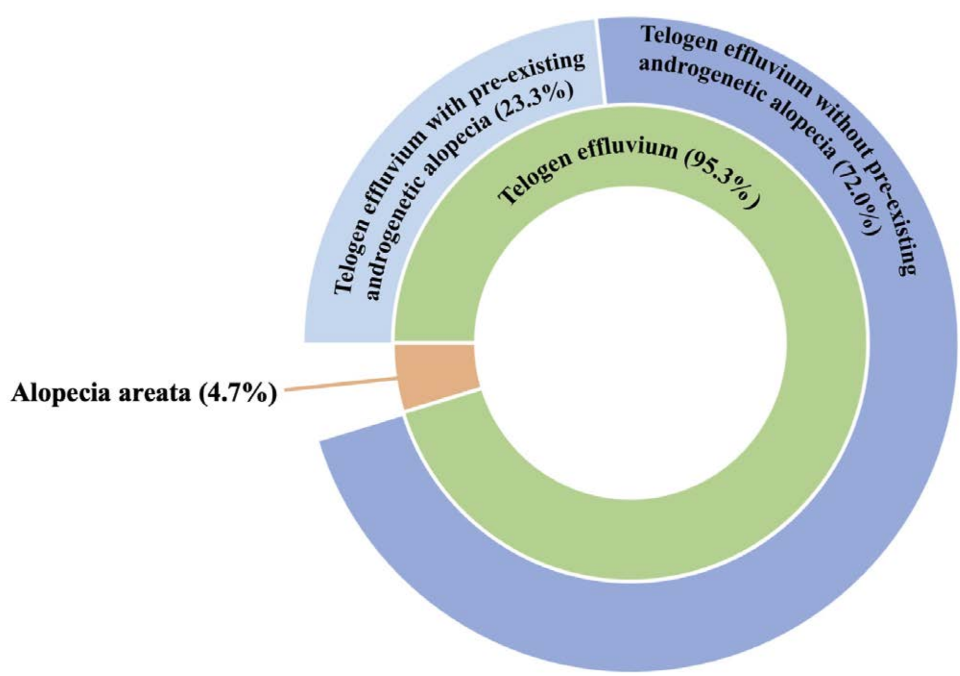

Figure 1. Types of acute hair loss following COVID-19 infection. COVID-19 = coronavirus disease 2019.

