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ESSENTIALS Dermatoses in pregnancy are common, they may be very itchy and may impact the life of a pregnant woman dramatically. There are four classical dermatoses of pregnancy. It is particularly important to recognize these as they may have serious health implications for mother and child. Intrahepatic cholestasis of pregnancy—occurs in 1/40 to 1/500 pregnancies and is the most serious cause of itch in pregnancy, with potentially substantial effects on mother and fetus. Treatment is with ursodeoxycholic acid. Atopic eruption of pregnancy—affects 1/300 pregnancies, typically with an eczematous eruption over abdomen and limbs. Treatment is with topical steroids. Polymorphic eruption of pregnancy—affects about 1/240 preg- nancies, usually beginning with red papules and plaques on the abdomen and thighs before spreading more widely. Treatment is with reassurance and emollients, with topical steroids if neccessary. Pemphigoid gestationis—occurs in 1/50 000 pregnancies and is due to circulating antibodies against the skin basement mem- brane zone. The eruption often begins around the umbilicus and spreads to the whole trunk, limbs, hands, and feet. If potent topical steroids fail systemic steroids are required. Transplacental transmis- sion to the fetus may occur. Recurrence in future pregnancies is to be expected. Introduction The skin undergoes profound alterations during pregnancy as a result of endocrine, metabolic, and physiological changes. Some of these are trivial and chiefly cosmetic, producing no or minor symptoms; some will improve during or after pregnancy and others can be distressing and/or of major medical import- ance. Pregnancy will profoundly modify expression of pre- existing skin disease, and there are dermatoses that are specific to pregnancy. Common skin changes in pregnancy Vascular changes and lesions There is increased skin blood flow during pregnancy, possibly re- sulting in oedema (e.g. manifest as tightening of rings and shoes), erythema, and itch. Spider naevi and palmar erythema are common, and there maybe erythema of the gums (with gingivitis) and the vulvovaginal area. Unilateral telangiectasia may appear for the first time, as may haemangiomas. Varicose veins may develop during pregnancy. Swollen skin around the ankles may be a first sign, worsening during the day and improving over night when lying down. Supportive stockings or flight socks should be worn: special pregnancy types are avail- able. If there is a family or personal history of the development of varicose veins or thrombophlebitis/phlebothombosis, further risk

assessment for venous thromboembolism is required. Pyogenic granuloma, a benign tumour with a tendency to ul- cerate and bleed, may develop on the skin or oral cavity, where they are known as pregnancy tumours (Fig. 14.13.1). They are 14.13 The skin in pregnancy Gudula Kirtschig and Fenella Wojnarowska Fig. 14.13.1 Pyogenic granuloma on the finger. Courtesy of Dr Jonathan Bowling, Oxford Radcliffe Hospital NHS Trust, UK.

14.13 The skin in pregnancy 2649 sometimes confused with melanoma and often recur after local destruction. Pigmentary changes and pigmented lesions Increased skin pigmentation is common, particularly in dark- skinned women, up to 90% of whom may be affected. There is darkening of the nipples, genitalia, and linea alba. In some women recent scars will darken. The unsightly and sometimes psychologic- ally distressing facial pigmentation of melasma (chloasma, formerly known as the ‘mask of pregnancy’) affects many women. It gets worse with sunlight and can be reduced by using high protection factor (SPF 50) UVB and UVA sunscreens (Fig. 14.13.2). Melasma often disappears spontaneously after delivery, but treatment with topical vitamin A derivatives and hydrochinon may be indicated after pregnancy in some women. Pigmented naevi can increase in size, in particular around the abdomen due to the increased body circumference, and pigmenta- tion during pregnancy. Any asymmetrical change is suspicious of malignant change. Melanoma may occur and is not associated with a poorer prognosis in pregnant women. Any rapidly changing, ir- regularly shaped, or irregularly pigmented lesion larger than 6 mm in diameter should be excised under local anaesthesia to exclude a dysplastic naevus or melanoma. Risk factors for developing a mel- anoma are fair skin, high density of freckles, red hair, more than 50 moles, the presence of more than five atypical moles (irregular shape and colour), a history of severe sunburns during childhood, espe- cially with blistering, and a family history of melanoma. Hair changes There is diminished shedding of hair due to prolongation of the anagen phase. This is perceived as thickening of the hair. The synchronized shedding after parturition gives rise to the distressing