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the SQUARE

Healthcare bulletin

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- Chikungunya: Re-Emerging Hazard
- Postmenopausal Mood Disorder
- Contact Dermatitis
- Hemophilia
- Poultry & Human Health

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the SQUARE
healthcare bulletin

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Editorial

Dear Doctor,

Hello again and welcome to this edition of *"the SQUARE"* healthcare bulletin!

In this issue, we have published a special feature on "Chikungunya: Re-Emerging Hazard". Chikungunya was first reported from Tanzania in 1952-53 and spread subsequently to sub-Saharan Africa, South East Asia and Pacific causing large epidemics. The recent re-emergence and travel-related spread of Chikungunya infection to Europe and the United States have drawn global attention. We have an article on "Postmenopausal Mood Disorder", one of the more exasperating symptoms of the menopause. Not only can menopause prompt uncomfortable physical symptoms, it can also turn a woman's emotions into an out-of-control pendulum, by afflicting her with mood swings. We bring the details on "Contact Dermatitis", an inflammatory condition of the skin encountered by the physicians very often. We also focused on "Hemophilia", an inherited disorder. There's no cure yet. But with proper treatment and self-care, most people with hemophilia can maintain an active, productive lifestyle. In addition, we have highlighted on "Poultry & Human Health", which is at present a growing concern for human health. Our regular feature "Test Yourself" also included in this edition.

We believe you will enjoy reading this publication and that the contents provided will prove helpful towards your goal of optimum health!

Wishing all of you a safe, healthy and peaceful life!

Omar Akramur Rab

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Chikungunya: Re-Emerging Hazard

Chikungunya fever is a self-remitting febrile viral illness that has been associated with frequent outbreaks in tropical countries of Africa and Southeast Asia. The illness has only recently become a concern in Western countries and temperate zones around the world. The recent re-emergence and travel-related spread of Chikungunya infection to Europe and the United States has drawn global attention. In fact, international travel stands out as one of the major risk factors for the rapid global spread of the disease.

The term "Chikungunya" often refers to both the virus (CHIKV) and the illness or fever (CHIKF) caused by this virus. It was derived from the African dialect Swahili or Makonde and translates as "to be bent over." In Congo, it is referred to as "buka-buka," which means "broken-broken." The name "chikungunya" derives from a word in the Kimakonde language, meaning "to become contorted" and describes the stooped appearance of sufferers with arthralgia. The roots of this viral illness date back to 1953, when it was first described during an outbreak in a Swahili village in the Newala district of Tanzania, Africa.

Signs and symptoms

Chikungunya is characterized by an abrupt onset of fever frequently accompanied by joint pain. Other common signs and symptoms include muscle pain, headache, nausea, fatigue and rash. The joint pain is often very debilitating, but usually lasts for a few days or may be prolonged to weeks. Hence the virus can cause acute, subacute or chronic disease.

Most patients recover fully, but in some cases joint pain may persist for several months or even years. Occasional cases of eye, neurological and heart complications have been reported, as well as gastro-intestinal complaints. Serious complications are not common, but in older people, the disease can contribute to the cause of death. Often symptoms in infected individuals are mild and the infection may go unrecognized, or be misdiagnosed in areas where dengue occurs.

Transmission

Chikungunya has been identified in over 60 countries in Asia, Africa, Europe and the Americas. The virus is transmitted from human to human by the bites of infected female mosquitoes. Most commonly, the mosquitoes involved are *Aedes aegypti* and *Aedes albopictus*, two species which can also transmit other mosquito-borne viruses, including dengue. These mosquitoes can be found biting throughout daylight hours, though there may be peaks of activity in the early morning and late afternoon. Both species are found biting outdoors, but *Ae. aegypti* will also readily feed indoors. After the bite of an infected mosquito, onset of illness occurs usually between 4 and 8 days but can range from 2 to 12 days.

Pathogenesis

At the early stage of the disease, the organs targeted for chikungunya virus replication were lymphoid tissues, liver, CNS, joints, and muscle, and the persistence of chikungunya virus could be found later in the lymphoid organs, liver, joints, and muscle, macrophages being the main reservoir. In humans, acute chikungunya virus infection is characterized by a very early viremia at fever onset that can increase up to 10^9 to 10^{12} RNA copies/ml and lasts up to 12 days. In vitro studies have shown that human epithelial and endothelial cells, primary fibroblasts, and monocyte-derived macrophages are susceptible to chikungunya virus infection, whereas activated B and T CD4+ lymphocytes, monocytes and monocyte-derived dendritic cells were refractory to chikungunya virus infection.

Disease outbreaks

Chikungunya occurs in Africa, Asia and the Indian subcontinent. Human infections in Africa have been at relatively low levels for a number of years, but in 1999-2000 there was a large outbreak in the Democratic Republic of the Congo, and in 2007 there was an outbreak in Gabon.

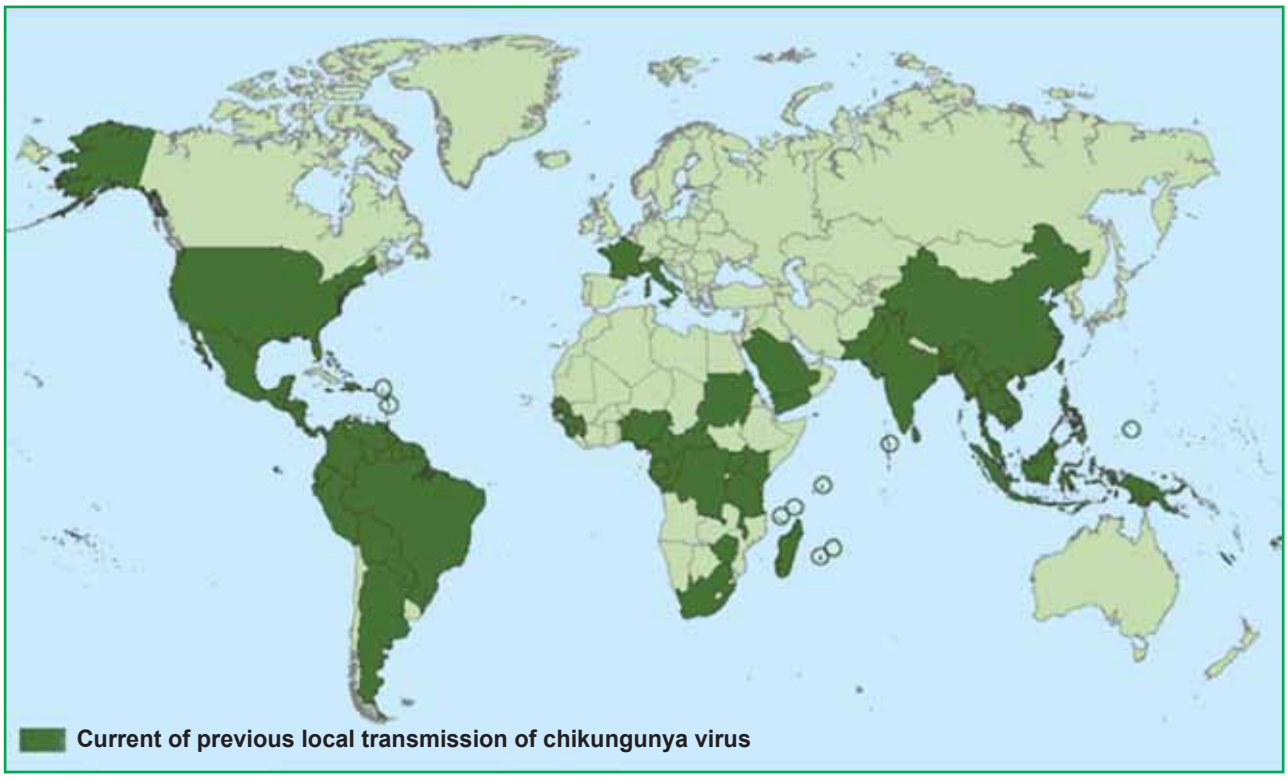
Starting in February 2005, a major outbreak of chikungunya occurred in islands of the Indian Ocean. A large number of imported cases in Europe were associated with this outbreak, mostly in 2006 when the Indian Ocean epidemic was at its peak. A large outbreak of chikungunya in India occurred in 2006 and 2007. Several other countries in South-East Asia were also affected. Since 2005, India, Indonesia, Maldives, Myanmar and Thailand have reported over 1.9 million cases. In 2007 transmission was reported for the first time in Europe, in a localized outbreak in north-eastern Italy. There were 197 cases recorded during this outbreak and it confirmed that mosquito-borne outbreaks by *Ae. Albopictus* are plausible in Europe.

