

Rashes & Skin Conditions in Pregnancy

1. Introduction and who the guideline applies to:

This guideline is intended for the use by all UHL staff involved in the care of pregnant patients who have presented with skin disorders.

Related UHL Documents:

[Chickenpox in Pregnancy UHL Obstetric Guideline C16/2015](#)

[Obstetric Cholestasis UHL Obstetric Guideline C1/2013](#)

[HIV Screening and Management in Pregnancy UHL Obstetric Guideline C63/2004](#)

Background:

Hormonal changes in pregnancy may result in physiological alterations in the skin. Itching is relatively prevalent in pregnancy. Common Causes include dry skin, atopic eczema, irritant contact dermatitis and scabies.

This guideline will focus on the different causes of rash in pregnancy including viral exanthems (a rash compatible with a systemic viral illness).

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2. Guidance:

Normal Skin Changes in Pregnancy:

Hormonal changes in pregnancy may result in physiological alterations in the skin, for example:

- Appearance or darkening of a vertical band down the middle of abdominal skin (linea nigra) which may normally appear paler than normal skin (linea alba)
- Melasma – patches of light brown facial pigmentation – it develops in 70% women in second half of pregnancy (using a sunblock cream will help even up the pigmentation).
- Darkening of areola, nipple and genital skin
- Increased redness of palms (palmar erythema)
- Telangiectasia (dilated small red blood vessels), venulectasia (dilated bigger blue-coloured vessels) and varicose veins
- Striae gravidarum (stretch marks)
- Hair thinning (telogen effluvium) in the post-partum period between 4 and 20 weeks after delivery. Usually recovers within 6 months.

Common Rashes in Pregnancy:

Polymorphic Eruption of Pregnancy

Also known as pruritic urticarial papules and plaques of pregnancy (PUPPP). Features include:

- Onset in the 3rd trimester and remission occurs within a few days of delivery.
- It more frequently arises in primigravidae and multiple pregnancies
- Itchy erythematous papules and plaques first appear on abdominal striae and then spread to trunk and proximal limbs; umbilicus is spared
- Direct immunofluorescence is negative (unlike pemphigoid gestationis)

Treatment –

1. Generous amounts of emollients to be used to wash with (avoiding soaps and shower gels) and applied at least twice daily to avoid skin getting dry: Zeroderm ointment or Epimax cream. If not tolerated, Hydromol ointment or Diprobase cream.
2. 1% hydrocortisone or Eumovate[®] ointment or cream generously to itchy rash can be safely used to start with if rash is very mild, but usually stronger topical steroids such as Betnovate[®] or Dermovate[®] ointment or cream will be required, if rash is very itchy.
3. Sedative antihistamine – Chlorpheniramine (Piriton[®]) or Promethazine (Phenergan[®]) at night.
4. Systemic steroids are occasionally required for intractable pruritis.

Pemphigoid Gestationis

This is a rare blistering disease due to circulating IgG autoantibodies similar to those found in bullous pemphigoid, targeting a basement membrane zone protein BPAG2 (BP180) within the hemidesmosome. Features include:

- The onset of pemphigoid gestationis is most often in the 2nd trimester (weeks 13 to 26), but it may arise at any stage and usually improves at the end of pregnancy but may even be worse postpartum
- It can recur with menstruation, with oral contraceptives and in further pregnancies
- The itchy papules mainly affect the abdomen, including umbilicus, but may generalise, with grouped or annular red papules, plaques, target lesions, annular wheals. After variable delay (usually 2 weeks) vesicles and large tense bullae form.
- Autoimmune and associated with bullous pemphigoid and other autoimmune conditions e.g. Grave's disease, vitiligo, insulin dependent diabetes, rheumatoid arthritis.
- Direct immunofluorescence (a test done as part of a skin biopsy) shows deposition of C3 and or IgG or other antibodies
- Severe pemphigoid gestationis should be treated by oral corticosteroids.

