Molluscum Contagiosum Virus Infection Can Trigger Atopic Dermatitis Disease Onset or Flare

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PRACTICE **POINTS**

- Molluscum contagiosum virus (MCV) infection appears to aggravate atopic dermatitis (AD) symptoms in a subset of pediatric patients.
- In susceptible children, the first onset of AD symptoms can occur during the course of MCV infection.

Predisposition to cutaneous viral infections is known to be a minor criterion of Hanifin and Rajka's diagnostic standard of atopic dermatitis (AD); however, the causal relationship between molluscum contagiosum virus (MCV) infection and AD onset or aggravation has not been widely explored. The objective of this study was to identify pediatric patients with AD onset or flare triggered by MCV infection as well as to characterize the setting under which MCV may trigger AD onset or flares in children. Fifty children with prior or current MCV infection presenting sequentially to an outpatient pediatric dermatology practice over a 1-month period were evaluated. Results indicated that children who contract MCV infection may be targets for skin care interventions to prevent and/or control AD.

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Molluscum contagiosum virus (MCV) is a common pediatric viral infection of the skin and/or mucous membranes.[1] It has been noted in increasingly younger patient populations, ranging from congenital cases resulting from perinatal/vertical transmission to transmission from cobathing and pool usage.[2,3] Adolescent cases of MCV infection presumed to be sexually transmitted also have been reported.[1]

An association between MCV infection and atopic dermatitis (AD) has been reported to be caused by a predisposition to prolonged and severe cutaneous viral infections.[4] However, the exact nature of the relationship between MCV and AD is unknown. It is not clear if there is a greater incidence of MCV infection in AD patients, a greater number of MCV lesions when MCV infection and AD co-occur,[5] or just more associated dermatitis in the setting of the combination of AD and MCV.[6]

The purpose of this study was to identify pediatric patients with AD onset or flare of AD triggered by MCV infection as well as to characterize the setting under which MCV may trigger AD onset or flares in children.

Methods

Medical records for 50 children with prior or current MCV infection who presented sequentially to an outpatient pediatric dermatology practice over a 1-month period were identified. Institutional review board approval was obtained. Patients were categorized according to the following parameters, which were identified as available data entry points: age at examination (last available); age at onset of MCV infection; duration of MCV infection (months); history of cobathing and with whom as well as presence of MCV infection in the cobather; usage of pools just prior to onset of MCV infection; enrollment in daycare just prior to onset of MCV infection; family and/or personal history of AD and/or psoriasis; presence of AD prior to onset of MCV infection; persistence of AD after clearance of MCV (yes/no); duration of AD following resolution of MCV infection; location of AD; location of MCV infection; number of MCV lesions documented; presence of unusual MCV morphology; therapeutics received; and comorbidities. Statistics were run using spreadsheet software.

Results

The age range of the 50 patients with MCV infection was 1 to 13 years, with an average age of 3.6 years at the

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TABLE 1. Characteristics of Pediatric Patients With MCV Infection

Characteristic	Value
Mean age, y (range)	
At examination	4.5 (1–13)
At onset of MCV infection	3.6 (1–13)
Mean duration of MCV infection at the time of evaluation, mo (range)	9.54 (1–24)
Sex, n (%)	
Male	18 (36)
Female	32 (64)
History of cobathing, n	
Yes	31
No	18
Unknown	1
Cobather, n	
Brother	6
Sister	21
Unspecified sibling	3
Cousin	1
Cobather has MCV infection, n	
Yes	20
No	11
History of swimming, n	
Yes	32
No	18
Family history of AD, n	
Yes	30
No	17
Unknown	3
Family history of psoriasis, n	
Yes	3
No	36
Unknown	11
Daycare attendance, n	
Yes	33
No	17
Medical history of psoriasis, n	
Yes	1
No	49
Presentation of AD prior to onset of MCV infection, n	
Yes	30
No	20
Comorbidities, n	
MRSA/MSSA superinfection	8
Verruca vulgaris	2
Diabetes mellitus	1
Hypothyroidism	1
Impetigo (unspecified bacteria)	1
Pityriasis alba	1
Giant lesions on buttocks	2
Giant lesions on thighs/neck	1
Linear lesions	1
Mean no. of MCV lesions, n (range)	9.7 (1–70)
Location of dermatitis, n	
Face	2
Chest	5
Abdomen	6
Back	4
Arms/antecubital region	10
Axillary region	2
Legs	28
Popliteal region	21

Abbreviations: MCV, molluscum contagiosum; AD, atopic dermatitis; MRSA, methicillin-resistant *Staphylococcus aureus*; MSSA, methicillin-sensitive *S aureus.*

TABLE 2. Characteristics of Pediatric Patients With AD Onset Triggered by MCV Infection