In December 2013, France reported 2 laboratory-confirmed autochthonous cases in the French part of the Caribbean island of St Martin. Since then, local transmission has been confirmed in over 43 countries and territories in the WHO Region of the Americas. This is the first documented outbreak of chikungunya with autochthonous transmission in the Americas. As of April 2015, over 1 379 788 suspected cases of Chikungunya have been recorded in the Caribbean islands, Latin American countries, and the United States of America. 191 deaths have also been attributed to this disease during the same period. Canada, Mexico and USA have also recorded imported cases.

On 21 October 2014, France confirmed 4 cases of locally-acquired chikungunya infection in Montpellier, France. In late 2014, outbreaks were reported in the Pacific islands. Currently chikungunya outbreak is ongoing in Cook Islands and Marshall Islands, while the number of cases in American Samoa, French Polynesia, Kiribati and Samoa has reduced. WHO responded to small outbreaks of chikungunya in late 2015 in the city of Dakar, Senegal and the state of Punjab, India.

In the Americas in 2015, 693 489 suspected cases and 37480 confirmed cases of chikungunya were reported to the Pan American Health Organization (PAHO) regional office, of which Colombia bore the biggest burden with 356 079 suspected cases. This was less than in 2014 when more than 1 million suspected cases were reported in the same region.

In 2016 there was a total of 349 936 suspected and 146 914 laboratory confirmed cases reported to the PAHO regional office, half the burden compared to the previous year. Countries reporting most cases were Brazil (265 000 suspected cases), Bolivia and Colombia (19 000 suspected cases, respectively).



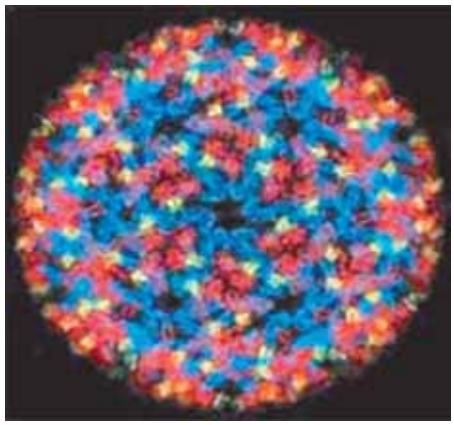
Chikungunya: Re-Emerging Hazard

2016 is the first time that autochthonous transmission of chikungunya was reported in Argentina following an outbreak of more than 1 000 suspected cases. In the African region, Kenya reported an outbreak of chikungunya resulting in more than 1 700 suspected cases. In 2017, Pakistan continues to respond to an outbreak which started in 2016.

Bangladesh found the first case in 2008 in northern Rajshahi and Chapainawabganj districts. In Dhaka, Institute of Epidemiology Disease Control and Research (IEDCR) noticed the presence of disease later in 2011. In 2017 Chikungunya outbreaks in Bangladesh mostly in the capital city Dhaka. Dhanmondi, Lalmatia and Kalabagan are the most affected areas. According to IEDCR 196 cases of Chikungunya has so far been confirmed from April to May 25, 2017.

The Chikungunya virus

Chikungunya virus is an Alphavirus with a positive sense single-stranded RNA genome of approximately 11.6kb2. The “molecular signatures” of chikungunya virus, which constitute genetic fingerprints, in virus envelope protein E1 have also been found. The scientists suggest that, this signature may confer an advantage and favour the multiplication of the virus in Aedes albopictus, its vector (protein E1 is in fact involved in attaching the virus to the mosquito's cell membranes). Genetic analysis showed three distinct lineages: the West African cluster, the East-Central and South African cluster (ECSA), and the Asian cluster. To date, no difference in virulence between the different strains of chikungunya virus has been shown in humans.



Chikungunya Virus

Evolution of Chikungunya virus

Phylogenetic analysis has revealed that the Chikungunya virus genome has remained stable over the years since its first discovery in 1952. Comparison of two Asian Chikungunya virus strains that were isolated 10 years apart showed 99.4% identity. Most of the early strains isolated from the Reunion Island Chikungunya virus outbreak were similar to the ECSA cluster. However, the strains isolated from the 2006-2007 outbreak revealed an alanine to valine amino acid mutation at position 226 in the E1 glycoprotein. The presence of alanine (A226) denotes cholesterol-dependent growth and replication of Chikungunya virus in certain Aedes species.

This particular mutation raised considerable interest in the research field, as it resulted in cholesterol-independent growth and replication of Chikungunya virus and was exclusively seen in Chikungunya virus isolated from Aedes albopictus mosquitoes. This mutation was also associated with enhanced fitness in Chikungunya virus, enabling it to infect *Ae. albopictus* mosquitoes and other species that normally lack cholesterol. This ability of the Chikungunya virus to adapt to a new species has significant implications with respect to the range of transmission and spread across the globe. The above findings led experts to speculate that Chikungunya virus would no longer be restricted to the tropical countries but could spread to temperate regions, as *Ae. albopictus* predominates in temperate zones where *Ae, aegypti* is scarce.

Overlap and confusion with Dengue Fever

Chikungunya fever has to be distinguished from dengue fever which has the potential for considerably worse outcomes including death. The two diseases can often be seen simultaneously in the same patient. Observations from previous outbreaks in Thailand and India have characterized the principal features distinguishing chikungunya from dengue fever. In the former, shock or severe haemorrhage is not observed. The onset is more acute and the duration of fever is much shorter in chikungunya fever. In chikungunya fever, maculopapular rash is more frequent than in dengue fever. In the early stage when rashes are absent, malaria has to be ruled out. With the presence of rashes, measles or German measles need to be ruled out.

Differential diagnosis with other arthropod-borne viruses of the Alphavirus genus (Ross River, Barmah Forest, O'nyong nyong, Sindbis and Mayaro viruses) is difficult, but these are comparatively rare.

Major manifestations that differentiate Chikungunya from Dengue :

Distinguishing features	Chikungunya fever	Dengue fever
Clinical signs and symptoms		
1. Onset of fever of 40°C	Acute	Gradual
2. Duration of fever	1-2 days	5-7 days
3. Maculopapular rash	Frequent	Rare
4. Presence of shock and severe haemorrhage	Rare	Common
5. Arthralgia	Frequent and lasting over a month	Infrequent and shorter duration
Laboratory parameters		
1. Leukopenia	Frequent	Infrequent
2. Thrombocytopenia	Infrequent	Frequent

Diagnosis

Several methods can be used for diagnosis. Serological tests, such as enzyme-linked immunosorbent assays (ELISA), may confirm the presence of IgM and IgG anti-chikungunya antibodies. IgM antibody levels are highest 3 to 5 weeks after the onset of illness and persist for about 2 months. Samples collected during the first week after the onset of symptoms should be tested by both serological and virological methods (RT-PCR).

The virus may be isolated from the blood during the first few days of infection. Various reverse transcriptase-polymerase chain reaction (RT-PCR) methods are available but are of variable sensitivity. Some are suited to clinical diagnosis. RT-PCR products from clinical samples may also be used for genotyping of the virus, allowing comparisons with virus samples from various geographical sources.

Treatment

There is no specific antiviral drug treatment for chikungunya. Treatment is directed primarily at relieving the symptoms, including the joint pain using anti-pyretics, optimal analgesics and fluids. Aspirin and other non-steroidal anti-inflammatory drugs (NSAIDS) should be avoided until dengue can be ruled out to reduce the risk of bleeding. There is no commercial chikungunya vaccine.

Prevention and control

The proximity of mosquito vector breeding sites to human habitation is a significant risk factor for chikungunya as well as for other diseases that these species transmit. Prevention and control relies heavily on reducing the number of natural and artificial water-filled container habitats that support breeding of the mosquitoes. This requires mobilization of affected communities. During outbreaks, insecticides may be sprayed to kill flying mosquitoes, applied to surfaces in and around containers where the mosquitoes land and used to treat water in containers to kill the immature larvae.