Treatment –

1. Potent topical corticosteroids (0.1% mometasone furoate, Elocon® cream) or very potent (Clobetasol propionate, Dermovate® cream)
2. Most require systemic corticosteroids
3. Sedative antihistamines

Prurigo of Pregnancy

- Occurs at 25-30 weeks
- Pruritic groups of red/brown excoriated papules.
- Improves at delivery although papules may persist for several months post-partum

Treatment –

1. Emollients and topical steroids
2. Antihistamines
(as per treatment of Polymorphic Eruption of Pregnancy above)

Pruritic folliculitis of pregnancy

- Mainly occurs in third trimester, sometimes second
- Widespread distribution, predominantly on the trunk thighs and arms. The eruption consists of acneiform, pruritic, erythematous, follicular papules and pustules.
- Usually settles prior to delivery and resolves within 2 weeks after delivery.

Treatment –

1. Antiseptic emollient to wash with (Dermol 500 lotion or Eczmol cream)
2. Moisturize regularly avoiding greasy/thick emollients
3. Topical steroids if itchy (ie bethametasone cream)
4. Antihistamines

Table 1: Incidence & risk to fetus

Condition	Incidence	Fetus	Parity	Recurrence
Polymorphic eruption of Pregnancy (PEP, PUPP)	Commonest About 0.5%	No effects	Primiparous Multiple pregnancies	Rare
Pemphigoid gestationis	Rare (1 in 10,000-1 in 60,000)	Increased risk to fetus associated with low birth weight and preterm delivery. Neonate may have similar rash – in 10% cases and it is mild and transient.	Primiparous or Multiparous	Usually recurs in future pregnancies (earlier onset and more severe)
Prurigo	1:300	No effects	Multiparous	Is possible
Folliculitis	1:3000	No effects	Unknown	Unknown

Intrahepatic Cholestasis (Obstetric Cholestasis)

OC affects about 1% of pregnancies. It results in unexplained pruritus (itch) during the second and third trimesters, with raised blood levels of bile acids and/or liver enzymes. Intrahepatic cholestasis is associated with an increased risk of preterm delivery and stillbirth. Characteristically there is no rash seen in this condition.

See “[Obstetric Cholestasis UHL Obstetric Guideline](#)” guideline for further information.

Viral rashes In Pregnancy

Adapted from the Health Protection Agency (HPA) 2011 “Guidance on Viral rash in Pregnancy”.

Rubella, parvovirus B19, measles and chickenpox infections are of particular significance for the fetus and intervention can prevent or reduce the potential for adverse outcomes. Any febrile illness, including those that can present with a rash, may be associated with an increased risk of fetal loss in the first trimester. The specific risk associated with each individual viral infection is therefore difficult to ascertain.

Pregnant women may present with a generalised rash or after contact with a person who has a generalised rash, the cause of which is not always clinically apparent.

Viral infections that commonly present with a generalised rash illness in the UK include:

- Varicella
- Cytomegalovirus - usually no rash but, rarely may present with mild maculopapular rash
- Enterovirus
- Human herpes virus 6 &7
- Epstein-Barr virus
- Measles
- Parvovirus B19
- Rubella

Rubella

The clinical features and consequences for the fetus of primary rubella in pregnancy are well established. The risk to the fetus of primary rubella in the first 16 weeks gestation is substantial with major and varied congenital abnormalities being associated with infection in the first trimester.

Rubella infection between 16 and 20 weeks gestation is associated with a minimal risk of deafness only and rubella prior to the estimated date of conception or after 20 weeks carries no documented risk.

If investigation is required, the request form must clearly state that the woman is pregnant, recent rubella is a possibility and provide full clinical and epidemiological details. It is recommended that, irrespective of a request for specific rubella or parvovirus B19 testing, all sera from women with rash illness are simultaneously investigated for both infections. It is recommended that the laboratory investigates all cases of possible rubella by simultaneous testing for rubella-specific IgG (or total rubella antibody) and IgM. Acute rubella infection can also be diagnosed by PCR (polymerase chain reaction) testing on saliva, urine and blood.

No pregnant woman should have rubella diagnosed on the basis of a single positive rubella specific IgM alone. Results must be interpreted in relation to full clinical and epidemiological information.

To demonstrate rubella seroconversion, the booking blood should also be tested for rubella IgG and the result compared with the current blood. If the booking blood is rubella IgG NEG and the current blood is rubella IgG POS, this demonstrates seroconversion during pregnancy and is a more reliable indicator primary rubella infection. Rubella IgG avidity on the current blood will then indicate when the rubella infection is likely to have occurred. Although routine antenatal rubella IgG screening ceased in April 2016, the booking blood sample should still be available for testing.

