Commonviralskininfection

Doneby •NabaaNasratMajeed. •GhadeerAbdAlraheemAbdAlaziz. •MahawissamTawfeeq. •MaryamMohammedBaker. •MustafaNeamahSahon. •BaneenAliHussien. •HebatullahAliMehdi. •MohammedHafidhSulaiman. •Mustafaaliasfoor. •AhmedAlaanKadhim. Supervisedby: Dr.ZeenaSaeedAl-Fadhily I Introduction Commonviralskininfection Skin lesions are prominentfeatures ofmanyviraldiseases.In some instances,characteristicskinlesionssuggestaspecificviralillness,the diagnosisofwhichcanbequicklyestablishedbyappropriateprocedures.In addition to clinicalmanifestations,laboratory methods including virus isolationareusedtodiagnoseviralinfections. Inviraldiseases,prophylaxishasprovedmoresuccessfulthanthespecific treatmentofestablishedinfection.However,recentprogressinmolecular biologyhasfacilitatedthedevelopmentofnewvaccinesandnewdrugsto treatviralinfections. Manyviralskininfectionscanbediagnosedclinicallyanddonotrequirea dermatologyreferralunlessthereisdiagnosticuncertaintyorifthepatient fallsintoaspecialhigherriskgroup.Immunocompromisedpatientsareat higherriskofpickingupviralskininfectionsandearlyspecialistadvice shouldbesought. Themostcommonskinviralinfectionsinclude: 1.Herpessimplexvirusinfections. 2.VaricellaZostervirusinfections. 3.HumanPapillomavirusInfections. 4.MolluscumContagiosum. 5.Measles(rubeola). 6.Rubella(Germanmeasles). 7.Roseolainfantum. 8.Erythemainfectiosum(fifthdisease). 9.Handfootmouthdisease.[1] II Humanpapillomaviralinfection Wartsarenon-cancerous(benign)skingrowthsthatdevelopondifferent partsofthebodyandcomeinvariousforms.Theyarecausedbyviruses. Wartsarecontagiousandverycommon:Mostpeoplewillhaveoneat somepointintheirlives.Althoughtheycanaffectpeopleatanyage,warts aremostcommonamongchildrenandteenagers.Mostwartsareharmless andwillgoawayontheirownwithinafewweeksormonths.Buttheycan bebothersomeandunattractive,andsomepeoplefeelashamed.Thereare anumberofdifferenttreatmentsthatcanmakewartsgoawaymore quickly– buttheydon'talwayswork.Viralwartsaren'tthesameas“senile warts”(seborrheickeratosis),whichusuallyfirstappearinolderageand aren'tcontagious.Senilewartsarealsoquiteharmless,butpermanent. Thisinformationisaboutviralwartsonly.[2] Symptoms Mostwartsdon'tcauseanybothersomesymptoms.Somemaycause itching,tightnessorafeelingofpressure.Wartsmightbepainfultoo, particularlythoseonthesolesofyourfeet.Somewartshavesmallblackor brownishdotscausedbyclottedbloodthathasleakedfromcapillaries(very finebloodvessels)intheskin.Wartsmayappearaloneoringroups,which maythencoverlargerareasofskin[2,3].Themaintypesofwartsinclude: ¬ Plantarwartsmostlyoccuronthe anklesandsolesofthefeet.Those onthebottom ofthefeetandtoes are sometimes referred to as verrucas.Theycanbecomequite III large.Becausethesolesofyour feethave to supportyourbody weight,plantarwartsdonot grow outward likeotherkindsof warts.Theyarepushedinwardwhenyoustandorwalk.This