For protection during outbreaks of chikungunya, clothing which minimizes skin exposure to the day-biting vectors is advised. Repellents can be applied to exposed skin or to clothing in strict accordance with product label instructions. Repellents should contain DEET (N, N-diethyl-3-methylbenzamide), IR3535 (3-[N-acetyl-N-butyl]-aminopropionic acid ethyl ester) or icaridin (1-piperidinecarboxylic acid, 2-(2-hydroxyethyl)-1-methylpropylester). For those who sleep during the daytime, particularly young children, or sick or older people, insecticide-treated mosquito nets afford good protection. Mosquito coils or other insecticide vaporizers may also reduce indoor biting.

Basic precautions should be taken by people travelling to risk areas and these include use of repellents, wearing long sleeves and pants and ensuring rooms are fitted with screens to prevent mosquitoes from entering.

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Postmenopausal Mood Disorder

Menopause is the permanent cessation of menstruation resulting in the loss of ovarian follicle development. It is considered to occur when 12 menstrual cycles are missed.

Menopausal transition or perimenopause, is the period between the onset of irregular menstrual cycles and the last menstrual period. Postmenopause is the phase following the last menstrual period.

Mood disorders during menopause

Depression

Although most women transition to menopause without experiencing psychiatric problems, an estimated 20% have depression at some point during menopause. Studies of mood during menopause have generally revealed an increased risk of depression during perimenopause, with a decrease in risk during postmenopausal years.

The strongest predictor of depressed mood was a prior history of depression, along with fluctuations in reproductive hormone levels associated with depressed mood. Higher testosterone levels may directly lead to higher depressive symptoms during the menopausal transition. Menopausal status, however, remains an independent predictor of depressive symptoms.

Problems with sleep

Insomnia occurs in 40-50% of women during the menopausal transition. Women with insomnia are more likely than others to report problems such as anxiety, stress, tension and depressive symptoms.

Sleep disturbances during menopause have been associated with estrogen deficiency. Elevated LH levels during late menopause produce poor sleep quality through a thermoregulatory mechanism, resulting in high core body temperatures. Rates of sleep apnea increase with age, rising from 6.5% in women aged 30-39 years to 16% in women aged 50-60 years. The pathophysiology is not known, but theories include a relation to postmenopausal weight gain or to decreased progesterone levels (because progesterone stimulates respiration). Besides undergoing changes in estrogen and progesterone levels, postmenopausal women experience declines in levels of melatonin and growth hormone, both of which have effects on sleep.

Schizophrenia

In most cases, schizophrenia first manifests in young adulthood, with the rate of new cases declining in both male and female individuals after this time. A second peak in the incidence of schizophrenia is noted among women aged 45-50 years; this second peak is not observed in men.

Some researchers have observed a worsening of the course of schizophrenia in women during the menopausal transition. These observations suggest that estrogen may play a modulatory role in the pathophysiology of schizophrenia.

Panic disorder

Panic disorder is common during perimenopause. New-onset panic disorder may occur during menopause, or preexisting panic disorder may worsen. Panic disorder may be most common in women with many physical symptoms of menopause. These attacks were associated with negative life events, functional impairment, and medical comorbidity.

Obsessive-compulsive disorder

New-onset obsessive-compulsive disorder (OCD), a relapse of OCD, or a change in OCD symptoms may occur during menopause. Fluctuations in OCD have been correlated with the menstrual cycle and with pregnancy, suggesting that hormone levels may contribute to the disorder.

Bipolar disorder

Exacerbation of mood symptoms during menopause has been noted in women with preexisting bipolar disorder. Research has suggested that women with bipolar disorder have higher rates of depressive episodes during the menopausal transition.

The frequency of depressive episodes in this population appears to be higher than during premenopausal years. Earlier studies suggested an increase in rapid cycling during the menopausal transition; however, this finding has not been reproduced.

Pathophysiology

Depression during perimenopause is likely due to fluctuating and declining estrogen levels in part.

Steroid hormones, such as estrogen, act in the central nervous system (CNS) by means of various mechanisms. For instance, they stimulate the synthesis of neurotransmitters, the expression of receptors, and influence membrane permeability.

Estrogen increases the effects of serotonin and norepinephrine, which are the neurotransmitters most related to depression by decreasing monoamine oxidase (MAO) activity in the CNS, increasing serotonin synthesis by up-regulating 5-HT1 receptors & down-regulating 5-HT2 receptors and lastly increasing norepinephrine activity in the brain.

Because estrogen facilitates the actions of serotonin and norepinephrine, a decline in estrogen concentrations may, in turn, decrease levels of these hormones and may be related to depressive symptoms in the menopausal transition of some women.

Etiology

Causes of menopause-related mood disorders may include hormonal changes, life stressors, psychological or social conditions, and a preexisting tendency toward depression.

Hormonal changes

Observations and data suggest that depression is significantly linked to times of hormonal change in women. For example, the disparity in depression rates between women and men begins at puberty. Also, hormonal changes are thought to contribute to premenstrual dysphoric disorder, as well as mood changes post-partum and in perimenopause.

Furthermore, estrogen affects both serotonin and norepinephrine, the 2 neurotransmitters thought to be most directly associated with depression. However, absolute levels of gonadal hormones are not correlated with depression. Estrogen and progesterone levels do not distinguish a woman with depression from one without depression.

When hormone concentrations were measured in perimenopausal or postmenopausal women with depression, no abnormal levels were found. Rather, a certain subset of women seem to be predisposed to experience mood disturbances triggered by hormonal fluctuations. This subset includes women with a history of mood disorders or of premenstrual and postpartum mood-related symptoms. The risk of depression appears to be higher during perimenopause, when hormone levels are changing, than during postmenopause, when estrogen and progesterone levels are low but stable.

Life stressors

Societal roles and expectations may contribute to the heightened rate of depression in women. Women with particular types of stressors seem to be at increased risk for perimenopausal depression. Such stressors include the following:

- ❏ Lack of social support
- ❏ Unemployment
- ❏ Surgical menopause
- ❏ Poor overall health status

Dysphoric mood during the early perimenopausal transition is most common in women with relatively low educational status. Therefore, low levels of education may be a marker for other stressors, such as ongoing low socioeconomic status.

An Australian study of women transitioning to menopause revealed more depression in women with the following states:

- ❏ Negative mood before menopause
- ❏ Negative attitude toward menopause and aging
- ❏ Smoking
- ❏ Little or no exercise
- ❏ No partner
- ❏ A number of bothersome symptoms
- ❏ Poor self-perceived health
- ❏ Negative feelings toward partner
- ❏ A number of perceived problems
- ❏ Interpersonal stress

Other stressors that tend to correspond with perimenopause and that are postulated to relate to depression include the following:

- ❏ Onset of illness in self or others
- ❏ Care of aging parents
- ❏ Changes in employment

Psychological or social conditions

Numerous psychological and social theories have been suggested to explain why women may become depressed during perimenopause. Some of these are related to the following factors:

- ❏ Change in the childbearing role
- ❏ Loss of fertility, which may be associated with a loss of an essential meaning of life
- ❏ Empty-nest syndrome - Surveys have indicated, however, that women whose children have moved out of the house tend to report more happiness and enjoyment in life than others do
- ❏ Societal value of youth - In societies where age is valued, women tend to report having fewer symptoms at the menopause transition

Preexisting tendency toward depression

Personal or family histories of major depression, postpartum depression, or premenstrual dysphoric disorder seem to be a major risk factor for depression in the perimenopausal period. However, perimenopausal depressive syndrome is a risk even in women without a history of depression.

Epidemiology

Each year in the United States, 1.3 million women reach menopause. An estimated 20% of these women experience depression. The mean age of onset for the menopausal transition is 47.5 years.

According to a study conducted in Bangladesh and published in the Journal of Medicine, 82% of postmenopausal women suffer from depression which has one of the worst impacts on their quality of life (QOL) among all factors.

Depression is approximately twice as common in women as in men (21% vs. 12.7%). Moreover, depressive episodes are more recurrent, longer, worse, and more impairing for women than for men. In addition, the prevalence of dysthymia and minor depression is increased among women. These differences have not been noted for mania. Sex-related differences emerge at the age of 11-15 years.

The racial distribution of perimenopausal depression is not known. However, in countries where older women are highly valued, women experience fewer symptoms overall during menopause.

