Review Article

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Risk factors and comorbidities associated with central centrifugal cicatricial alopecia

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ABSTRACT

Central centrifugal cicatricial alopecia (CCCA) is the most common form of scarring alopecia that most often affects Black women. The disease typically begins with hair loss in the center scalp, which progresses in a centripetal fashion. Both environmental insult and genetics have been implicated in CCCA etiology, although the exact pathophysiology remains unknown. Nevertheless, it is important that providers feel comfortable educating their patients on risk factors (RFs) for the development or worsening of CCCA, and potential comorbidities associated with the condition. Thus, the goal of this review was to summarize these factors. A comprehensive literature search was performed, and studies were included if they reported research on RFs for or comorbidities associated with, CCCA. A total of 15 studies were included: *n* = 5 researching RFs for CCCA and *n* = 10 researching comorbidities associated with CCCA. There was an association suggesting an increased risk of CCCA with traction hairstyles in *n* = 2/3 studies, previous pregnancies in *n* = 1/1 studies, and use of chemical hair relaxers in *n* = 1/3 studies. Additionally, age and total years of hair loss were associated with increased CCCA severity in *n* = 2/2 studies. Type 2 diabetes was positively associated with CCCA in *n* = 3/5 studies, uterine leiomyomas in *n* = 1/2 studies, hyperlipidemia in *n* = 1/2 studies, and vitamin D deficiency in *n* = 1/1 studies. Conflicting results regarding RFs and comorbidities associated with CCCA exist within the literature. Thus, further investigation in larger cohorts must be done, and future research into genes implicated in CCCA and their potential role in the development of other diseases is recommended.

Keywords: alopecia, central centrifugal cicatricial alopecia, hair loss

Introduction

Central centrifugal cicatricial alopecia (CCCA) is the most common form of scarring alopecia, and most often affects Black women between the ages of 30 and 55. In fact, the reported prevalence of CCCA ranges from 2.7% to 5.6% of Black women.[1] The disease course typically begins with hair loss in the center of the scalp that progresses outward from the center point in a centrifugal fashion.[1] The disease is characterized by permanent hair loss with signs and symptoms including itching, burning, flaking, or development of acneiform lesions on the scalp.[2]

CCCA can have a profound impact on the quality of life for Black women. For example, a study published in the British Journal of Dermatology investigating a group of Black individuals that were 98% women, reported that the mean quality of life index (QLI) in those with CCCA was 53.3%. These authors considered any QLI > 50 to be a significant impairment, as validated by a previous study. They noted that in particular, subjective symptoms of CCCA (eg, I am sad about the appearance of my hair), had a QLI of 77.02, while objective symptoms of CCCA (eg, my scalp is visible) had a QLI of 60.69.[3] Another study, with all subjects being Black women with CCCA, found that the top 5 factors reported as most important for these women when seeking CCCA care included (1) physician experience with black hair/CCCA, (2) patient hairstyling practices, (3) physician ethnicity, (4) availability of effective treatments, and (5) treatment cost. They also found that the majority of women strongly agreed with the following statements: "hair is a major factor in female appearance," "my hair loss bothers me," "I feel embarrassed, self-conscious, or frustrated about my hair loss," "I believe hair forms a major expression of oneself," "I feel anxious or worried about my hair loss," "I feel less attractive because of my hair loss," and "I feel less confident because of my hair loss."[4]

What is known about this subject in regard to women and their families?

- It is understood that central centrifugal cicatricial alopecia most commonly occurs in Black women and can often be genetically linked.

What is new from this article as messages for women and their families?

- This article provides a novel overview of the possible risk factors associated with developing central centrifugal cicatricial alopecia and possible comorbidities associated with the condition.