post-partum telogen effluvium three months after delivery. This is completed 6–12 months later and treatment is not needed. Hirsutism may begin or worsen in pregnancy, driven by an increase in androgens and usu- ally resolving a few months after delivery. Pilosebaceous changes/acne The development of acne during pregnancy is unpredictable. Skin in pregnancy is often more greasy, termed ‘pregnancy glow’. The increase in oestrogens usually improves acne, but there may be worsening of acne in some unfortunate patients. Acne treatment in pregnancy may be tricky as topical and oral vitamin A derivatives must be avoided because they are teratogenic. Topical treatment with benzoyl peroxide and clindamycin for limited areas (most com- monly the face) are the treatments of choice in pregnancy. Striae gravidarum Striae gravidarum (stretch marks) are common in pregnancy, af- fecting about 50% of women. They are more frequently seen in young women, in women with a raised body mass index, and those who have large babies. They are familial in about 50% of cases and are more likely if a woman has had them previously. The breasts and sides and lower areas of the abdomen are the typical sites, but thighs and arms can be affected. They start as linear depressed purple lines and fade to pale, atrophic, scar-like lesions. They may be itchy. There is an associ- ation with subsequent tendency to prolapse. There is no good treat- ment. Olive oil massage, castor oil, cocoa butter, glycolic or fruit acids, homeopathic creams and/or oils are used, but the benefit of these is not proven. Pruritus Itching occurs in about 20% of pregnancies, frequently in associ- ation with an inflammatory dermatosis such as atopic eczema, poly- morphic eruption of pregnancy, allergic reactions, or infectious diseases. The underlying dermatosis must be treated. Oral antihista- mines such as loratadine are safe to be used in pregnancy and can be used for symptomatic relief (see

Table 14.13.1). Pruritus may occur without physical signs, other than scratch marks. The most serious cause is intrahepatic cholostasis of pregnancy, which is diagnosed in about 3% of itchy pregnant women (see below and Chapter 14.9). If no underlying dermatosis exists emollients and antihistamines may be useful. Urticaria Urticaria (hives) and dermographism (wealing in response to pres- sure, e.g. scratching) may be precipitated by pregnancy and are very itchy conditions. Urticaria has been attributed by some authors to physiological changes in vascular reactivity. Physical factors such as Fig. 14.13.2 Melasma. Courtesy of Dr Christina Ambros-Rudolph, University of Graz, Austria. Table 14.13.1 Antihistamines safe to be used in pregnancy Group Generic name Sedating useful at night if pruritus prevents
sleep Chlopheniramine, chlorphenamine Clemastine Dimetinden Nonsedating treatment of choice Cetirizine Loratadine

 pressure and heat may evoke it. Particular drugs or foods may be the cause in some patients and must be avoided in such cases. Treatment with a nonsedating antihistamine such as loratidine, cetirizine or sedating chlopheniramine is safe. Cutaneous infections Candida of the vulva as well as the vagina is common and oc- curs in about 15% of pregnant women, causing itching, burning, and discharge. During pregnancy, treatment with miconazole or clotrimoxazole cream or vaginal pessaries is preferred and may need to be repeated several times or preventative treatment may be neces- sary. Oral antiyeast treatments must not be used for vulvo-vaginal yeast infection during pregnancy. Dermatophyte infections (tinea/ringworm) are common and may affect pregnant women. They typically manifest interdigitally (athlete’s foot) or in the groin, but can affect any body site including the nails. In uncomplicated cases tinea is usually treated with top- ical antifungals; in pregnancy, clotrimazole and miconazole are preferred. Oral antifungals must be avoided in pregnancy and treat- ment for onychomycosis postponed until after delivery. Cutaneous and genital warts thrive in pregnancy, often commen- cing, proliferating, or enlarging. Treatment for genital warts should be started as soon as possible. However, in the last eight weeks of pregnancy methods that destroy the warts and harm the skin over large areas should be avoided so there is no damage to the skin be- fore delivery. The choice of the therapy is dependent on the type, the extent, and the location of the warts. Localized lesions can be treated with freezing (cryotherapy), electro surgery or with trichloracetic acid (TCA, 33–50%), which is a liquid that ‘burns’ or ‘peels’ the warts away and can be applied to the lesions with a cotton tip by a phys- ician once every one to three weeks. Imiquimod has been used in pregnancy without observed adverse effects, but it is not licensed for use in pregnancy. Podophyllin or 