cancausepainortendernessduetothepressure.Italso makesitdifficulttotreatthiskindofwart. ¬ Commonwartsareskingrowthsthat rangefromthesizeofapinheadtothe sizeofapea.Theyharden,making them roughandscalytothetouch. Commonwartsareoftenfoundonthe backofthehands,thefingers,theskin aroundthenails,andonyourfeet. ¬ Flatwartsaresmall,slightlyraisedwarts thatareoftenjustafewmillimeterswide. Sometimestheyarelightbrownincolor. Theyaremostcommonlyfoundonthe face,particularlyon the forehead and cheeks. Handsandlowerarmsareoftenaffectedtoo. ¬ Mosaicwartsarewhiteandaboutthesizeofapinhead.They areusuallyfoundontheballsofthefeetorunderthetoes,but mayalsospreadandcoverlargerareasontheentiresoleof thefoot.Mosaicwartsareflatterthanplantarwarts,andthey onlyrarelyhurtwhenyouwalk.[2,3] IV ¬ Filiform warts have a thread-like, spiky appearance.Becausetheyoftenappearonthe faceandsometimeslookliketinybrushes,they are usually considered to be especially bothersome. ¬ Genitalwartsaresmallhardnoduleswithrough surfaces.Theyare sexuallytransmitted and affectonlythegenitalarea.Thisinformation doesnotcovergenitalwarts. Sometypesofskincancermaylooklikewarts,buttheyareveryrare. Cornsarealsosometimesmistakenforplantarwarts.Butcornshavea singlevisiblecoreofdensehardskinintheircenter,whileplantarwarts oftenhavebrownishdots. Causes Wartsarecausedbyhumanpapillomaviruses(HPV),ofwhichthereare morethan100differenttypes.Thesevirusescanentertheskinthrough smallcutsandcauseextracellgrowth.Theouterlayerofskinturnsthicker andharder,formingaraisedwart.Wartvirusesaremainlyspreadbydirect skincontact,buttheymayalsobespreadbytouchingobjectsliketowels orrazors.Theyaremorelikelytoinfectmoistandsoftorinjuredskin.[2] Epidemiology Wartsareacommonmedicalproblem.Wartsarecommonworldwide and affectapproximately10%ofthepopulation.Inschool-agedchildren,the V prevalenceisashighas10%to20%.Theyaremorecommonamong immunosuppressedpatientsandmeathandlers.Wartscanoccuratanyage. Althoughrareininfancyandearlychildhood,prevalenceincreasesamong school-agedchildrenandpeaksat12to16years.Wartsaretwiceascommon inWhitesasinBlacksorAsians..Themale-to-femaleratioisapproximately equal.[2,3] Treatmentdependonnumberoflesions,siteandtypeofwart,cosmetic disability. • Keratolytics. • Chemicalcautery. • Retinoicacid. • Cryotherapy. • Electrocautery. • Topical5-flurouracil.Podophyllin20%. • Imiquimod5%. • Lasertherapy.[4] Herpessimplexvirus Herpessimplexvirustype1(HSV-1)isalineardsDNAvirusthatisa memberoftheAlphaherpesviridaesubfamily.HSV-1isresponsiblefor establishingprimaryandrecurrentvesicularеruptions,primarilyinthe VI orolabialand genitalmucosa.HSV-1 infection hasawidevarietyof presentations,includingorolabialherpes,herpeticsycosis(HSVfolliculitis), herpes gladiatorum,herpetic whitlow,ocularHSV infection,herpes encephalitis,Kaposivaricelliform eruption (eczema herpeticum),and severeorchronicHSVinfection.AntiviraltherapylimitsthecourseofHSV infection.Thisactivitydescribestheevaluation,andtreatmentherpes simplexvirustype1andreviewstheroleoftheinterprofessionalteam in evaluatingandtreatingpatientswiththiscondition. .Ingeneral,thepathogenesisofHSV-

1infectionfollowsacycleofprimary infectionofepithelialcells,latencyprimarilyinneurons,andreactivation. HSV-1 isresponsibleforestablishing primaryand recurrentvesicular eruptions,primarilyintheorolabialandgenitalmucosa.HSV-1infection hasawidevarietyofpresentations,includingorolabialherpes,herpetic sycosis(HSVfolliculitis),herpesgladiatorum, herpetic whitlow,ocularHSV infection,herpes encephalitis,Kaposi varicelliform eruption(eczemaherpeticum),andsevereorchronicHSV infection.Antiviraltherapylimitsthecourseofhsvinfection.