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CCCA diagnosis is often made clinically.[5] Biopsy showing perifollicular fibrosis, lymphocytic infiltrates, and reduced follicular concentrations supports the diagnosis of CCCA; however, histopathology may mimic other hair loss disorders (eg, lichen planopilaris).[6] The exact pathophysiology of CCCA remains unknown. One proposed mechanism of disease follows that CCCA occurs when there is an environmental insult to the scalp resulting in a primary immune response that leads to an autoimmune reaction involving the peroxisome proliferator-activated receptor gamma and transforming growth factor (TGF)-beta cascades.[7] Additionally, recent research suggests genetics may contribute to CCCA development. Specifically, mutations in the *PADI3* gene encoding proteins for hair shaft development were linked to CCCA development.[1] Although the mechanism of the disease is unclear, early intervention and treatment of CCCA may prevent permanent hair loss. Current treatment protocols involve the use of topical steroids or steroid injections at sites of hair loss, while severe cases that may overlap with other inflammatory hair disorders (eg, lichen planopilaris) are often treated with immunosuppressant medications.[5]

Much research is still needed to inform clinicians about proper CCCA management. Current understanding of disease pathogenesis primarily hypothesizes that inflammatory destruction of hair follicles is the primary driver of disease progression. However, this hypothesis does not explain the later stages of disease that are often characterized by fibrosis without significant inflammatory cells. Additionally, providers must be aware of the risk factors (RFs) for the development or worsening of CCCA to help guide their diagnosis of the condition. It is also important that providers are aware of potential comorbidities associated with the condition to improve patient outcomes. Thus, both RFs and associated comorbidities should be relayed from providers to their patients. Limited work has been done to summarize these education points for clinicians beyond the CCCA associations shown with type 2 diabetes and uterine leiomyomas.[8] Thus, the goal of this review was to summarize the RFs and comorbidities associated with CCCA to provide further education to providers, further inform etiopathogenesis, and propose further treatment algorithms.

Methods

A comprehensive literature search was performed using PubMed, Embase, and Web of Science. The preferred reporting items for systematic reviews and meta-analysis were used to guide methodology as shown in Figure 1. The databases were searched using the term "central centrifugal cicatricial alopecia." Studies were included if they reported on RFs or comorbidities associated with CCCA. No geographic or language restrictions were employed. An initial title and abstract screen were performed by 2 independent researchers (MG and AF). The remaining articles then went through full text review. Data were extracted from the remaining articles by 2 independent researchers (MG and AF) with any discrepancies settled by a third researcher (MV).

Results

Fifteen studies were included: 5 researching RFs for CCCA or the development of severe CCCA and 10 researching potential comorbidities associated with CCCA. Any comorbidities shown to be associated with CCCA across studies are summarized in Table 1.

Risk factors

Environmental and medical RFs for developing CCCA were researched by Kyei et al.[9] They had 326 Black women complete a RF questionnaire at churches and health fairs in Cleveland, Ohio. This survey was followed by a scalp examination by dermatologists using the scalp alopecia photographic scale. A total of 52/326 women had scores of 3–5, which represented probable CCCA (17%). Results showed significantly higher rates of type 2 diabetes mellitus (T2DM) in those with CCCA compared to the 224/326 women (72%) with scores of 0–1, representing no CCCA (P = .01). In addition, the prevalence of a bacterial scalp infection or traction hairstyles were significantly higher in those participants with CCCA compared to those without (P = .045, P = .02).

Narasimman et al.[10] used a retrospective cohort design to research RFs for development of CCCA. A total of 74 Black women over 18 years of age with CCCA were matched to 96 controls of Black women over 18 years of age with different hair loss conditions. Results showed higher rates of previous pregnancies at the time of diagnosis in women with CCCA (n = 23/74, 31%) compared to controls (n = 5/96, 5%) (OR = 11.71; $P \leq .001$). Additionally, women with CCCA were more likely to have used chemical hair relaxers (n = 63/74, 85%) than controls (n = 39/96, 41%) (OR = 12.37; $P \leq .001$).

Hair grooming practices and their associated risk of CCCA were researched by Gathers et al.[11]. They retrospectively identified 118 Black women with a clinical and histological diagnosis of CCCA and compared them to a control group of 312 Black women without a history of hair loss. The Hair Grooming Assessment Survey was sent via mail to all women in the study. A total of 50 cases and 46 control responses were included in the analysis. Results showed that women with CCCA were more likely to have worn cornrows/braids with artificial hair compared to controls (OR = 1.6; P = .03). Additionally, women with CCCA were more likely to have worn sewn-in weaves (OR = 2.8; P = .04) or a texturizer (P = .033). Finally, women with CCCA were more likely to report damage, tenderness, or pulling from the use of cornrows or weaves. There was no correlation seen between the use hair relaxers, hot combs, or scalp burns and CCCA.