Unless seroconversion has been shown, further testing by alternative rubella specific IgM tests, testing an acute sample and a sample taken 10–14 days later for rubella IgG, and measuring the strength of binding of specific IgG (avidity) is advised. IgG avidity is low soon after a primary infection, but matures over a few weeks to become more strongly binding.

The management of primary rubella or symptomatic rubella re-infection would depend on the gestation of pregnancy at which rubella occurred and the individual circumstances of the woman.

Parvovirus B19 (B19V)

There is a wide range of potential consequences of parvovirus B19 infection. These extend from minor febrile illness to erythema infectiosum (fifth disease, slapped cheek syndrome), a generalised rash illness clinically indistinguishable from rubella, aplastic crises in patients with increased red cell turnover, arthropathy, and persistent infection in the immunocompromised.

Infection in the first 20 weeks of pregnancy can lead to intrauterine death (risk 15% c.f. 5% in control group; excess risk 9%) and hydrops fetalis (risk 3% if infection between 9–20 weeks

gestation of which about half die. These consequences usually occur some 3–5 weeks after the onset of maternal infection, but can be later.

Permanent congenital abnormality and/or congenital anaemia have rarely been identified as a consequence of intrauterine infection.

In patients with a rash, recent parvovirus B19 infection can be confirmed or excluded by testing for parvovirus B19 specific IgM on the first serum obtained. Failure to detect parvovirus B19 specific IgM excludes infection in the four weeks prior to collection of the serum. Hence infection cannot be excluded if investigation commences more than four weeks after onset of rash illness.

If parvovirus B19 IgM is detected in the first 20 weeks of pregnancy, confirmation is required by alternative assay, e.g. detection of high titre B19V DNA or IgG seroconversion using an antenatal booking blood. Acute parvovirus B19 infection can also be diagnosed by PCR testing on saliva and blood.

The management of proven parvovirus B19 infection has become more active with the demonstration that intrauterine transfusion of the fetus improves the outcome. On diagnosis of parvovirus B19 infection, specialist advice from Fetomaternal Consultant should be sought including the need for serial ultrasound scanning and Doppler assessment in the case of development of hydrops fetalis.

Measles

The clinical features and complications of measles in the child and adult are well established and include disseminated rash, coryza, conjunctivitis, pneumonia, otitis media, encephalitis. Measles in pregnancy is relatively uncommon but can be associated with severe maternal morbidity as well as fetal loss and preterm delivery.

Although rare, neonatal measles has been associated with sub-acute sclerosing panencephalitis (SSPE) with a short onset latency and fulminant course and acquiring measles infection before one year of age is associated with an increased risk of SSPE.

The serological diagnosis of measles is well established. A serum at first presentation should be collected and sent for laboratory testing for measles-specific IgM and IgG. Acute measles infection can also be diagnosed by PCR testing on saliva, urine and blood.

Recent measles infection can be confirmed or excluded by testing for measles-specific IgM on serum sample taken more than four days, but within one month, after the onset of rash. When measles has been confirmed the management of the pregnancy should continue as normal. Although no congenital infection or damage would be anticipated, follow-up of the infant should be considered.

Epstein-Barr Virus (Glandular Fever)

Infectious mononucleosis (IM) is a common presentation of primary Epstein-Barr virus (EBV) in young adults. IM is characterised by generalised lymphadenopathy, fever, sore throat and typical haematological and serological findings, including the detection of heterophil antibody. A generalised maculopapular rash may be an associated accompanying feature, particularly if ampicillin, or a similar antibiotic, has been taken.

Primary EBV infection in pregnancy (whether clinically-apparent as IM or asymptomatic) carries no specific risk to the fetus. EBV infection results in a latent infection with persistent excretion in the throat of a proportion (c. 20%) of individuals. Hence exposure to EBV can occur irrespective of whether the contact patient has IM, and exposure to IM does not require investigation and the patient can be reassured.

Cytomegalovirus (CMV)

CMV can be another cause of infectious mononucleosis, although primary infections are generally mild or even asymptomatic. Rarely patients may present with a generalised maculopapular rash.