Clinical presentation

Patient history

Symptoms of perimenopause include the following:

- ❏ Hot flashes
- ❏ Cold sweats
- ❏ Irregular menstrual bleeding
- ❏ Urogenital atrophy and dryness with resultant dyspareunia (see Gynecologic Pain), itching, and urinary urge incontinence
- ❏ Cognitive and affective disturbance

Essential criteria for major depression include depressed mood, decreased interest or pleasure in activities, or both. Additional criteria include the following:

- ❏ Increased or decreased appetite
- ❏ Weight change
- ❏ Insomnia or hypersomnia
- ❏ Psychomotor agitation or retardation
- ❏ Feelings of worthlessness or guilt
- ❏ Decreased concentration
- ❏ Indecisiveness
- ❏ Suicidal ideation and plans
- ❏ Homicidal thoughts and plans

Overlapping symptoms of depression and perimenopause include the following:

- ❏ Low energy
- ❏ Impaired concentration
- ❏ Sleep disturbances
- ❏ Weight changes
- ❏ Libido changes

Symptoms of depression may include the following suggestive features from a Mental Status Examination:

- ❏ Motor retardation or agitation
- ❏ Latency of response to questions
- ❏ Slowed thought process
- ❏ Diminished prosody of speech
- ❏ Poor eye contact
- ❏ Poor grooming or hygiene (may indicate advanced depression)

Physical examination

Physical findings associated with perimenopause include urogenital atrophy, as well as flushing and diaphoresis during hot flashes.

Differential diagnosis

Problems to be considered in the differential diagnosis include the following:

- ❏ Adjustment Disorders
- ❏ Anemia
- ❏ Depression
- ❏ Dysthymic Disorder
- ❏ Hypothyroidism
- ❏ Dementia
- ❏ Depression secondary to a general medical condition
- ❏ Endocrine disorders (eg, hypothyroid)
- ❏ Substance abuse
- ❏ Use of medications, such as beta-blockers

Laboratory, imaging and other studies

Thyroid stimulating hormone (TSH) levels should be determined. The patients should be monitored for hypothyroidism because thyroid disease is an independent risk factor for depression in menopausal women.

Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels should also be measured. Because ovarian production of inhibin and estrogen declines during perimenopause, FSH and, later, LH levels begin to increase. An FSH level higher than 40 IU/L is often used as a marker of menopausal changes. Patients may begin to notice changes before laboratory values reflect the changes.

A hematocrit should be obtained; anemia can be associated with depressive-type symptoms. Dual-energy X-ray absorptiometry (DEXA) scanning is indicated to evaluate bone mineral density (BMD). Depression may be a risk factor for osteoporosis in postmenopausal women. A Mental Status Examination (MME) must be performed.

Treatment & management

In treating mood disorders that arise in association with menopause, primary care providers, gynecologists, and psychiatrists often find it helpful to work collaboratively.

Treatment of perimenopausal depression

Drugs used to treat perimenopausal depression include antidepressants and hormones.

Antidepressant therapy

For major depression, standard antidepressants are first-line treatments. Selective serotonin reuptake inhibitors (SSRIs) are the antidepressants most commonly used in the treatment of perimenopausal depression. These drugs act by inhibiting serotonin reuptake transporters in the presynaptic neuron, making more serotonin available at the synaptic cleft. The time to onset of action is 4-6 weeks.

SSRIs are thought to be generally safe and effective. They do pose a risk of serotonin syndrome, as well as several common adverse effects (eg, nausea, diarrhea, anorexia, excessive sweating, decreased libido or anorgasmia, headache, jitteriness, dizziness, sedation or activation, insomnia and akathisia). Several of these medications inhibit the cytochrome P450 (CYP450) enzymes; therefore, it is prudent to check for drug interactions.

Hormone replacement therapy

For mild depression, hormone replacement therapy alone may be appropriate. Estrogen may be used when traditional antidepressants failed, when patients refuse psychotropic medications or when patients experience other clinically significant vasomotor symptoms. Women who have surgically induced menopause have an increased risk of depression, and they may be especially likely to benefit from hormone replacement therapy.

Data from several studies suggested that estrogen replacement therapy had antidepressant effects or that it enhanced the effects of antidepressant treatment in perimenopausal women. While some other studies did not show that estrogen adds to the effects of SSRIs. In general, such treatments appear to be helpful for managing depressive symptoms in perimenopause but not in postmenopause.

Debate exists regarding whether the antidepressant effect is attributable to the effect of estrogen on vasomotor symptoms. Some studies reveal an antidepressant benefit only in women with vasomotor symptoms. Results of other studies suggest an independent antidepressant effect.

Improvement of mood and quality of life

In women with mild mood-disorder symptoms that do not meet the criteria for depression, hormone replacement therapy may be considered. The effects of estrogen treatment have been studied in perimenopausal women without depression to see if it has a positive effect on mood or quality of life.

Results from small studies have suggested a small positive impact on mood. However, most data suggest that, among healthy women without depression, estrogen has no favorable effect on quality of life or mood.

Treatment of hot flashes

SSRIs are sometimes used to treat hot flashes. Paroxetine, controlled-release paroxetine, extended-release venlafaxine, and escitalopram may provide some benefit. Clonidine and gabapentin have been shown to reduce hot flashes.

Treatment of sleep disorders

Estrogen may be helpful in relieving vasomotor symptoms that disrupt sleep or that may have a direct effect on sleep itself. In a study of postmenopausal women with hot flashes, night sweats, insomnia, anxiety or mood swings, low-dose estrogen and low-dose micronized progesterone improved sleep to a greater extent than could be explained by a reduction in vasomotor symptoms. One study using polysomnographic analysis found that isoflavone treatment is also effective in reducing insomnia symptoms.

Maintenance of cognitive function

Data from observational studies suggest that estrogen may prevent or delay the onset of dementia, including Alzheimer disease, especially when estrogen therapy is started in young postmenopausal women. The data do not suggest that estrogen is helpful for maintaining cognitive function in all perimenopausal and postmenopausal women.

Other measures

Negative anticipation of menopause seems to be associated with elevated rates of depression and physical symptoms of menopause. Educational groups that help women learn what to expect during menopause decrease anxiety, depression and irritability. Psychiatric hospitalization is indicated for patients who are at imminent risk of harming themselves or others and for those whose depres-sive symptoms render them unable to care for themselves.

A healthy, balanced diet is advisable. Regular exercise is thought to be helpful in diminishing the symptoms of depression.

Prognosis

Although morbidity and mortality secondary to perimenopausal depression has not been studied, depression is known to be a significant health problem in women. According to the World Health Organization's Global Burden of Disease Study, unipolar depression is the leading cause of disease-related disability in women. In this study, unipolar major depression was second to only ischemic heart disease in terms of associated morbidity and mortality.

The Study of Women's Health Across the Nation suggests that women are at higher risk for major depression during and immediately after the menopausal transition and that the risk is smaller when they are premenopausal.

Patient education

Patients may benefit from learning that mood symptoms are not uncommon during menopause. Providing education about depressive symptoms can help women understand their experiences and recognize depression as a treatable illness. Family members may also benefit from learning about the symptoms of major depression and the association of its onset with the menopausal transition. This information may help them understand changes they observe in their family member.

Patients should be educated about emergency mental health services. They should be aware of how to obtain help if they have suicidal thoughts.

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Contact dermatitis is a localized rash or irritation of the skin caused by direct contact the inducing substance to which the skin reacts. Such substances are toxic to the skin and are termed primary irritants. Others may induce an immunologic reaction called allergic contact dermatitis and include plant oils, metals, cleaning solutions, cosmetic additives, perfumes, industrial chemicals, topical antibiotics, and latex rubber additives.

Contact dermatitis is not caused by an infectious microorganism and is not contagious. Since the condition can produce open raw skin, a secondary infection can occur on the damaged skin. This secondary infection can be contagious.

Types

Usually two types of contact dermatitis:

- ❏ Allergic contact dermatitis
- ❏ Irritant contact dermatitis

Prevalence and risk factors

The prevalence of contact dermatitis in the general U.S. population has been variably estimated between 1.5% and 5.4%. Contact dermatitis is the third most common reason for patients to seek consultation with a dermatologist.