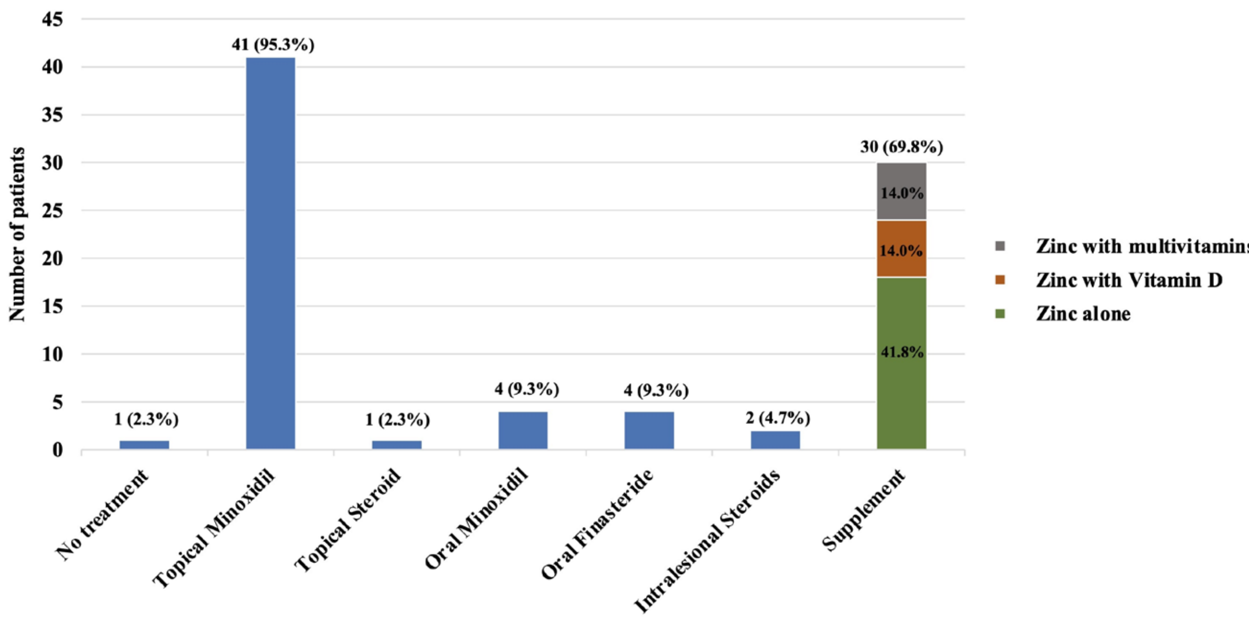

Figure 2. Treatments of acute hair loss following COVID-19 infection. COVID-19 = coronavirus disease 2019.

Table 3

Factors affecting the hair shedding scale (N = 30).

	Hair shedding scale 1–3 N (%) (N = 12)	Hair shedding scale 4–6 N (%) (N = 18)	Univariable analysis		Multiple analysis	
			Crude OR (95% CI)	*P* value	Adjusted OR (95% CI)†	*P* value
Age at diagnosis (y), mean ± SD	44.1 ± 14.2	48.9 ± 14.9	1.03 (0.97–1.08)	0.42	–	–
Onset of hair loss after COVID-19 diagnosis (d), median (IQR) (N = 29)	30.0 (30.0–60.0)	30.0 (30.0–60.0)	1.00 (0.97–1.03)	0.94	–	–
Sex						
Female	7 (58.3)	17 (94.4)	12.14 (1.19–123.62)	0.03*	19.98 (1.27–315.56)	0.03*
Male	5 (41.7)	1 (5.6)	1.00		1.00	
Underlying diseases						
Hypertension	3 (25.0)	7 (38.9)	1.91 (0.38–9.59)	0.69	–	–
Diabetes mellitus	2 (16.7)	6 (33.3)	2.50 (0.41–15.23)	0.42	–	–
Dyslipidemia	2 (16.7)	4 (22.2)	1.43 (0.22–9.38)	1.00	–	–
Depression	1 (8.3)	1 (5.6)	0.65 (0.04–11.45)	1.00	–	–
Allergy	0 (0.0)	1 (5.6)	0.59 (0.43–0.80)	1.00	–	–
Preexisting hair loss						
Androgenetic alopecia	7 (58.3)	1 (5.6)	0.04 (0.01–0.43)	<0.01*	0.03 (0.01–0.38)	<0.01*
Telogen effluvium	0 (0.0)	1 (5.6)	0.59 (0.43–0.80)	1.00	–	–
Alopecia areata	0 (0.0)	1 (5.6)	0.59 (0.43–0.80)	1.00	–	–
Weight loss						
Weight loss	3 (25.0)	11 (61.1)	4.71 (0.94–23.68)	0.05	2.28 (0.25–20.72)	0.47
Stress Level						
Stress level (score 0–10), median (IQR) (N = 27)	8.0 (5.0–8.0)	8.0 (1.25–10.0)	0.99 (0.79–1.24)	0.86	–	–
Severity of COVID-19 (N = 29)						
Asymptomatic and mild	9 (75.0)	10 (58.8)	1.00	0.45	–	–
Moderate and severe	3 (25.0)	7 (41.2)	2.10 (0.41–10.66)		–	
Abnormal laboratory investigation						
Ferritin (N = 7)	0 (0.0)	1 (16.7)	0.83 (0.58–1.19)	1.00	–	–
Vitamin D (N = 7)						
Vitamin D insufficiency	1 (100.0)	2 (33.3)	0.66 (0.30–1.48)	0.43	–	–
Vitamin D deficiency	0 (0.0)	4 (66.7)	1.00		–	

CI = confidence interval, COVID-19 = coronavirus disease 2019, IQR = interquartile range, OR = odds ratio, SD = standard deviation.
* Statistically significant at *P* value <.05.
†Adjusted with age, sex, preexisting androgenetic alopecia, and weight loss.