Following primary infection the virus remains latent and can periodically reactivate throughout life, and especially in pregnancy. The fetus can be infected either during primary infection or reactivation, and CMV infection is now the commonest cause of viral congenital infection. It is estimated that the overall birth prevalence of congenital CMV infection in the UK is around 3/1000. However, there is no treatment currently recommended to prevent or reduce mother-to-child transmission.

If primary infection or re-infection is suspected it should be appropriately investigated with CMV-specific assays and, if indicated, referral to an appropriate specialist unit. Acute CMV infection can also be diagnosed by PCR testing on saliva, urine and blood.

Enteroviruses

Enterovirus infection (Coxsackie virus groups A and B; echovirus; enterovirus 68-71) may have a wide range of manifestations such as meningitis, rash, febrile illness, myocarditis and Bornholm disease. Sporadic enterovirus infection is not uncommon, but major epidemics have not been seen in the UK for some years. Except for poliovirus, no vaccines are available.

Vertical transmission has been documented in pregnancy. Whilst infection with Coxsackie virus during pregnancy has been associated with early onset neonatal hepatitis, congenital myocarditis, early onset childhood insulin dependent diabetes mellitus and abortion or intrauterine death there is no clear causal relationship. There are no known treatments or preventative methods.

Hand, foot and mouth disease is an enteroviral infection characterised by vesicular lesions of hands, feet, and mouth; the latter soon break down to ulcers. Pregnant women presenting with the characteristic features of hand, foot and mouth, or who have been in contact with the infection may be reassured that there is no adverse consequence for the fetus.

Varicella: see [Chickenpox in Pregnancy UHL Obstetric Guideline](#)

Disseminated primary chickenpox (varicella-zoster virus – VZV infection) presents as an illness characterised by vesicular rash, and clinical diagnosis is highly specific although not very sensitive as sub-clinical and mild cases occur. Chickenpox is endemic within the UK, with more than 85% of young adults having been infected. The incubation period is 7–24 days. This can be prolonged if the patient is on steroids, immunosuppressed or has received VZIG (varicella zoster immunoglobulin).

For investigation and consideration of VZIG, and contact management, the patient is considered infectious 48 hours before the rash appears and until vesicles crust over.

Although often just a clinical diagnosis, chickenpox can be confirmed by testing a vesicle swab or oral fluid for VZV DNA by PCR. VZV IgG seroconversion is also possible by testing VZV IgG on a current blood and the earlier booking blood.

Management of Rash in Pregnancy

At booking, midwives should:

- Enquire if women have had a rash illness or had contact with a rash illness during the current pregnancy. Those with a recent rash should be investigated.
- Advise women to inform their midwife, GP or obstetrician urgently if they develop a rash at any time in pregnancy. They should be advised to avoid any antenatal clinic or maternity setting until clinically assessed, to avoid exposing other pregnant women.
- Advise women that they should inform their midwife, GP or obstetrician urgently if they have contact at any time in pregnancy with someone who has a rash. If a woman can give a history of chickenpox or shingles and has had contact with either of these during pregnancy she can be offered reassurance that she is not at any risk.
- Appropriate referral and investigation should be initiated for women with uncertain or no known history of chickenpox.
- All pregnant women with rash illness, or contact with rash illness, should be referred for medical management and laboratory investigation.
- Before any testing or screening is undertaken women should be provided with information regarding screening and diagnostic tests, the meaning and consequences of both, what to expect in terms of results and further options for management.

When any diagnostic testing is undertaken it should be made clear to the woman that:

- Tests to establish the initial diagnosis will usually be on samples of blood.
- The requirement for more invasive tests such as amniocentesis, is uncommon, and is only required in rare situations as advised by a specialist.
- Further testing may be necessary in order to confirm the diagnosis, which may prolong the time to result.
- If investigation is commenced some weeks after rash or contact, it may not be possible to confirm or refute a particular diagnosis.

Decisions on management of the pregnant woman diagnosed with any of the infections potentially causing congenital pathology in her first 20 weeks of pregnancy are best made in a specialist fetal medicine unit to enable patient access to counselling, serial ultrasound scanning and further follow up, investigations and treatment, where appropriate, should ultrasound be abnormal.