Exogenous and endogenous risk factors may contribute to develop contact dermatitis (Box 1 and Box 2). Although genetics, gender and age can be risk factors, the inherent nature, toxicity and degree of cutaneous exposure to chemicals are probably more important. A history of atopic dermatitis is linked to an increased susceptibility to irritant dermatitis because of a lower threshold for skin irritation, impaired skin barrier function and slower healing process. Wet work and chemical exposure are major precipitating factors for the development of irritant contact dermatitis (ICD) in patients with atopic eczema. Although atopic patient's down regulate Th1 cells with a decreased propensity to contact sensitization, allergic contact dermatitis (ACD) is often seen in patients with atopic eczema and nickel sensitization is increased in atopic patients. Other skin conditions with the highest risk of allergic contact dermatitis (ACD) include stasis dermatitis, hand and foot eczemas and chronic actinic dermatitis.

Box 1: Exogenous Factors Influencing Contact Dermatitis
Body temperature
Cutaneous penetration of chemical
Environment (temperature, humidity)
Mechanical factors (pressure, friction, abrasion)
Properties of the chemical (pH, chemical activity)
Other exposure factors: duration, prior or simultaneous exposures, direct versus airborne

Box 2: Endogenous Factors Influencing Contact Dermatitis
Age
Atopic dermatitis
Individual susceptibility
Lack of hardening
Primary sensitive (hyperirritable) skin
Race
Secondary hyperirritability of the skin (status eczematicus)
Sensitivity to ultraviolet light
Skin permeability

Causes

Allergic contact dermatitis

Allergic contact dermatitis occurs when the skin develops an allergic reaction after being exposed to a foreign substance. This causes the body to release inflammatory chemicals that can make the skin feel itchy and irritated.

Common causes of allergic contact dermatitis include contact with:

- ❏ Jewelry made from nickel or gold
- ❏ Latex gloves
- ❏ Perfumes or chemicals in cosmetics and skincare products
- ❏ Poison oak or poison ivy

Irritant contact dermatitis

Irritant contact dermatitis is the most common type of contact dermatitis. It happens when the skin comes in contact with a toxic material.

Toxic substances that can cause irritant contact dermatitis include:

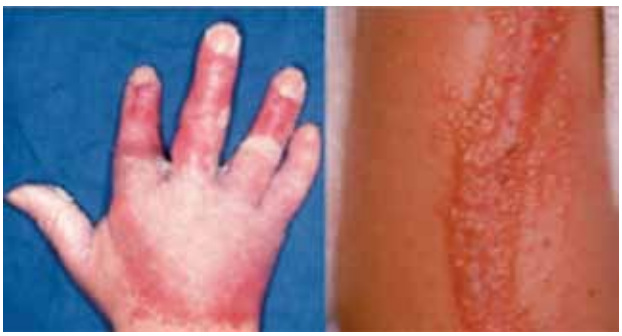
- ❏ Battery acid
- ❏ Bleach
- ❏ Drain cleaners
- ❏ Kerosene
- ❏ Detergents
- ❏ Pepper spray

Irritant contact dermatitis can also occur when the skin comes in contact with less irritating materials - like soap or even water - too often. People whose hands are frequently exposed to water, such as hairdressers, bartenders, and healthcare workers, often experience irritant contact dermatitis of the hands, for example.

Symptoms

Contact dermatitis symptoms depend on the cause and how sensitive you are to the substance.

Allergic contact dermatitis

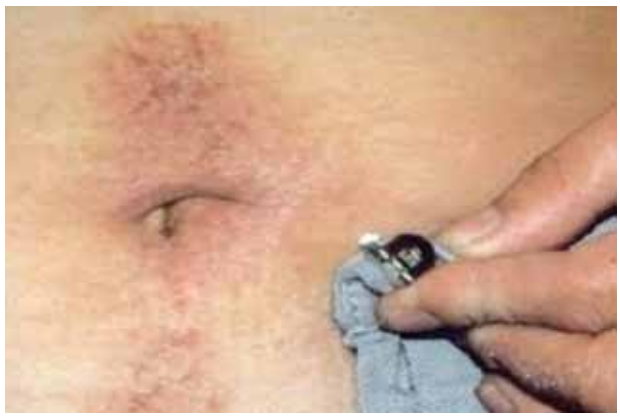


Symptoms associated with allergic contact dermatitis include:

- ❏ Dry, scaly, flaky skin
- ❏ Hives
- ❏ Oozing blisters
- ❏ Skin redness
- ❏ Skin that appears darkened or leathery
- ❏ Skin that burns
- ❏ Extreme itching
- ❏ Sun sensitivity
- ❏ Swelling, especially in the eyes, face or groin areas

Irritant contact dermatitis

Irritant contact dermatitis may cause slightly different symptoms, such as : blistering, cracking skin due to extreme dryness, swelling, skin that feels stiff or tight, ulcerations, open sores that form crusts.



Diagnosis

Usually contact dermatitis is diagnosed from symptoms and physical examination. Blood tests and X-rays are not helpful. The evaluation of allergic contact dermatitis may require patch testing.

Patch testing

If the history and the clinical presentation reveal one or more risk factors for ACD, patch testing is indicated. The patch test is the only useful and reliable method-the gold standard-for diagnosing ACD and its proper performance and interpretation require considerable experience.

The patch test aims to reproduce in miniature the clinical eczematous dermatitis by applying allergens under occlusion on intact skin of patients with suspected contact dermatitis. A positive patch test represents an in vivo visualization of the elicitation phase of contact dermatitis. Patch testing for ACD is not meant to reflect an irritant reaction. The patch test is typically better than relying on history alone, trial and error or in vitro tests.

Commercially available individual patch test allergens in a dilute, nonirritating concentration are applied to the upper back for 48 hours. After the patch tests are removed, the sites of the patch tests are evaluated twice, usually after removal at 48 hours and again at 96 hours or beyond. Results at both readings are graded according to intensity of the reaction at the patch test site on a scale of 0 to 3+. Relevance of positive reactions to present or past episodes of dermatitis is determined by correlating the patch test results with chemicals, products and processes encountered in the environment.

Patch tests should generally not be applied if the patient's dermatitis is active or involves the back.

Treatment

Identifying and avoiding irritants and allergens

Identification and elimination of the offending irritant or allergen and protection from further exposure are important in managing contact dermatitis of all causes and types. Patients with ACD must be educated about potential sources of exposure and cross-reacting allergens, and they must be provided with lists of potential products and processes that contain the allergen and a list of nonsensitizing substitutes. Examples of allergen alternatives include topical erythromycin or mupirocin as substitutes for neomycin. Neomycins can cross-react with gentamycin and tobramycin. Bacitratin should generally be avoided in neomycin-sensitive patients because of coreactivity. Reasons for persistence of ACD include unidentified sources of allergens or irritants at home or at work, exposure to cross-reacting allergens, presence of underlying endogenous (e.g., atopic) eczema and adverse reactions to therapy.

In the case of hand dermatitis, practical management must include protective measures as well as the use of topical corticosteroids and lubrication. The use of vinyl gloves with cotton liners to avoid the accumulation of moisture that often occurs during activities involving exposure to household or other irritants and foods (e.g., peeling or chopping fruits or vegetables) may be helpful. In the workplace, verify that gloves are safe to use around machinery before recommending their use. Protective devices themselves can introduce new allergic or irritant hazards in the forms of rubber in gloves and solvents in waterless cleansers. Automation of industrial processes can reduce exposure but is the most expensive preventive measure. Barrier creams are generally a last resort and are probably best used in workers with no dermatitis.

Topical treatment

Once dermatitis develops, use of topical treatment is helpful. Topical corticosteroids are the mainstay of ACD therapy; however, their use in ICD is controversial. For mild or moderate localized dermatitis, topical corticosteroids applied twice daily are usually effective within a few days and should be continued for 2 weeks. Lower-potency agents should be applied to the face and intertriginous areas and higher-potency steroids should be reserved for the extremi-ties and torso. Frequent and prolonged use of topical corticosteroids in fold areas can cause atrophy, telangiectasia or striae and their use on the face can also cause steroid rosacea. Topical calcineurin inhibitors (e.g., tacrolimus or pimecrolimus) may be used as an alternative to low-potency topical corticosteroids in chronic ICD.

Emollients or occlusive dressings may improve barrier repair in dry, lichenified skin. Traditional petrolatum-based emollients are accessible and inexpensive and they have been shown to be as effective as an emollient containing skin-related lipids. To relieve pruritus, a lotion of camphor, menthol and hydrocortisone is soothing, drying and antipruritic. Pramoxine, a topical anesthetic in a lotion base can also relieve pruritus. Phototherapy with bath PUVA (Psoralen Ultraviolet A) therapy may be helpful for chronic contact dermatitis of the palms and soles.