[5,6,7] Epidemiology Ithasbeenhypothesized thatapproximatelyone-third oftheworld's populationhasexperiencedsymptomaticHSV-1atsomepointthroughout hisorherlifetime.HSV-1firstestablishesprimaryinfectioninpatientswith noexistingantibodiestoHSV-1orHSV-2.Non-primaryinitialinfectionis definedasinfectionwithoneHSVsubtypeinpatientswhoalreadyhave antibodiestotheotherHSVtype(i.e.,HSV-1infectioninapatientwithHSV -2antibodies,orviceversa). Reactivationresultsinrecurrentinfectionandmostcommonlypresentsas VII asymptomaticviralshedding. pregnancyhaveahigherrisk.[8,9,10] Pathophysiology HSV-1istypicallyspreadthroughdirectcontactwithcontaminatedsaliva orotherinfectedbodilysecretions,asopposedtoHSV-2,whichisspread primarilybysexualcontact. HSV-1beginstoreplicateatthesiteofinfection(mucocutaneous) andthen proceeds to travel by retrogradeflow downan axontothedorsalrootganglia(DRG).ItisintheDRG thatlatencyis established.Thislatencyperiod allows thevirus toremain ina non-infectiousstateforavariableamountoftimebeforereactivation. HSV-1 is sly in its ability toevade the immune system via severalmechanisms. Onesuch mechanismisinducinganintercellularaccumulationofCD1dmolecules inantigenpresentingcells. Normally, these CD1d molecules are transportedtothecellsurface,wheretheantigenispresentedresultingin thestimulationofnaturalkillerT-cells,thuspromotingimmuneresponse. When CD1d molecules are sequestered intercellularly,the immune responseisinhibited.HSV-1hasseveralothermechanisms by which it downregulates variousimmunologiccellsandcytokines. HSV-1isthemostcommonculpritoforolabialherpes(asmallpercentof casesareattributedtoHSV-2).Itisimportanttonotethatorolabialhsv-1 infectionismostcommonlyasymptomatic.Whentherearesymptoms,the mostcommonmanifestationisthe"coldsore"orfeverblister.Inchildren, symptomaticorolabialHSV-1infectionsoftenpresentasgingivostomatitis thatleadstopain,halitosis,anddysphagia.Inadults,itcanpresentas VIII pharyngitisandamononucleosis-likesyndrome. Symptomsofaprimaryorolabialinfectionoccurbetweenthreedaysand one week afterthe exposure.Patients willoften experience a viral prodromeconsistingofmalaise,anorexia,fevers,tenderlymphadenopathy, localized pain,tenderness,burning,ortingling priorto the onsetof mucocutaneouslesions.PrimaryHSV-1lesionsusuallyoccuronthemouth andlips.Patientswillthendemonstratepainfulgroupedvesiclesonan erythematous base.These vesicles exhibita characteristic scalloped border.Thesevesiclesmaythenprogressto pustules,erosions,and ulcerations.Within2to6weeks,thelesionscrustoverandsymptoms resolve. Symptomsofrecurrentorolabialinfectionaretypicallymilderthanthoseof primaryinfection,witha24-hourprodromeoftingling,burning,anditch. RecurrentorolabialHSV-1infectionsclassicallyaffectthevermillionborder

ofthelip(asopposedtothemouthandlipsasseeninprimaryinfection). Thegold standard fordiagnosing HSV-1 infection isHSV-1 serology (antibodydetectionviawesternblot).Themostsensitiveandspecific mechanism isviralpolymerasechainreaction(PCR).However,serology remainsthegoldstandard. Viralculture,directfluorescentantibody(DFA)assay,andTzancksmear arealternativemethodsofdiagnosing.Itisimportanttonotethatthe Tzancksmearidentifiesmultinucleatedgiantcells,soitcannotdistinguish betweenHSVandVZV.TheDFAassay,however,candistinguishbetween the2entities.