Several pathophysiological pathways have been proposed that may lead to COVID-19-associated hair loss. Previous research stated that when the COVID virus enters the host, the spike receptor binding domain binds to the angiotensin-converting enzyme 2 receptor in the lungs and many tissues, then induces immune responses by recruiting T lymphocytes, monocytes, and neutrophils, and finally results in the release of many cytokines such as tumor necrosis factor-α, interleukin-1 (IL-1), IL-6, IL-1, IL-6, IL-1β, IL-8, IL-12, and interferon-λ or commonly referred to as a "cytokine storm."[16] Moreover, it was hypothesized that the virus caused direct injury to the dermal follicular epithelium by engaging the ACE-2 receptor, which was also found to be expressed in the basal layer of the hair follicle.[1,17] The COVID virus has been proposed to stimulate antibody-dependent enhancement through interactions with fragmented crystallizable and/or complement receptors, similar to the theory of dengue infection. In vitro dengue virus activation of the caspase cascade, decreased bone morphogenetic protein-4 levels, and down-regulation of the noggin can cause dysregulation of hair growth, resulting in the premature catagen and increased hair shedding.[17,18] Many cytokines from cytokine storms will affect the early-dividing keratinocytes and dermal papilla cells. Tumor necrosis factor-α, IL-6, and interferon-λ inhibit hair follicle growth in vitro.[17,18] The stress from illness, socioeconomic situation, and quarantine time are associated with hair loss.[19] Stress increases stimulation of pro-inflammatory cytokines and drives epithelial and mesenchymal cells to apoptosis, causing the premature termination of hair growth.[18] The median stress level in our study was 8 (IQR 5–10), which is relatively high. It may explain hair loss, but it was not correlated with the severity of hair loss in our study ($P = 0.86$).

AA after COVID-19 infection was first reported in July 2020.[20] After a while, there was an increase in the number of case reports and publications describing non-scarring patchy alopecia linked to COVID-19 that showed exclamation mark hairs and a black dot on dermoscopy.[21] After a viral infection, such as COVID-19, AA can appear as either a new onset of hair loss or as an exacerbation in patients with preexisting AA.[6] A review study shows AA is more often in asymptomatic COVID-19 patients while CATE is found in symptomatic and asymptomatic patients.[22] In our study, we discovered 2 patients diagnosed with AA after COVID-19 infection, one of whom had previously been diagnosed with AA. All our AA patients were symptomatic (mild and moderate COVID-19 infection severity respectively). COVID-19 can regulate interferon and may induce a cytokine storm. This leads to increased levels of IL-6, which subsequently induced the expression of the class I major histocompatibility complex in the proximal outer root sheath, and the consequent loss of the immune privilege of the hair follicle led to the activation of T cells, which is valuable for the pathogenesis of AA.[23]

The limitations of the study included its retrospective cohort design conducted at a single center, which may have introduced selection bias due to the non-randomized inclusion of participants. Additionally, telephone interviews were utilized for data collection, potentially introducing recall bias. However, closed-end questions were employed during the telephone interviews and repeated twice to reduce the risk of recall bias. To enhance the study's statistical power, increasing the sample size may be beneficial in future study. Since every patient who underwent vitamin D level testing in this study exhibited vitamin D deficiency or insufficiency, further research on the association between vitamin D and hair loss following COVID-19 is recommended.

Our findings also provided the clinical implications and guidance for patient counseling. Patients with AGA should be advised to maintain their medication regimen during COVID-19 infection because our study revealed that patients with preexisting AGA reported less hair shedding scale. It could be explained by all of our patients were treated AGA. Additionally, nutritional support should be considered during a COVID-19 infection to prevent weight loss, as increased weight loss tended to be associated with more hair shedding.

In conclusion, our findings show that CATE in Thais during the Omicron outbreaks has earlier onset but less severe hair shedding. Further data analysis may provide answers to remaining questions about the risk of hair loss following COVID infection. We wish to be able to inform our patients about the nature of this new condition and help to reassure patients who are experiencing psychosocial problems related to hair loss.

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