Systemic treatment

Most cases of contact dermatitis are effectively managed without the use of systemic corticosteroids. Short courses of *systemic corticosteroids* are indicated for patients with severe vesiculobullous eruptions of the hands and feet or the face or with severe disseminated ACD, such as poison ivy, sumac or oak. Also *systemic drugs* such as cyclos-porine may be effective, but they should be limited periods. When dermatitis is complicated with a secondary infection, systemic antibiotics against *Staphylococcus aureus* and *Staphylococcus pyogenes* are preferred. *Antihistamines* such as diphenhydramine hydrochloride, hydroxyzine hydrochloride and doxepin hydrochloride may be administered at night for intense itching.

Prognosis

If the irritating substance causing the contact dermatitis is removed and are not exposed to it again, rash probably will disappear on its own in less than three weeks. Symptoms may go away sooner with treatment.

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Hemophilia

Hemophilia A and B are most common form of X chromosome-linked bleeding disorders caused by decreased activity of factor VIII and factor IX due to heterogenous mutations in the factor VIII (FVIII) and factor IX (FIX) genes. Both factors take part in the intrinsic pathway of blood coagulation and affected individuals have mild to severe forms of the diseases. The word 'Hemophilia' was first used by Hopff in 1828. Hemophilia is sometimes referred to as “the royal disease” because Queen Victoria of England (1837 to 1901) was a hemophilia B carrier and from her it spread to the royal families of Spain, Germany and Russia. People with hemophilia do not bleed any faster than normal but they can bleed for a longer time than normal.

linked recessive disorder occurring due to the absence or deficiency of the clotting factor IX. The inheritance pattern and the symptoms of hemophilia B are same as that of the classic hemophilia. The prevalence of hemophilia B is 1 in 30,000.

Hemostasis and blood coagulation

The process of blood coagulation, the subsequent dissolution of the clot and the beginning of the repair of the injured tissue, is termed as hemostasis. Blood clotting is considered as an important step in the hemostasis. This is a process that leads to the formation of fibrin clot. During an injury the sequential steps that lead to the formation of clot are:

Step I). Vascular constriction. This limits the flow of blood to the area of injury.

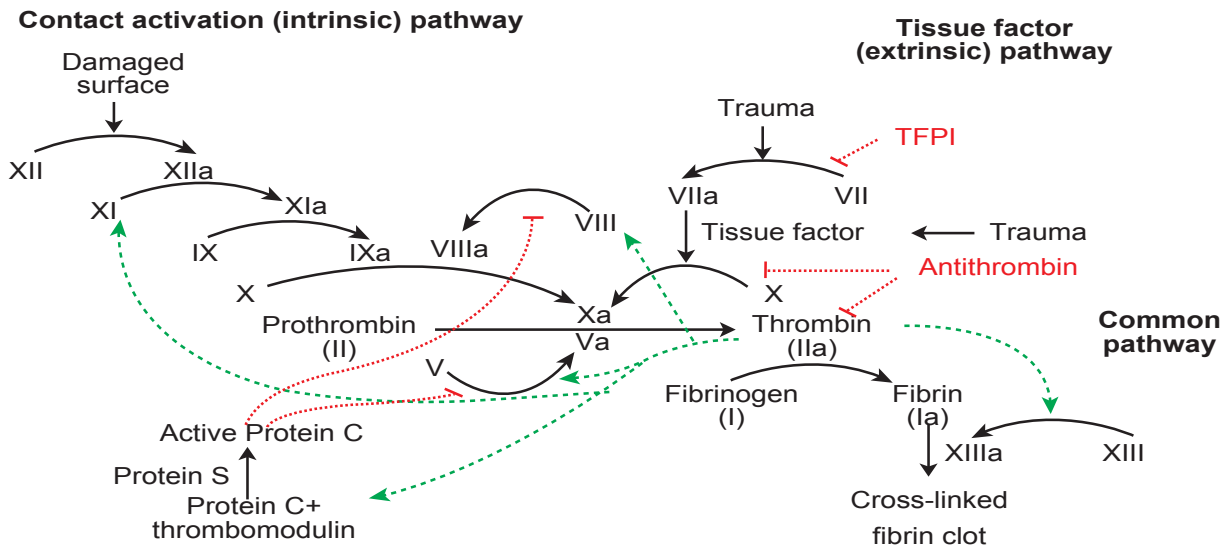

Figure 1: The role of coagulation factors on blood clotting. (the 'a' with the factor indicates the activated form)

Types of hemophilia

Hemophilia-A (Classic hemophilia): The most common type of hemophilia is called hemophilia A. It is otherwise called as the classic hemophilia. It is “X” linked recessive disorder occurred due to the absence or deficiency of clotting factor VIII (FVIII). Hence it affects only males. Females are said to be carriers. Carrier females usually are asymptomatic but can have bleeding symptoms (e.g., are easily bruised or have menorrhagia or excess bleeding after trauma) when they have significant reductions in factor VIII levels. The prevalence of hemophilia A is 1 in 5000 male live births.

Hemophilia B (Christmas disease): It is also an “X”

Step II). Platelets become activated by thrombin and aggregate at the site of injury, forming a temporary, loose platelet plug.

Step III). Formation of a strong fibrin clot.

Blood coagulation entails the interaction of a large number of plasma glycoprotein with blood platelets and vascular endothelial cells. It proceeds through three pathways which leads to the fibrin production. (Fig. 1)

1 Tissue factor pathway (Extrinsic)

2. Contact activation pathway (intrinsic)

3. Common pathway

Bleeding in hemophilia

The patient with hemophilia is deficient of FVIII/FIX/factor XI. The entire three factors are very much essential in the conversion of FX to its activated form. Deficiency of any of these factors fails to activate the factor X (Fig 2). If this is the case the total contact activation pathway (intrinsic) will be paralyzed. But the tissue factor pathway (extrinsic pathway) is normal in hemophilia patients which does not utilize the help of the above mentioned factors. So naturally one can think that whether this pathway is sufficient for the clot formation. but researches shows that Even though F VIIa-TF complex (Extrinsic pathway) is responsible for initial FXa generation, which provides sufficient thrombin to induce the local aggregation of platelets and the activation of the critical cofactors FV and F VIII in normal hemostasis, Persistent hemostasis requires the continued production of additional F Xa through the action of F IXa and F VIIIa, since the FXa generation by F VIIa-TF complex is inhibited by TFPI (Tissue factor pathway inhibitor). Thus, hemophilia patients bleed because the Xa generated through the action of F VIIa-TF complex and dampened by TFPI, is insufficient to sustain hemostasis and must be amplified through the action of F IXa and F VIIIa. And that persistent hemostasis requires the continued production of F Xa through the action of F IXa and F VIIIa.

parents, she is said to "carry" the hemophilia gene and is therefore called a "carrier." She can pass either gene onto her children. For each child, there is a 50% chance that a son will have hemophilia and a 50% chance that a daughter will carry the gene. On average, carriers of hemophilia will have about 50 per cent of the normal amount of clotting factor, but some carriers have far lower levels of clotting factor.