Khumalo and Gumedze[12] conducted a retrospective review of a total of 561 Black women, with 16 being diagnosed with CCCA. When dividing prevalence by age, the risk of CCCA was shown to be higher in older than younger women (odds ratio [OR] = 5.79; 95% confidence interval [CI] [1.63–20.64]; P = .007). However, these authors found no significant association with traction symptoms, such as tightness or painful hairstyling, or the use of relaxers and CCCA.

Suchonwanit et al.[13] researched which RFs correlated with increasing severity of CCCA. A total of 38 Black women with biopsy-proven CCCA were included. All women responded to a questionnaire and the severity of their CCCA was graded from 1 to 5 by a dermatologist using the photographic scale of CCCA. A amount of 22 of 38 women were early stage (defined as a score of 1–2) (57.9%), and 16/38 were late-stage (defined as a score of 3–5) (42.1%). Results showed a positive correlation between mean number of years of hair loss and severity of CCCA (P = .003). However, there was no association between family history of hair loss, use of any hair care product type, or history of any medical condition with CCCA severity.

Comorbidities

Roche et al.[14] analyzed rates of T2DM in Black women with CCCA compared to those without. A total of 395 women with CCCA were included; 181 of these women had a hemoglobin A1C (HgA1C) test, with 105/181 having T2DM (58%). The control group consisted of 38,885 women without CCCA, with 16,454/38,885 having a HgA1C test. Of those, 7,002/16,454 had a diagnosis of T2DM (43%). After controlling for obesity, considered a body mass index (BMI) greater than or equal to 30, women with CCCA had significantly higher odds of having T2DM compared to controls (OR = 4.13; 95% CI [2.76–6.18]; $P < .05$).