5-Fluorouracil must not be used in pregnancy. Genital herpes simplex infections during pregnancy can affect the unborn child. The baby can catch the virus by transmission from the mother via the placenta or during delivery. If the baby is infected be- fore delivery it is at risk of abnormalities, mainly of the brain and the eyes, but herpes virus transmission predominantly occurs during delivery and not during pregnancy. The risk for infection depends mainly on the severity and timing of the mother’s infection (highest if the mother is very ill with herpes, or the baby is premature). If the baby is infected by the virus during delivery or as a newborn the infection may be restricted to the skin, mucosa, and/or the eyes (45%), the infection may involve the brain (30%), or the infection may be wide spread involving many organs including lungs, liver, and the brain (25%). The risk of transmission from the mother to the baby at delivery is highest (30–50%) among women who ac- quire genital herpes (primary herpes infection) near the time of de- livery (within six weeks). In a primary infection during the first or the second trimester of pregnancy aciclovir or valaciclovir may be used, depending on the severity of the

disease. Antiviral treatment may be used for four weeks before delivery to prevent recurrences and viral shedding around delivery; a caesarean section is usually not indicated. Primary herpes infection during the third trimester must be treated with aciclovir or valaciclovir. A caesarean section in pregnant women developing a primary infection in the six weeks preceding delivery and in women with recurrent disease if they have lesions at the time of delivery is controversially discussed, but there is no certain reduction of the infectious risk for the baby. Lice may be seen as head lice or pubic/crab lice. A very safe and effective treatment for head lice is combing with dimeticon or, al- ternatively, coconut oil or vinegar water. For easier combing, con- ditioner (possibly containing tea tree oil) may be used. Malathion (lice resistance is reported) or pyrethrum extract and synthetic pyrethroids (permethrin topical 5% cream/scalp treatment) are the treatment of second choice. Pump sprays should be avoided because of the danger of systemic intake through the air. Pubic lice may be treated with malathion or permethrin 5% cream applied to the affected site. Scabies is a common and very itchy skin condition caused by human scabies mites. It can affect people of any age but is most common in the young. Itching is the main symptom, usually starting about a month after the mites were picked up. The itching affects the body and limbs but usually spares the head and neck, except in infants. The rash of scabies is a mixture of scratch marks and red scaly areas; later it can become superinfected. This itchy rash covers much of the skin, but the mites themselves show up mainly where they burrow, typically on the sides of the fingers and hands, and around the wrists, ankles, feet, breasts, and genitals. Usually several members of a family are affected and need to be treated. Permethrin seems more effective than other treatments, but there are no studies that prove absolute safety in pregnancy. Permethrin 5% cream is ap- plied all over the body, except the head, and washed off after about 12 hours; re-treatment of hands if washed with soap in between is recommended. Taking a bath or shower before treatment is not re- commended. The treatment should be repeated after seven days. Benzyl benzoate, malathion, and crotamiton seem less effective but are considered safe. Oral Ivermectin (200 microgr/kg body weight in one dose) is not recommended during pregnancy, however, harm to the baby after accidental use is not reported. The pregnancy dermatoses Historical perspective The striking blistering eruption known as ‘pemphigoid gestationis’ was described in 1867 by Wilson and named by Milton in 1872 as ‘herpes gestationis’. During the 1980s it was characterized as an auto- immune blistering disease by Black, Charles-Holmes, and Shornick, and renamed as ‘pemphigoid gestationis’ to emphasize the close relationship to the commoner autoimmune blistering disease bul- lous pemphigoid and to prevent confusion with viral herpes disease. Further skin diseases that arise in pregnancy have been confusing in their nomenclature and clinical descriptions, but recently Ambros- Rudolph and colleagues proposed a new and much simpler classifi- cation (Table 14.13.2). Intrahepatic cholestatis of pregnancy It is particularly important to recognize itch/pruritus due to intrahepatic cholestasis of pregnancy (obstetric cholestasis, cholestasis of pregnancy, and pruritus/prurigo gravidarum), which has important implications for the health of both mother and fetus (see Chapter 14.9). It is the most serious cause of itch in pregnancy.