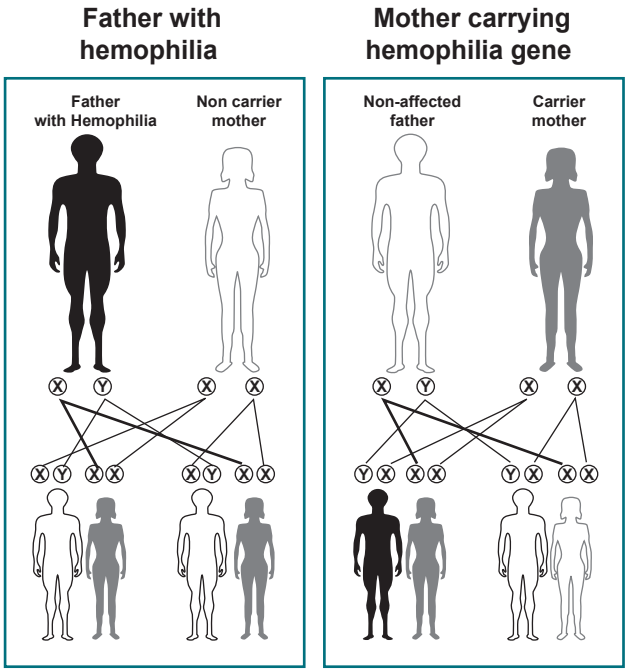

Fig-3: The inheritance pattern of hemophilia

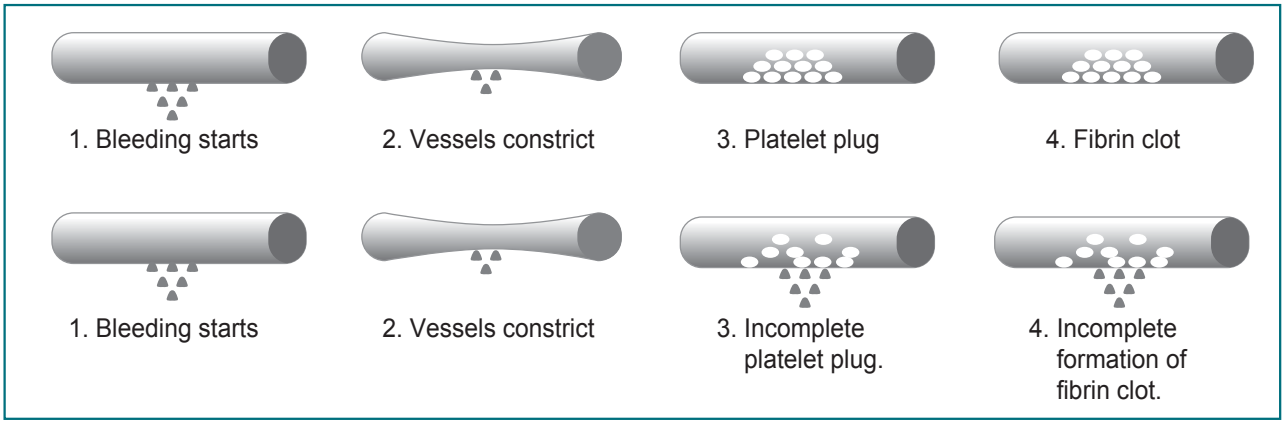

Figure 2: Schematic representation of why the hemophilia patients bleed than the normal individuals.

How a person get hemophilia

When the father has hemophilia but the mother does not, none of the sons will have hemophilia. All the daughters will carry the hemophilia gene. If a woman inherits a copy of the altered gene from either of her

Symptoms

People with hemophilia can bleed inside or outside the body. Most bleeding in hemophilia occurs internally, into the muscles or joints. The most common muscle bleeds occur in the muscles of the

upper arm and forearm, the iliopsoas muscle (the front of the groin area), the thigh, and the calf. The joints that are most often affected are the knee, ankle, and elbow. If bleeding occurs many times into the same joint, the joint can become damaged and painful. Repeated bleeding can cause other health problems like arthritis. The signs of hemophilia A and B are the same:

- ❏ Big bruises
- ❏ Bleeding into muscles and joints
- ❏ Spontaneous bleeding (sudden bleeding inside the body for no clear reason)
- ❏ Prolonged bleeding after getting a cut, removing a tooth, or having surgery.
- ❏ Bleeding for a long time after an accident, especially after an injury to the head.

Bleeding into a joint or muscle causes:

An ache or "funny feeling", swelling, pain and stiffness, difficulty using a joint or muscle

Severity of hemophilia

Severity of hemophilia A & B depends on the amount of clotting factor level, present in the plasma. People with severe hemophilia usually bleed frequently into their muscles or joints. They may bleed one to two times per week. Bleeding is often spontaneous, which means it happens for no obvious reason. People with moderate hemophilia bleed less frequently, about once a month. They may bleed for a long time after surgery, a bad injury, or dental procedure. A person with moderate hemophilia will rarely experience spontaneous bleeding. People with mild hemophilia usually bleed as a result of surgery or major injury. They do not bleed often and in fact, some may never have a bleeding problem.

Diagnosis

The diagnosis of hemophilia cannot be made on clinical findings. Diagnosis is made by measuring the level of factor activity in the blood. Hemophilia A is diagnosed by testing the level of factor VIII activity and Hemophilia B is diagnosed by measuring the level of factor IX activity. If the mother is a known carrier of hemophilia, testing can be done before a baby is born. Prenatal diagnosis can be done at 9 to 11 weeks by chorionic villus sampling (CVS) or by fetal blood sampling at a later stage (18 or more weeks).

Tests available for the diagnosis of hemophilia

1) Basic screening tests for hemophilia :

a) Bleeding time, b) Prothrombin time (PT), c) Platelet count, d) Activated partial thromboplastin time (APTT)

2) Factor assays :

3) Molecular genetic testing :

a) Sequence analysis, b) targeted sequence analysis

4) Linkage analysis (Mutation analysis) :

a) Tracking an unidentified mutation, b) Identifying the origin of a de novo mutation

5) Carrier detection :

a) Factor assay, b) DNA analysis, c) Phenotype analysis, d) Genotyping

6) Prenatal diagnosis (this is done if the fetus is suspected to have hemophilia) :

a) CVS (chorionic villous sampling), b) Amniocentesis

Present treatment options

Treatment for hemophilia today is very effective. Bleeding should be treated as quickly as possible.

1. Factor concentrates: Factor concentrates are the treatment of choice for hemophilia. They can be made from human blood (called plasma-derived products) or manufactured using genetically engineered cells that carry a human factor gene (called recombinant products). Infusions of factor concentrates are the most effective therapy available for hemophilia.

Level	Percentage of normal factor activity in blood	Number of international units (IU) per millilitre (ml) of whole blood
normal range	50% - 150%	0.50 - 1.5 IU
mild hemophilia	5% - 40%	0.05 - 0.40 IU
moderate hemophilia	1% - 5%	0.01 - 0.05 IU
severe hemophilia	less than 1%	less than 0.01 IU

There is no substitute for this therapy as it contains the corresponding factors in required quantity to stop the bleeding. Mainly there are two types of factor concentrates : a) Plasma deri-ved factor concentrates, b) Recombinant factors

2. **Cryoprecipitate:** It is prepared from the pooled blood and contains a moderately high concentration of clotting factor VIII, von willebr factor, fibrinogen and factor XIII (but not IX). It should have at least 80 IU of factor VIII activity and should be used as a replacement therapy in factor VIII deficiency. It is effective for joint and muscle bleeds, but is less safe from viral contamination than concentrates and is harder to store and administer. Cryoprecipitate can be made at local blood collection facilities.
3. **Fresh frozen plasma (FFP):** If the plasma obtained from the donor within 6 hours, it can be considered as fresh plasma and contain the entire clotting factor in near normal quantities. If this plasma is frozen at or below - 30˚C then it is called as FFP. In FFP the red cells have been removed, leaving the blood proteins including clotting factors VIII and IX. It is less effective than cryoprecipitate for the treatment of hemophilia A because the factor VIII is less concentrated. Large volumes of plasma must be transfused, which can lead to a complication called circulatory overload.
4. **Fresh whole blood:** When no other products are available, fresh whole blood can be used as it contains all the clotting factors. Since wet products are not virus inactivated, at present there is a significance risk of transmission of HIV/AIDS, hepatitis B and C. So screened donor has to be used to donate blood. The infusion should be continued until the bleeding stops.
5. **Adjuvant therapies:** Tranexamic acid It is an anti fibrinolytic drug that competitively inhibits the activation of plasminogen to plasmin, a molecule responsible for the degradation of fibrin. Tranexamic acid is a synthetic derivative of the amino acid lysine that exerts its antifibrinolytic effect by blocking lysine-binding sites on plasminogen molecules. This inhibits the interaction of plasminogen and the heavy chain of plasmin with lysine residues on the surface of fibrin. Although plasmin can still form, it cannot bind to and degrade fibrin. Tranexamic acid is mainly used to control mucosal bleedings. It is contraindicated to upper hematuria, due to the risk of formation of blood clots in the ureter and hydronephrosis.

Anti spasmodic and analgesics

For the management of pain during the bleeding episode analgesic like Paracetamol, Dextropropoxyphene, Buscopan, Codeine, Buprenorphine, Tramadol can be used. New generation COX -2 inhibitors are sometime also recommended. NSAIDs should be avoided because it increases the bleeding by inhibiting the platelet aggregation.