[11,12,13] IX Treatment/Management Forthetreatmentoforolabialherpes,thecurrentrecommendationisoral valacyclovir(2gramstwicedailyforoneday).Ifthepatienthasfrequent outbreaks,chronicsuppressioniswarranted.Forchronicsuppressionof immunocompetentpatients,oralvalacyclovir500mgdaily(forpatients withlessthantenoutbreaksperyear)ororalvalacyclovir1grambymouth daily(forpatientswithgreaterthan10outbreaksayear)isrecommended. Forthetreatmentofeczemaherpeticum,itisrecommendedtouse10to 14daysofeitheracyclovir(15mg/kgwitha400mgmaximum)3to5 timesdailyorValacyclovir1grambymouthtwiceaday. ForimmunocompromisedpatientswithsevereandchronicHSV,treatment is aimed at chronic suppression. For chronic suppression of immunocompromisedpatients,oralacyclovir400to8002to3timesdaily, ororalvalacyclovir500mgtwicedailyisrecommended.[14,15,16] Herpeszoster Varicella-zostervirus(VZV)causeschickenpoxandherpeszoster (shingles).Varicellaischaracterizedbyapruritic,maculopapular, vesicularrashthatevolvesintononinfectiousdriedcrustsovera 3-to7-dayperiod.Reactivationofthedormantvirusresultsin thecharacteristicpainfuldermatomalrash ofherpeszoster, whichisoftenfollowedbypaininthedistributionoftherash (postherpeticneuralgia). X VZVinfectiongivesrisetotwodistinctsyndromes.Theprimary infection,chickenpox,isacontagiousandusuallybenignfebrile illness.Afterthisinfectionresolves,viralparticlesremaininthe dorsalrootorothersensoryganglia,wheretheymaylaydormantfor yearstodecades. VZVreactivateswhenthehostmechanismsfailtocontainthevirus. Suchfailuremayresultfrom awide spectrum of conditions, ranging from stresstosevereimmunosuppression;occasionally, itfollowsdirecttrauma Herpeszoster(shingles)isanacute,cutaneousviralinfectioncaused bythereactivationofvaricella-zostervirus(VZV),aherpesvirusthat is the cause ofvaricella (chickenpox).Differences in clinical manifestations between varicella and herpes zosterapparently dependonanindividual'simmunestatus;thosewithnoprevious exposureto VZV,mostcommonlychildren,develop theclinical syndrome ofvaricella,whereas those with circulating varicella antibodiesdevelopalocalizedrecrudescence,zoster.[17,18] Zosterprobablyresultsmostoftenfrom afailureoftheimmune system tocontainlatentVZVreplication.Whetherotherfactors, such as radiation,physicaltrauma,certain medications,other infections, and stress, alsocantriggerzosterhasnotbeen determinedwithcertainty.Norisitentirelyclearwhycirculating varicellaantibodiesandcell-mediatedimmunemechanismsdonot preventrecurrentovertdisease,asis commonwithmostother viralillnesses. XI Herpeszostermanifestsinmanyways.Itshouldnotbeconsidered simplyaself-limiteddermatomalrashwithpain.VZVinfectionisan

acuteneurologicdiseasethatwarrantsimmediate evaluation.
ThatVZVisalwaysabenigndisorderisacommonmisperception.
OnceVZVinfectionresolves,manyindividualscontinuetosuffer pain—
aconditionknownaspostherpeticneuralgia(PHN).
Theacuteeruptivephaseismarkedbytheemergenceofvesicular eruptions.Patients may also experience some of the other symptoms seen in the preeruptive phase.Lesions begin as erythematous macules and papules thatquickly develop into vesicles.New lesionstendtoform overaperiodof3-5days, sometimescoalescingtoform bullae.Aftertheyform vesicles, lesionsprogressthroughstagesinwhich they rupture,release theircontents,ulcerate,andfinallycrustoverandbecomedry.