 In Europe about 0.2–2.4% of pregnant women will get the condi- tion; in Scandinavia and South America intrahepatic cholestatis of pregnancy is more common and it may also occur in women on the oral contraceptive pill. The itching begins typically in the third tri- mester and affects the abdomen, palms, and soles. The longer the itch persists, the more skin changes due to scratching may be present. Excoriations and prurigo nodules typically involve the shins, arms, and buttocks. Apart from these changes, there is usually no rash as- sociated with

intrahepatic cholestatis of pregnancy. Loss of sleep, loss of appetite, and an inability to perform normal daily tasks can be a result of the intense itching. Less common symptoms include dark urine and/or pale stools, jaundice, abdominal pain, and nausea. Liver function tests may be normal, but bile salts are typically raised. Other causes for itchy skin such as hepatitis, iron deficiency, specific derma- toses of pregnancy, or infectious causes should be excluded. The condition resolves post-partum but will recur in subsequent pregnancies. Reducing the bile acids is essential. Ursodeoxycholic acid (UDCA—a naturally occurring bile acid) is currently the best treatment for intrahepatic cholestatis of pregnancy. It is not licensed for use in pregnancy but may be prescribed on an individual basis. It improves liver function and helps to reduce the toxic bile acid concentration, and it is the only treatment that has been shown to reduce fetal risks in intrahepatic cholestatis of pregnancy. UDCA tablets, 15 mg/kg/day or simply 1 g daily, are given either as a single dose or divided into two to three doses and continued until delivery, when treatment can usually be stopped. Symptomatic management is with emollients and sometimes antihistamines.
Atopic eruption of pregnancy This condition includes entities formerly known as prurigo of preg- nancy and pruritic folliculitis. It may affect 1 in 300 pregnancies. It oc- curs in women with an atopic background (personal or family history), of whom about 20% have had previous eczema. The immunological changes of pregnancy and the tendency to pruritus may both contribute to the worsening of atopic eczema or its first occurrence with preg- nancy. Atopic eruption of pregnancy thus affects women who already have atopic eczema but experience a flare-up of the disease, and women with their first occurrence of eczema during pregnancy (80%). It can be severe and life-ruining, and life-threatening if secondary infection with herpes simplex (eczema herpeticum) or streptococci occurs. Atopic eruption of pregnancy commences early, in three-quarters of women before the beginning of the third trimester. There is in- tense pruritus, it typically presents with an eczematous eruption over abdomen and limbs (Fig. 14.13.3). The lesions can be chiefly eczematous with red, dry, and scaly skin, with areas of excori- ation and thickening or lichenification. Pre-existing atopic eczema often deteriorates becoming more widespread and may result in erythroderma in the most severe cases. Another presentation is with excoriated papules and nodules (prurigo of pregnancy). The least common form is follicular pruritic papules and pustules (prur- itic folliculitis), which may present in the third trimester and in a small series was associated with male infants and low birth weight. Secondary infection with Staphylococcus aureus and streptococci is a frequent complication. Histopathology is usually nonspecific, but may show a perivascular infiltrate with thickened epidermis. Direct and indirect immunofluorescence are negative. Treatment is with moderate to potent topical steroids that al- though absorbed do not adversely affect the fetus. The use of emollients may lessen the requirements for topical steroids, and steroids should be used in the minimum quantities and strengths necessary to control the disease (see Table 14.13.3). Many topical Table 14.13.2 Four major pregnancy dermatoses (numbers are based on 505 pregnant patients with skin problems;
Ambros-Rudolph et al. 2006) Pregnancy dermatosis Frequency (%) Effect on fetus Effect on mother Intrahepatic cholestatis of pregnancy 3 Can be substantial (see Chapter 14.9) Can be substantial (see Chapter 14.9) Atopic eruption of pregnancy 50 None described Usually improves after delivery Polymorphic eruption of pregnancy 22 None described Pemphigoid gestationis 4a Small for dates May be major, usually resolves months after delivery a Raised frequency as tertiary referral centre.