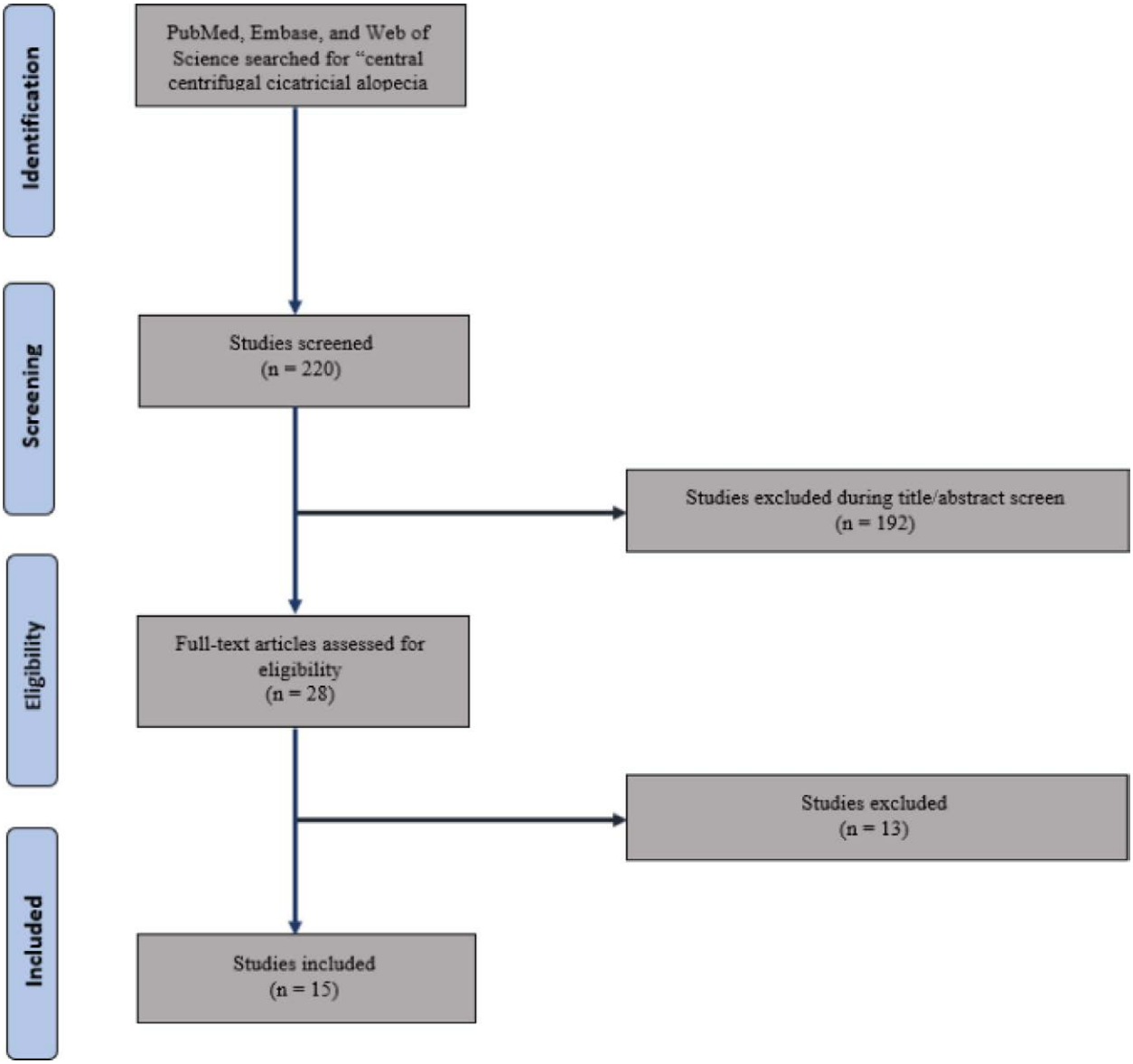

Fig. 1. The preferred reporting items for systematic reviews and meta-analysis (PRISMA) guidelines utilized in the methodology.

Ali et al.[15] researched if an association existed between HgA1C levels in prediabetic and diabetic patients with CCCA. Any Black patient who had had both a HgA1C level and a CCCA clinical photograph taken within 3 months was included. The severity of CCCA was graded through the pictures using the CCCA photographic severity scale. A total of 35 patients with prediabetes/T2DM with CCCA and 15 controls without prediabetes/T2DM with CCCA were included. Results showed a significantly higher CCCA severity score of 3.16 in the experimental group compared to 2.57 in controls (P = .043).

Coogan et al.[16] also reviewed the association between CCCA and T2DM. They created a supplemental questionnaire for the Black Women's Health Study asking about hair loss. Respondents rated the severity of their hair loss based on photographic scales created by the Duke Hair Disorders clinic. A total of 6,162 women responded to the hair loss survey and were compared to 9,641 women who responded to a 2015 Black Women's Health Study survey. A amount of 150 of 6,162 women had physician-diagnosed or biopsy-proven CCCA. Results showed that women with CCCA had a hazard ratio of 2.24 for having T2DM compared to controls (95% CI [1.35–3.71]).

Dina et al.[17] reviewed medical records of Black women 18 years or older and determined which women had diagnoses of uterine fibroids or CCCA. Results showed that 13.9% of patients with CCCA had uterine fibroids (n = 62/447) compared to 3.3% of patients without CCCA (n = 16,212/486,657), (OR = 4.68; 95% CI [3.57–6.12]).

Samrao et al.[18] researched if CCCA was associated with an increased risk of a wide range of fibroproliferative disorders, including interstitial lung disease, atherosclerosis of the aorta, nonalcoholic steatohepatitis, end-stage renal disease, and uterine fibromas. All Black women with CCCA were identified via ICD-10 codes, and only women with biopsy-proven CCCA were included. A total of 427 patients with biopsy-proven CCCA were included and matched to 1,281 controls based on age, sex, and ethnicity. Results showed no increased risk for any of the fibroproliferative disorders studied.

Brown-Korsah et al.[19] looked at the risk of colorectal and breast cancers in patients with CCCA. All Black women 18 years or older with a biopsy-proven diagnosis of CCCA were included in the retrospective chart review. Results showed that 159/742 women with CCCA had a biopsy to support diagnosis, and of these women, 7/159 had breast cancer (4.4%). This was a significantly higher rate when compared to the 4,079 controls (1.8%) (OR = 2.49; 95% CI [1.06–4.92]; P = .02). There was no significant difference in colorectal cancer rates between women with CCCA and controls.