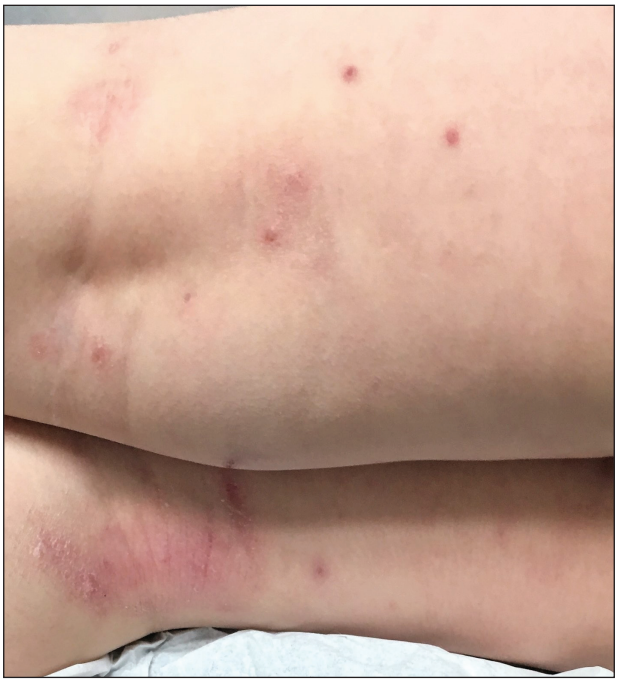
Characteristic	Value
Sex, n	
Male	1
Female	4
Mean age at onset of MCV infection, y (range)	3.6 (1–6)
Duration of MCV, mo (range)	5.8 (2–12)
Location of AD triggered by MCV, n	
Arms/antecubital region	2
Popliteal region	4
Buttocks	1
Number of MCV lesions, n (range)	8 (2–28)
Treatment received, n	
Topical corticosteroids	5
Cantharidin	3
Curettage/extraction	1

Abbreviations: AD, atopic dermatitis; MCV, molluscum contagiosum.

TABLE 3. Characteristics of Pediatric Patients With AD Flares Triggered by MCV Infection

Characteristic	Value
Sex, n	
Male	10
Female	1
Mean age at onset of MCV infection, y (range)	4 (1–11)
Mean MCV duration, mo (range)	9.6 (2–24)
Duration of ongoing AD flare after MCV resolution, mo (range)	6.2 (4–9)
Family history of AD, n	11
No. of MCV lesions, n (range)	2.5 (2–5)
Location of AD flare, n	
Chest	2
Abdomen	3
Back	1
Arms	3
Axillary region	2
Antecubital region	3
Legs	9
Popliteal region	7
Buttocks	3
Treatment received, n	
Topical corticosteroids	11
Topical mupirocin	8
Cantharidin	6
Cryotherapy	3
Extraction	2

Abbreviations: AD, atopic dermatitis; MCV, molluscum contagiosum.



Molluscum contagiosum virus infection with surrounding dermatitis in the popliteal region and legs in a child with atopic dermatitis.

onset of infection (reported by parents/guardians) and 4.5 years at presentation to the pediatric dermatology office (Table 1). Children 3 years of age or younger were more likely to have MCV lesions below the waist ($P<.05$). The majority of patients were female, but AD onset or flares triggered by MCV infection were not associated with sex.

The role of cobathing is unknown; however, 62% (31/50) of patients previously or currently cobathed at home, suggesting it may be a risk factor for MCV infection. An association of MCV lesions in the popliteal region trended toward being more likely with cobathing, but the association was not statistically significant.

Children with AD onset triggered by MCV infection statistically were more likely to have flexural localization of MCV and AD lesions and were statistically more likely to have a family history of AD ($P<.04$)(Table 2). Children with AD flares triggered by MCV infection were more likely to have MCV and AD lesions of the popliteal region and legs ($P<.05$)(Figure) and family history of AD ($P<.04$)(Table 3). Location of MCV lesions on the upper and lower extremities, buttocks, and genitalia were more likely to be associated with presence of any dermatitis than facial and/or truncal lesions ($P<.05$). Treatment of the MCV infection did not appear to impact the course of AD when present, but prospective interventions would be needed to assess this issue.

Superinfection with methicillin-resistant and methicillin-sensitive *Staphylococcus aureus* as well as atypical giant lesions of the intertriginous neck, inner thighs, and buttocks also were noted, but AD was uncommon in these cases. Given the limited number of cases, statistical significance could not be assessed.

Comment

Cutaneous infections with *Malassezia* have been postulated to trigger AD in infancy,[1] while systemic viral infections such as varicella-zoster virus may be protective against AD when acquired in younger children.[7] It appears that MCV infection in young children (eg, 3 years or younger) with specific localization to the flexural areas has the potential to trigger AD in susceptible hosts. Larger studies are needed to chart the long-term disease course of AD in these children. Due to the small size of this study, it is unclear if the rise of MCV infections since the 1980s has contributed to increased AD.[8] Susceptible children appear to have a family history of AD and localization of MCV lesions on the legs, buttocks, and antecubital region. Atopic dermatitis risk appears to be highest when MCV lesions are localized to intertriginous or flexural locations.

In addition to triggering the onset of AD, MCV infection also can trigger persistent flaring of AD, especially in the popliteal region and legs. Atopic dermatitis flares can occur at any age, but they appear to cluster in preschoolers and typically are not prevented by AD or MCV treatments; however, randomized trials are needed to identify if early intervention of MCV has a preventive benefit on AD onset or flares, and longer-term observation is needed to identify true disease course modification. Reduction of the number of MCV lesions previously has been demonstrated with institution of topical corticosteroid therapy.[6] Therefore, institution of atopic skin care generally is advisable in the setting of MCV infection. Future studies should address the potential use of interventions to prevent the triggering of AD onset or flares in the setting of MCV infection in children.[5]

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