Fig. 14.13.3 Atopic eruption of pregnancy in a 24-year-old gravida 2 at 19 weeks' gestation: small red pruritic papules and eczematous features on the trunk (and limbs). Courtesy of Dr Christina Ambros-Rudolph, University of Graz, Austria.

Section 14 Medical disorders in pregnancy 2652 steroids contain antiseptics and antibiotics that will be absorbed, and some may be contraindicated in pregnancy. The sedating anti- histamine chlorpheniramine may help with sleep. Secondary infec- tion often requires systemic antibiotics such as oral penicillins or erythromycin. The condition resolves in days to weeks after delivery. It may recur in one-third of pregnancies. Polymorphic eruption of pregnancy Polymorphic eruption of pregnancy was formerly known as ‘pruritic urticated papules and plaques of pregnancy’ or ‘toxic erythema of pregnancy’. Its aetiology is unknown, but there is an association with a low serum cortisol. It affects 1 in 240 singleton pregnancies, being most common in first pregnancies, with multiple births (hence fol- lowing in vitro fertilization)—perhaps related to the mechanical ef- fect of the abdominal stretching or to an increased immune complex load—and with a male fetus. This condition usually begins in the third trimester and occasion- ally post-partum. Red papules and plaques typically begin in striae on the abdomen and thighs and then spread to the whole trunk and limbs, including the hands and feet. They are very itchy, and the itching can be so severe as to prevent sleep. Initially the lesions are raised red papules (Fig. 14.13.4) and plaques; with time they become more diverse in morphology, occasionally polycyclic or blistering. The histopathology shows oedema, perivascular lymphocytes, and eosinophils. Immunofluorescence does not demonstrate any circulating or bound immunoreactants. Treatment is with reassurance and emollients (e.g. cold cream containing 1–2% menthol). This is helpful, but not always sufficient. Antihistamines and moderate to very potent topical steroids, which may be absorbed through the skin (see Table 14.13.3), may be re- quired, and occasionally systemic steroids for induction of remission. The condition resolves over days to weeks after delivery. It does not usually recur. The outcome of the pregnancy is not adversely affected. Pemphigoid gestationis Pemphigoid gestationis, formerly herpes gestationis (a name best abandoned as ‘herpes’ refers to the herpetiform grouping of the blis- ters rather than herpes infection), is an autoimmune blistering disease characteristically occurring in pregnancy. Pemphigoid gestationis is the most severe of the pregnancy dermatoses. It is due to circu- lating antibodies against adhesion molecules of the skin basement membrane zone. Very potent topical or systemic steroids are usu- ally required. Transplacental transmission to the fetus may occur. Recurrence in future pregnancies is to be expected. The aetiology is only partially understood. The pathogenicity of the circulating basement membrane zone antibodies is demon- strated by transplacental transmission of the disease. The major target antigen is BP180/collagen XVII (chief epitope being the trans- membrane NC16A domain); BP230 is a less common antigen. Both antigens are present in skin, mucosa, and amnion, associated with the hemidesmosome and adhesion complex linking epithelium to dermis/mesenchyme, which are targets in other autoimmune blis- tering diseases. The placenta shows increased expression of antigen- presenting cells, but it is unclear why breakdown of tolerance occurs, and why normal components of amnion and stratified squamous epithelium become antigenic. The mothers have the HLA DR 3, 4, haplotype and are C4 null, and there is an association with thyroid and less commonly other autoimmune disease. Pemphigoid gestationis occurs in approximately 1 of 50 000 pregnancies. It commences from the second trimester onwards and quite often in the first week post-partum (range from five weeks of gestation to four weeks post-partum). It usually occurs in the first and subsequent pregnancies, although 8% of pregnancies are skipped. The eruption typically begins around the umbilicus and spreads to the whole trunk, limbs, hands, and feet, including the palms and soles, and rarely the face. The mouth and vulva may be involved showing blisters or erosions. Vesicles and blisters are character- istic, but lesions comprise annular lesions, papules, and plaques (Fig. 14.13.5). Pruritus is severe and sleep often impaired. Table 14.13.3 Examples of topical steroids. Prolonged treatment with very

potent topical steroids may lead to fetal growth
restriction; see guidelines for the use of topical steroids in pregnancy Group Generic name Mild Hydrocortisone 1% Moderately potent Hydrocortisone 1% with urea Clobetasone butyrate 0.05% Flurandrolone Potent Betamethasone valerate 0.025%/0.1% Betamethasone dipropionate Hydrocortisone 17-butyrate Fluticasone propionate Mometasone furoate Very potent Clobetasol propionate 0.05% Fig. 14.13.4 Polymorphic eruption of pregnancy: urticated papules and plaques on the thigh.

14.13 The skin in pregnancy 2653 Transplacental transmission of antibodies to the fetus occurs in about 3% of affected pregnancies, the neonate developing transient self-limiting blistering (Fig. 14.13.6). Histopathology demonstrates an eosinophilic infiltrate, papil- lary oedema, and subepidermal blisters. Direct immunofluores- cence demonstrates that C3 component of complement and IgG1 are bound at the basement membrane zone of the dermoepidermal junction. The patient's serum has circulating IgG1 basement mem- brane zone antibodies that bind C3. These immunoreactants are also found at the basement membrane zone of the amnion (Fig. 14.13.7). Treatment with potent or very potent topical steroids and chlorpheniramine is recommended, however, systemic steroids (e.g. prednisolone 0.3–0.5 mg/kg body weight daily) may be required, the dose adjusted according to disease activity. There is usually a post- partum flare, necessitating increased steroids. Azathioprine and Ciclosporin may reduce steroid requirement in selected cases. The disease slowly resolves post-partum, but may persist for sev- eral months. Recurrence in subsequent pregnancies is usual, only about 8% being spared. The classical teaching is that it recurs earlier and is more severe in subsequent pregnancies, but this has not al- ways been our experience. Onset of pemphigoid gestationis in the first or second trimester and presence of blisters may lead to adverse pregnancy outcomes, including decreased gestational age at delivery, preterm birth, and children with low birth weight. Such pregnancies should be considered high risk and appropriate obstetric care should be provided. Systemic corticosteroid treatment, in contrast, does not substantially affect pregnancy outcomes, and its use for pemphigoid gestationis in pregnant women is justified. Classical dermatoses affecting pregnant women Psoriasis Psoriasis improves in most women during pregnancy, but can de- teriorate. Therapy poses special problems as most systemic treat- ments are contraindicated. Methotrexate is a folic acid antagonist and can cause miscarriage; acitretin is teratogenic; fumaric acid causes leukopenia (whether this affects the fetus is unknown, but case reports have shown no harm). Oral psoralens with UVA (PUVA) are still not proven to be safe, but topical PUVA and UVB light treatment is safe. Ciclosporin and tumour necrosis factor (TNF) α inhibitors are reserved for severe cases. Topical therapy with steroids can be used if needed. Coal tars and dithranol have been widely used in pregnancy but are not proven to be safe. The new vitamin D analogues are not licensed for use in pregnancy, but there is no hint from case reports that indicate harm if used in usual doses. The ideal is minimum treatment, encouraging emollient use and if necessary UVB.