Table 1

Comorbidities associated with CCCA

Comorbidity	Number of studies showing positive association	Results
Type 2 diabetes mellitus	3/5	1. Significantly higher odds of T2DM in patients with CCCA (OR = 4.13; 95% CI [2.76–6.18]; P < .05)[14] 2. Significantly higher CCCA severity score in patients with T2DM compared to controls (P = .043)[15] 3. Significantly higher risk for having T2DM in patients with CCCA (HR = 2.24; 95% CI [1.35–3.71])[16] No significant association between DM2 and CCCA[20,21]
Uterine fibroids	1/2	1. Significantly higher odds of UF in patients with CCCA (OR = 4.68; 95% CI [3.57–6.12])[17] No significant association between UF and CCCA[18]
Hyperlipidemia	1/2	1. Significantly higher rates of hyperlipidemia in patients with CCCA (P ≤ .0001)[20] No significant association between hyperlipidemia and CCCA[21]
Vitamin D insufficiency/ deficiency	1/1	1. Significantly higher risk for vitamin D insufficiency/deficiency in patients with CCCA (OR = 5.43; 95% CI [–5.43–177.63]; P = .0018)[23]

CCCA, central centrifugal cicatricial alopecia; CI, confidence interval; HR, hazard ratio; OR, odds ratio; T2DM, type 2 diabetes mellitus; UF, uterine fibroids.

Leung et al.[20] analyzed comorbidities associated with CCCA by comparing 53 Black women with CCCA ages 28–62 to 212 Black women without CCCA from the Dallas Heart Study. Hyperlipidemia was seen at significantly higher rates in 35/53 patients with CCCA (66%), compared to 35/212 patients from the Dallas Heart Study ($P \leq .0001$). There was no statistical difference between rates of hypertension, obesity, and T2DM between groups. In contrast, Jafari et al.[21] matched 153 Black women with CCCA to 153 controls with nonscarring alopecia based on age, sex, ethnicity, and insurance status. They showed no increased risk of comorbidities between patients with CCCA and controls with nonscarring alopecia: hypertension ($P = .759$), obesity ($P = 1.000$), peripheral artery disease ($P = .759$), dyslipidemia ($P = .819$), T2DM ($P = .647$), hyperparathyroidism ($P = 1.000$), adrenal nodule ($P = 1.000$), HIV ($P = 1.000$), hidradenitis suppurativa ($P = 1.000$), psychiatric diseases ($P = .142$), or seborrheic dermatitis ($P = .884$).

McKenzie et al.[22] compared rates of anxiety or depression between Black women with CCCA to Black women with psoriasis or alopecia areata. They used a cross-sectional study design and pulled records of all Black women 18 years or older seen at Perelman School of Medicine between July 2017 and July 2019. Results showed no significant difference in rates of depression or anxiety in women with CCCA ($n = 27/270$, 10%), psoriasis (10/84, 11.8%), and alopecia areata ($n = 7/69$, 10.1%). Overall, there was no increased risk for anxiety or depression in CCCA compared to psoriasis ($P = .68$) and alopecia areata ($P = .84$).

Finally, the risk of vitamin D deficiency/insufficiency in Black women with CCCA was researched by Collins et al.[23]. They performed a retrospective review of 54 Black women with CCCA, 27 of whom had a documented vitamin D level. Authors defined deficiency as vitamin D level less than 20 ng/mL and insufficiency as 21–29 ng/mL. Results showed that women with CCCA had a much higher risk of vitamin D deficiency/insufficiency than those without (OR = 5.43; 95% CI [5.43–177.63]; $P = .0018$).

Discussion

A total of 15 studies were included: $n = 5$ researching RFs for CCCA and $n = 10$ researching comorbidities associated with CCCA. Across studies analyzing RFs ($n = 5$), results were mixed on significant positive associations with CCCA. There was an association suggesting an increased risk of CCCA with traction hairstyles in $n = 2/3$ studies, previous pregnancies in $n = 1/1$ studies, and use of chemical hair relaxers in $n = 1/3$ studies. Additionally, age and total years of hair loss were associated with increased CCCA severity in $n = 2/2$ studies. Mixed results were also shown across studies analyzing CCCA's association with comorbidities ($n = 10$). Type 2 diabetes was positively associated with CCCA in $n = 3/5$ studies, uterine fibroids in $n = 1/2$ studies, hyperlipidemia in $n = 1/2$ studies, and vitamin D deficiency in $n = 1/1$ studies. These mixed results suggest the need for additional research in larger cohorts to properly inform clinicians of the RFs and comorbidities, so they can educate their patients on these associations.