Patientsremaininfectiousuntilthelesionshavedried.[19]
Duringthisphase,almostalladultpatientsexperiencepain(ie,acute neuritis).Afew experienceseverepainwithoutanyevidenceofa
vesiculareruption(ie,zostersineherpete),andasmallnumberhave acharacteristic eruptionbutdonotexperiencepain. Symptomsandlesionsintheacuteeruptivephasetendtoresolve over10-15days.However,lesions mayrequireuptoamonthto XII
completelyheal,andtheassociatedpainmaybecomechronic. IntheUnitedStates,approximately95% ofadults—and99.5% of adultsaged40yearsorolder—haveantibodiestoVZVandthusare vulnerabletoreactivationofinfection.]Apersonofanyagewitha
previousvaricellainfectionmaydevelopzoster,buttheincidence increases with advancing age as a consequence ofdeclining immunity.[20,21] Approximately4%ofpatientsdeveloparecurrentepisodelater inlife.Recurrentzosteroccursalmostexclusivelyinpeople
whoareimmunosuppressed.Approximately25%ofpatientswith HIVand7-9%ofthosereceivingrenaltransplantationorcardiac transplantationexperienceaboutofzoster.
Overtheperiodofalifetime,10-20%ofthosewithprimaryinfections went ontoexperienceepisodesofherpeszoster.High-riskgroups,such aselderlypopulationsandimmunocompromisedpeople,might
experiencecumulativeincidencesashighas50%.Theestimated
annualnumberofcasesintheUnitedStatesisapproximately1 million.
Sincetheintroductionofwidespreadvaccinationforvaricellain
1995,theincidenceofprimaryVZVinfectionintheUnitedStates hasbeenreduced byup to 90%.However,theeffectofthis vaccination,as wellas thatof the subsequently approved vaccinationforherpeszoster,onthecurrentandfutureincidence XIII
ofherpeszosterremainstobedetermined. Itisrareinchildrenandyoungadults,exceptinyoungerpatients with AIDS,lymphoma,othermalignancies,and otherimmune deficienciesandinpatientswhohavereceivedbone marrow or kidneytransplants.[19] Diagnosis Adermatologistcanoftendiagnoseshinglesbylookingatthe rashonyourskin. Ifthereisanyquestionaboutwhetheryouhaveshingles,your
dermatologistwillscrapeabitoffluidfrom ablister.Thiswillbe
senttoalabwhereadoctorwilllookatthefluidunderahigh□poweredmicroscope. Treatment:
TopicalTreatments Thereareavarietyoftopictreatments,includingtopicalacyclovir 5%cream,lidocaine,andcapsaicin. Antiviralagents
Manystudieshavefoundacycloviranditsderivatives(valacyclovir,
famciclovir,penciclovir,anddesciclovir,whichisnotavailable in

theUnitedStates)tobesafeandeffectiveintreatingactivedisease and preventing PHN. Their mechanism of action involves preventing VZV replication through inhibition of viralDNA polymerase.Valacyclovirandfamciclovirarenotapprovedbythe USFood and Drug Administration(FDA)fortreatmentofherpes zosterinchildren;acyclovirismorecommonlyused. XIV Antiviraltherapymaydecreasethelengthoftimefornew vesicle formation,thenumberofdaystoattaincompletecrusting,andthe daysofacutediscomfort.Usually,theearlierantiviralmedications arestarted,themoreeffectivetheyareinshorteningthedurationof zosterandinpreventingordecreasingtheseverityofPHN.Ideally, therapy should be initiated within 72 hours of symptom onset.[20,21] Chickenpox Varicella infection is caused byvaricella-zostervirus (VZV),a ubiquitous,highly contagious,human alpha-herpes virus.The diseaseisusuallynamedchickenpox;itisgenerallymildandself□limiting and has worldwide distribution.Varicella results from primaryVZVinfection,afterwardsthevirusestablishesinalatent stateincellsofthedorsalrootganglia,and,whenreactivated, causesherpeszosterinfectionorshingles.Thus,herpeszoster occursonlyinindividualswhohadprimaryVZV infection. The averagecourseofvaricellainchildrenincludesfeverof2-3days' duration,accompaniedbyavesicularrashinvolvingamedianof300 lesions.Personsover15yearsofageandchildrenunderoneyear