A severe form of pustular psoriasis, impetigo herpetiformis, may occur in pregnancy and is best managed with bed rest, emollients, and moderate potent topical steroids or low doses of oral prednisolone. In severe cases topical PUVA or oral Ciclosporin are used, and induction of labour may be indicated if the mother is at risk. Cutaneous lupus erythematosus Cutaneous lupus erythematosus may be adversely affected or im- proved or unchanged by pregnancy. However, such patients should be screened for anti-Ro and anticardiolipin antibodies, preferably prior to conception, to identify at-risk pregnancies (see Chapter 14.14). Fig. 14.13.5 Pemphigoid gestationis: urticated papules and blisters. From Charles-Holmes R, Black MM (1990). Herpes

gestationis. In: Wojnarowska F, Briggaman RA (eds) Management of blistering disease, pp. 93–104. Chapman & Hall, London, with permission. Fig. 14.13.6 Pemphigoid gestationis: urticated papules in the neonate. From Charles-Holmes R, Black MM (1990). Herpes gestationis. In: Wojnarowska F, Briggaman RA (eds) Management of blistering disease, pp. 93–104. Chapman & Hall, London, with permission. Fig. 14.13.7 Pemphigoid gestationis: linear deposition of C3 at the amnion basement membrane zone as demonstrated by immunofluorescence. The nuclei are counterstained with propidium iodide. Courtesy of B.S. Bhogal and M.M. Black, St John's Institute of Dermatology, St Thomas's Hospital, London.

Section 14 Medical disorders in pregnancy 2654 Autoimmune bullous diseases Linear IgA disease, an autoimmune blistering disease with linear IgA basement membrane zone antibodies, usually improves with pregnancy, such that some patients can discontinue their therapy, usually dapsone. Despite the deposition of immunoreactants in the amnion basement membrane zone, the fetus is not ad- versely affected. There is usually an exacerbation three months post-partum. Pemphigus vulgaris is an autoimmune blistering disease with widespread mucosal and/or cutaneous erosions caused by IgG antibodies to desmosomal components of the epithelium. The desmosomal antibodies are directed at desmoglein 3, a major adhesion molecule in mucosa and neonatal skin, and can be transmitted across the placenta, causing severe neonatal pem- phigus with devastating results to the fetus. This does not occur in the related pemphigus foliaceus, which is endemic in Brazil, characterized by superficial cutaneous erosions and mediated by desmoglein 1 antibodies that do not cause oral lesions or af- fect neonatal skin. Both forms of pemphigus may worsen in pregnancy and treatment may require systemic steroids and im- munosuppressants like azathioprine. Spontaneous remission after pregnancy is described. Vulval dermatoses Many dermatoses may affect the vulval skin, and this may be par- ticularly distressing in pregnancy as concerns regarding delivery may arise. A disease commonly seen at the vulva is lichen sclerosus, a chronic inflammatory condition of unknown cause. This is usually treated with very potent topical steroids. Their use should be limited during pregnancy to a minimum, but the disease may improve during pregnancy and does not inhibit vaginal delivery, although episiotomy may be required and should be anticipated. FURTHER READING Ambros-Rudolph CM, et al. (2006). The specific dermatoses of preg- nancy revisited and reclassified: results of a retrospective two- center study on 505 pregnant patients. J Am Acad Dermatol, 54, 395–404. Chi CC, et al. (2009). Pemphigoid gestationis: early onset and blister formation are associated with adverse pregnancy outcomes. Br J Dermatol, 160, 1222–8. Chi CC, et al. (2015). Safety of topical corticosteroids in preg- nancy. Cochrane Database Syst Rev. Oct 26;(10):CD007346. doi:10.1002/14651858.CD007346.pub3. Review. European Academy of Dermatology and Venereology. Patient Information Leaflets. http://www.eadv.org/patient-corner/leaflets/ Jenkins RE, Hern S, Black MM (1999). Clinical features and management of 87 patients with pemphigoid gestationis. Clin Exp Dermatol, 24, 255–9. Kirtschig G, Cooper S (2016). Gynaecologic dermatology: symptoms, signs and clinical management. Jaypee Brothers, New Delhi. Kirtschig G, Schäfer C (2015). Dermatological medications and local therapeutics. In: Schaefer C, Peters P, Miller RK (eds) Drugs during pregnancy and lactation, 3rd edition. Elsevier, London,
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