Of the included studies, all investigated Black women. This heightened prevalence among this population suggests that genetic factors play an important role in CCCA development. Familial inheritance of the disease in an autosomal dominant pattern has been described in the literature.[24] At the molecular level, CCCA development has been associated with mutations in the *PADI3* gene; the enzyme encoded by this gene works to modify proteins in the hair follicle to promote proper follicular development.[1] Additionally, increased expression of fibroproliferative genes has been shown to occur in areas of the scalp affected by CCCA.[25] It has been shown that fibroproliferative disorders occur at higher rates in African American populations, possibly due to the historically protective effects of fibrotic alleles against helminth infections in Africa.[26] Thus, the genetic predisposition to CCCA among families may be linked to the inheritance of profibrotic genes and may explain associations with conditions such as uterine fibroids. Keeping this in mind, physicians may consider screening patients with CCCA for menorrhagia, infertility, and other common manifestations of uterine fibroids.

Current treatment for CCCA focuses on the use of anti-inflammatory medications to halt disease progression. However, although initial inflammation occurs in CCCA, persistent and worsening fibrosis is often present without inflammatory signs later in disease progression.[25] For many patients who do not show improvement with these anti-inflammatory treatment regimens, the potential use of antifibrotic agents in the future may be beneficial. Therapies such as Interleukin-13 (IL-13) blockers have shown to have potential benefit in the progression of idiopathic pulmonary fibrosis, and medications such as losartan and statins have shown potential for halting CCCA progression.[26,27] Given the continued debate among the true pathogenesis of CCCA, it is recommended that first-line therapy prioritize tension-free hairstyles, antidandruff shampoos, and topical steroid/steroid injections. For those with recalcitrant disease, oral immunomodulators, oral minoxidil, and hydroxychloroquine have shown some benefit within the literature and should be reserved for second-line options.[2] These cases of refractory disease may not represent true CCCA, and biopsy for diagnostic confirmation should be considered. Future treatment considerations may include the use of hair grafting and antifibrotic therapy. A treatment algorithm is proposed in Figure 2.

The lack of inflammatory signs often shown at later stages of disease progression supports the hypothesis that CCCA may be primarily driven by dermal fibrosis. Subash et al.[7] proposed