tend to havemoresevereVZV infection and ahigherrateof complications.The mostcommon in children is a secondary bacterialinfectionof theskin;theincidenceofextracutaneous complicationsofvaricellaislowandconsistsofcerebellarataxia, encephalitis,hepatitis,Reye'ssyndromeandpneumonia,the latter beingthemostcommoninadults.Womenwhocontractvaricellain XV thefirst20weeksofpregnancyareinriskoftheirfetusdeveloping congenitalvaricellasyndrome,andofprematuredelivery.Ifonsetis justbeforeterm,severeneonatalvaricellawithrapidlydisseminated infectionandextensivevisceralinvolvementoccursintheinfant.In addition,pregnantwomenwhocontractvaricellainthelasttrimester areatriskofseverepneumoniaanddeath.Mostcasesofherpes zosteroccurinindividualsover45yearsoldandtheincidence increaseswithadvancingage,itisa localized,painful,vesicular rashinvolvingoneorseveraladjacentdermatomes.Depressionof cell-mediatedimmunityisthemainriskfactorfordevelopingherpes zoster.Itiscommoninpatientstreatedwithimmunosuppressive drugsformalignantdiseasesortopreventrejectionofbonemarrow ororgan transplants.Patients with AIDS and those receiving systemicsteroidtherapyforchronicdiseasessuch asrheumatoid arthritisorlupuserythematosushavealsoanimportantrisk. Incidenceofvaricella—Varicellaoccursthroughouttheyearin temperateregions,buttheincidencetypicallypeaksinthemonths ofMarchthroughMay[2].Accordingtonationalseroprevalencedata from thepre-vaccineera,greaterthan95percentofpersonsinthe UnitedStatesacquiredvaricellabefore20yearsofage,andfewer than2percentofadultsweresusceptibletoinfection[3-6].Priorto 1995 the Centers forDiseaseControland Prevention (CDC) estimatedtheyearlyincidenceofchickenpoxin theUnitedStatesatapproximatelyfourmillioncases,withnearly 11,000admissionsand100deaths.[22,23,24]

XVI ClinicalpresentationInitialsymptomsofchickenpoxincludefatigue, amildfever,lackofappetite,andafeelingofbeinggenerallyunwell. Thisisquicklyfollowed(usuallywithin24hours)bythedevelopment ofaredrash,whichusuallyappearsonthechestand/orbackfirst, laterspreadingtotheface,scalp,arms,andlegs. Twelveto48hourslatertherashdevelopsintosmallredspots. Thesethenturnintoyellowfluid-filledblisters,whichburstanddry up3-4daysaftertheyappear.Theremaybeseveralcropsofspots occurringover4-5days.Thespotscauseitching,whichmaybe severe.Theymayoccuralloverthebody,includingthemouthand genital area.Somepeoplemayhaveonlyafew spotswhereas otherswillhavehundreds.Symptomsstartappearing10-21days afterexposuretothevirus.Fullrecoveryfrom chickenpoxusually takes7-10daysafterthesymptomsfirstappear.[22,23] XVII Treatment There is no specific treatmentforchickenpox,butthere are pharmacyremediesthatcanalleviatesymptoms.Theseinclude paracetamoltorelievefever,andcalaminelotionandcoolinggelsto easeitching.Inmostchildren,theblisterscrustupandfalloff naturallywithinonetotwoweeks.[23] Vaccinationandprevention Apersonwithchickenpoxisinfectiousfrom1-2daysbeforetherash firstappearsuntilafterthefinalcropofblistershaveformedscabs, approximately5-10dayslater.Thenatureoftheinfectiousperiod makesitverydifficultto preventthe disease from spreading. Nonetheless,childrenshouldstayawayfromdaycareorschool,and publicplaces,whiletheyareinfectious.Adultswithchickenpoxwho workwithchildrenshouldalsostayhome. The chickenpox (varicella)vaccine is the bestwayto prevent chickenpox. Onedoseofthevaccineprovidesapproximately99% protection againstseverechickenpoxand80%protectionagainstchickenpox ofanyseverity.Vaccinationmaypreventorreducetheseverityof chickenpoxifitisgivenwithin3–5daysofexposuretosomeone withthedisease. Thevaricellavaccineisavailablefrom GPs.Itisfullyfundedfor infantsat15monthsofageorchildrenat11yearsofagenot previouslyinfectedwithorvaccinatedagainstchickenpox,aspartof the New Zealand Ministry of Health's nationalimmunisation XVIII schedule.Thevaricellavaccineisalsorecommendedandfundedfor certainhigh-riskgroupsandisavailableatacosttootherpatients .