If patient does not have systemic illness then treat with emollients, steroids and antihistamines as in the guideline according to diagnosis and obtain Dermatological opinion if needed.

Dermatology contact details

A junior doctor is available Mon-Fri 9-5 telephone 07971 255779. If there is no reply, the alternative is the dermatology SpR via switchboard 8:30-00:00 weekdays, 8:30-22:30 weekends and consultant outside of these hours for emergencies only.)

3. Training and Education:

None

4. Supporting References:

HPA Guidance on Viral rash in pregnancy 2011 updated July 2019
<https://www.gov.uk/government/publications/viral-rash-in-pregnancy> (last accessed 01/11/2021)

DermNet NZ – Skin changes in pregnancy <http://www.dermnetnz.org/topics/skin-problems-in-pregnancy/Handbook of Obsteric Medicine – Catherine Nelson Piercy>

Health Protection Agency (HPA) 2011 “Guidance on Viral rash in Pregnancy”.

5. Monitoring Compliance

None

6. Keywords:

Skin Itching Rash Pregnancy

**The Trust recognises the diversity of the local community it serves. Our aim therefore is to provide a safe environment free from discrimination and treat all individuals fairly with dignity and appropriately according to their needs.
As part of its development, this policy and its impact on equality have been reviewed and no detriment was identified.**

CONTACT AND REVIEW DETAILS	
Guideline Lead (Name and Title): S Agarwal – Consultant Obstetrician I Helbling – Consultant dermatologist	Executive Lead: Chief Nurse
Details of Changes made during review: Dermatology contact details updated Updated treatment options for pruritic folliculitis –removed topical antimicrobials	

APPENDIX: RED RASH IN PREGNACY

Condition	Virus	Incubation Period	Appearance	Transmission
Rubella	Rubella virus	Usually 16 - 18 days (Range 14 - 23 days)	Rash lasts 3 days, lymphadenopathy up to 1 week and joint pains up to 3 weeks.	Person to person through respiratory secretions. Congenital
Fifth Disease	Erythrovirus (parvovirus) B19	4 - 14 days	7 - 10 days from onset of rash, polyarthralgia can last from 3 weeks to over 2 years	Person to person by respiratory secretions, fomite, blood products. Congenital
Rubeola	Measles virus	10 - 12 days	4 days before rash to 4 days after	Respiratory, aerosol, and fomites
Roseola	Human herpes virus -6	10 - 14 days	Fever lasts 3 - 5 days then rash develops	Saliva - from mother to infant; from child to child. Intra uterine (rare)
Hand Foot and Mouth	Enteroviruses eg Coxsackie virus A16	3 – 7 days	7 - 10 days after onset of symptoms	Person to person via nose and throat discharges, saliva, blister fluid or faeces
Infectious mononucleosis	Epstein-Barr virus	1 - 2 months	1 - 4 weeks (increases with age)	Close contact –aka 'kissing disease' and Glandular Fever
HIV Infection	Human immunodeficiency virus	1 – 4 weeks after exposure	One study found the average length of ARS symptoms to be about 22 days.	Sexual transmission Bloodborne
Arbovirus Infection	Arbovirus: Alphaviruses		5 – 10 days dependent on	Mosquito

	Chikungunya Ross River virus Sindbis Flaviviruses Dengue West Nile Fever Yellow fever Bunyaviruses LaCrosse encephalitis Reoviruses		virus, inability to work for up to 3/12 and rheumatic symptoms for up to 1 year	
Rickettsial infection	Rickettsia African Tick Bite fever Mediterranean Spotted fever Scrub typhus	Within 2 weeks of bite	5 – 10 days dependent on virus	Tick bites
Syphilis	Treponema pallidum	Secondary syphilis appears 3 - 6 weeks after primary infection.	May persist for weeks to months	Sexual contact
Scarlet Fever	Bacterial infection (streptococcal)	Rash appears within 12 - 48 hours of the sore throat and prodromal symptoms	Skin rash fades after 6 – 7 days, but skin flaking and peeling may continue for 10 - 14 days.	Respiratory and droplet spread

Appendix 2: Images of rashes in pregnancy



POLYMORPHIC ERUPTION OF PREGNANCY



FOLLICULITIS



PRURIGO OF PREGNANCY



PEMPHIGOID GESTATIONIS