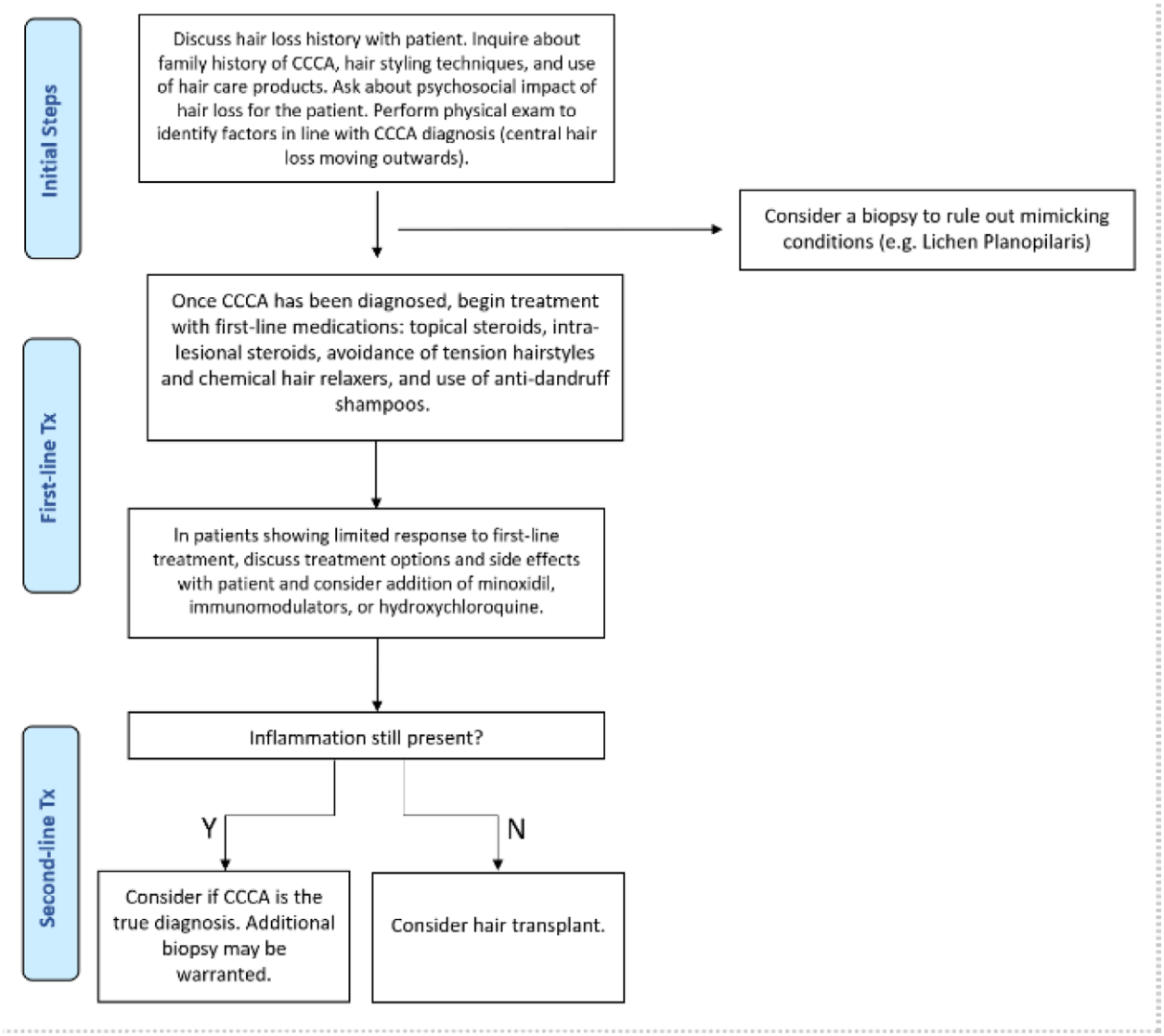

Fig. 2. Proposed treatment algorithm for CCCA.

a mechanism in which a primary insult (eg, traction hairstyles, chemical relaxers) may lead to lymphocytic infiltration of hair follicles on the scalp.[7] Some people may be at higher risk for this inflammatory cascade due to genetic predispositions such as decreased peroxisome proliferator-activator receptor gamma (PPAR-gamma) levels in the follicle. The decreased PPAR-gamma levels may lead to TGF-beta fibrotic processes taking over and driving the progression of disease.[27,28] If this hypothesis proves to be true, immunosuppressants may not be indicated for CCCA, and investigation into therapies blocking fibrosis pathways for management of CCCA would be imperative.

As for the potential association of CCCA and metabolic syndromes such as DM2 and hyperlipidemia, decreased expression of PPAR-gamma, which leads to lipid accumulation and infiltration of lymphocytes in CCCA, has been shown in both patients with CCCA and DM2.[29] However, the association between CCCA and these metabolic diseases still remains unclear across studies, with some showing a positive association and others no significance. Vitamin D has been shown to support keratinocyte differentiation in hair follicle formation and may underscore why its deficiency has been linked to CCCA in the literature.[23] In a study by Conic et al.,[30] it was found that African Americans had odds of severe vitamin D deficiency 6.3 fold greater than Caucasians, which may further support why CCCA disproportionally affects African Americans. Regardless, the association between CCCA and these comorbidities remains unclear, and it is recommended that clinicians base their screening on patients with CCCA, (such as HgA1C, lipids, blood pressure, and vitamin D levels), on a case-by-case basis.

Common RFs described for the development of CCCA often involve hairstyling practices such as traction hairstyles and the use of chemical relaxers. Results have been mixed in their connection to CCCA development, but as described above, an initial insult to the hair follicle can lead to lymphocytic infiltration and progressive fibrosis in predisposed individuals; these hairstyling techniques, may represent the initial insult leading to the pathologic cascade described in this mechanism.[7] Due to the unclear association, clinicians should consider cautioning patients who may be at increased risk of CCCA about these haircare practices.

Limitations of this review include the small cohort sizes among studies and the retrospective nature of the research. Future work should continue to focus on potential RFs and comorbidities in larger cohorts. Additionally, given CCCA's connection to fibroproliferative genes and uterine fibroids, additional research into the use of antifibrotic agents in the treatment of CCCA should be considered.

Conflicts of interest

None.

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Study approval

N/A

Author contributions

MG: Study design, data collection, and manuscript writing. AF: Data collection and manuscript writing. MV: Project oversight and manuscript editing.

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