[23,24,25] Molluscumcontagiosum: Molluscum contagiosum iscausedbyapoxvirusoftheMolluscipox genus.Preschoolandelementaryschool-agedchildrenaremore commonlyaffected.The virus is transmitted byclose physical contact, autoinoculation, and fomites. Typically, molluscum contagiosum presentsasasymptomatic,discrete,smooth,flesh□colored,dome-shapedpapuleswithcentralumbilicationfromwhicha plugofcheesymaterialcanbeexpressed.Someauthorssuggest watchfulwaiting of the lesions.Many authors suggest active treatment of lesions for cosmetic reasons or concerns of transmission and autoinoculation. Active treatments may be mechanical(e.g.cryotherapy,curettage,pulseddyelasertherapy), chemical(e.g.cantharidin,potassium hydroxide,podophyllotoxin, benzoylperoxide,tretinoin,trichloroaceticacid,lacticacid,glycolic acid,salicylicacid),immune-modulating(e.g.imiquimod,interferon□alpha,cimetidine)and anti-viral(e.g.cidofovir).Recentpatents relatedtothemanagementofmolluscum contagiosum arealso retrieved and discussed. These patents comprise of topical compositions and herbal Chinese medicine with limited documentationoftheirefficacy.[26,27] Epidemiology:ofMC islargelyofpoorquality.Thelargest XIX

incidenceisinchildrenagedbetween0and14years,where theincidenceraterangedfrom 12to14episodesper1000 childrenperyear.IncidenceratesintheUKwerehighestin those aged 1-4 years.Meta-analysis suggests a point prevalencein children aged 0-16yearsofbetween5.1%and 11.5%.There is evidence for an association between swimmingandhavingMCandMCismorecommoninthose witheczema;however,thereislittleevidenceforotherrisk factors. The diagnosis:ofMC is clinicallybased on the distinguishing characteristicsofthelesions. Complications • Inflammationorinfection(cellulitis) • Irritation • Conjunctivitisifthelesionsareontheeyelids • Abscess[27,28,33] Treatment: Currently,theneed foractivetreatmentin patientswith MC is controversial,giventheself-limitedcourseofinfection,thelarge numberoftherapeuticalternativesavailable,andthelackofevidence todefinethebesttherapy.Thereisaconsensusthattreatment shouldbeindicatedinpatientswithextensivedisease,secondary XX complications (bacterial superinfection, molluscum dermatitis, conjunctivitis),or aesthetic complaints.A retrospective study evaluatedtheresolutionrateofthelesionsintreatedanduntreated MCpatients,showingaresolutionat12monthsof45.6% inthe treatedgroupand48.8%intheuntreatedgroup.At18months,they foundaresolutionrateof69.5%and72.6%inthetreatedversusthe untreatedgroup,respectively.From thiscardinalstudy,itappears thatactivetreatmentdoesnotimprovetheresolutionratewhen comparedtoobservationalone. Forallpatients,generalmeasuresarerecommendedtopreventthe spreadofMCV.Itshouldbeadvisednottoscratchorrubthelesions; besides,patientsshouldnotsharetowels,tub,orbathutensils.Active treatments can be classified as mechanical, chemical, immunomodulatory,andantiviral.[29,31,32,34] XXI Patients&methods Thisstudyisaretroanalysisstudyconductedindermatologyoutpatients' clinicinMerjanteachinghospitalovertheperiodofApril-May2022. Datacollectedbyfourthstagemedicalcollegestudentsfrom hospital records. Anypatientattendedoutpatientclinicinthishospitalintheyear2021 whowerediagnosedwithcutaneousvirusinfectionregardlessthetype wereincludedinthisstudy. Noexclusioncriteria Demographicdatalikename,sex,ageofthepatient,dateofvisitingwere collected. Typeofskinviralinfectionwasdiagnosedbydermatology specialistintheclinic. XXII Resultsanddiscussion