Prevention

The transmittance of the hemophilia to the next generation can be prevented by the following methods:

1) Prenatal intrauterine diagnosis with termination of pregnancy as an option
2) Pre implantation genetic diagnostic testing (PGD)
3) IVF with egg/sperm donation

Conclusion

People with hemophilia can live perfectly healthy lives. An estimated 400,000 people worldwide are living with hemophilia and only 25% receive adequate treatment. It is very hard to live with this bleeding disorder. As the number of patients reported with hemophilia is comparatively less than other major diseases like cancer, cardiac diseases and diabetics it seems to be less concentrated area by researchers. As technologies develop, the days are not far away when hemophilia will be treated with ease and even possibility of complete cure. Gene therapy, oral delivery of factor concentrates, long acting infusions, controlled release implants, prolonged release of other adjuvant therapy drugs are the areas to be concentrated for making the life of hemophilic patients easier.

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Unless all the necessary precautions are taken to the poultry production, marketing and processing chains, poultry meat and eggs can be contaminated by infectious agents that are harmful to humans. Poultry products can also be contaminated with the antimicrobial and anti-parasitic drugs or pesticides used on farms. The ingestion of antimicrobials can cause antimicrobial-resistant bacteria to develop in humans. Campylobacter and Salmonella infections are among the most important food safety hazards. These bacteria account for more than 90 percent of all reported cases of bacteria-related food poisonings worldwide. Most of these cases are related to the consumption of poultry and poultry products, but all domestic livestock are potential reservoirs of infection. Reported cases of Campylobacter and Salmonella infections are believed only a fraction of the true number of cases. Consuming raw or undercooked poultry or poultry products has been implicated as a potential risk factor for human cases of influenza h5n1 infection (hPai). Poultry meat should be well cooked, with the core temperature reaching 70°C for at least one second during cooking. Data on food-borne diseases in low-income countries are scarce. There is no precise and consistent global information about the full extent of the occurrence of food poisoning and the costs related to unsafe food.

The risk for consumers

In many countries, eating habits have undergone major changes over the last two decades. The middle class is growing, and this group of people eats more meat and goes out more often for meals. Migration from rural to urban areas has also resulted in changed eating patterns. New food production, preparation, and distribution techniques have developed in response to these changes. There is a large increase in “fast food” and other ready- to-eat foods, which means that consumers have less control over the selection, preparation, and storage of the meat they consume. Children and people in stress situations, such as those facing malnutrition, war or natural disasters, are especially at risk of food-borne bacterial diseases. The main symptom is diarrhea, and infection can be fatal (with 0.01 percent mortality in infected people in high-income countries) as the causal agent is a bacteria, these diseases can be treated by antibiotics, but access to treatment is difficult in many low-income countries. Another problem is the development of resistance to antibiotics among zoonotic bacteria.

Production systems

Backyard poultry production is an important activity for many rural households. Consumption of meat and eggs from this production system is considered safe because of the habits usually observed among consumers purchasing or preparing birds from backyard poultry production. Preparation is usually just after slaughter. Because a chicken provides one meal for a family, there are usually no leftovers. The meat is thoroughly cooked, which reduces the risk associated with the consumption of sick birds that is

Table : Quality control and risk factors in low-income countries

Characteristics	Production system		
	Backyard	Smallholder intensive	Industrialized
Production chain	Short	Medium	Long
Quality control during production	-	±	+++
Quality control during slaughter	-	±	+++
Product	Live birds	Live or locally slaughtered birds	Frozen parts, defrosted at the market
Contact between consumer and live product	+++	++ in live-bird markets or poultry shops	-
Refrigeration chain	Not necessary, immediate preparation of the whole carcass	Often not available	Often interrupted because of long chain
Consumer risk from bacterial contamination	+	++	+++
Consumer risk from resistant bacteria	-	+	+
Consumer risk from veterinary drugs residues and pesticides residues	-	+++	-

observed in many poor rural areas. If birds are infected, there is a risk of human infection with pathogens during the handling of live birds and during preparation. People with little or no experience of poultry farming may invest in small holder intensive poultry production and may build a small broiler or layer chicken house, often near new settlements or suburbs. In these small-scale operations, the use of antibiotics - which is sometimes adopted to compensate for poor performance resulting from inexperience in management, is not adequate. The risk of consumers ingesting antimicrobials and/or antibiotics is particularly important. In general, poultry meat and egg products from large-scale commercial operations are subject to efficient control processes and are safe. Large companies normally take considerable care to avoid bad publicity resulting from the commercialization of unsafe food products. However, one of the most common problems for large-scale commercially produced poultry meat in low-income countries is the lack of refrigeration during marketing. The grid table gives an overview of risk factors for food-borne diseases related to the consumption of poultry and poultry products from production systems in low-income countries.

Poultry processing plants

A typical poultry processing plant can process tens of thousands of chickens per day. Common complaints include warts, infections from bone splinters, and rashes from the chlorine water (used to wash carcasses contaminated with faeces). Employees have to do a lot of fast and repetitive movements. They often suffer from injuries caused by the knives, saws, and machinery. Cuts and lacerations are continuous hazards for workers frequently handling knives. Other injuries are also common. According to an Occupational Safety and Health Administration (OSHA) study, back injuries account for 40% of all poultry processing plant injuries. Workers who cut or pull the meat from the bone use quick and repetitive motions that put pressure on their wrists and hands. This situation makes these people vulnerable to debilitating conditions of the nerves, muscles and tendons. Carpal Tunnel Syndrome is the most severe type of such disorders.

When the tendons passing through a narrow channel in the wrist are overused, they swell and press on the nerve that controls feeling in the hand. According to a 1995 report published in the American Journal of Independent Medicine, 50% of workers reported three or more ongoing problems in the upper extremities, including decreased vibration sensitivity in their fingertips, impaired pinch strength and numbness.

Zoonotic infections

Zoonotic diseases are transmitted from animals to humans and include bacterial, viral, fungal and parasitic diseases. Salmonellosis, campylobacteriosis, chlamydiosis, tuberculosis, Newcastle Disease and avian influenza are amongst the most common zoonotic diseases transmitted from poultry to humans. Poultry workers are at a greater risk of being affected by these diseases.

These and other health hazards in poultry commercial settings must be addressed through improvement in the working environment. In order to achieve this very important goal, both employers and employees are responsible. Training of employees plays a vital role in reducing the occurrence of these problems. Always know your work environment, the contaminants and the potential hazards. Safety must always come first!

Reducing risks

The appearance of clinical signs in infected humans and the importance of these signs will depend on several factors. On a chilled carcass taken out of the refrigerator, most bacteria need an adaptation time of about two hours before they start to multiply. Usually, only high numbers of bacteria will cause disease, and only in more vulnerable people. Consumers can reduce the risk of bacterial food-borne diseases by refrigerating the meat from the moment it is bought until the moment of preparation (heating) for consumption. Temperature and cooking time are critical in minimizing risk. Contaminated parts are less likely to cause food poisoning problems if the meat is well cooked. However, some bacterial toxins are heat-stable, and will not be deactivated. Proper attention to minimizing bacterial contamination and proliferation is required from slaughter until cooking. Coagulated blood, blood pudding, and chicken-and-duck blood soup can contain harmful pathogens if not well cooked.

The World Health Organization (WHO) has elaborated the Five Keys to Safer Food programme.

Messages for food handlers and consumers have been developed, to decrease the incidence of food-borne diseases. Educational and training tools have also been developed. Education is an important measure for preventing risks to human health from poultry products. Thoroughly cooking in stew pans is fairly common in developing countries. The widely practiced habit of washing the skin or cutting the surface of the poultry meat before cooking helps to reduce bacterial contamination.

Consumer protection

The pattern of food-borne disease outbreaks has changed during the last two decades. In the past, most outbreaks were acute and localized and resulted from a high level of contamination. Now, more outbreaks affect several countries at once, resulting from low-level contamination of widely distributed commercial food products. Risks of the contamination of poultry products by residues and bacte-ria exist everywhere, owing to the globalization of poultry production and trade. Counteracting this, the relative risk of contaminated poultry products reaching the market has reduced in the last decade, thanks to faster and more reliable diagnostic tools, the establishment of a world epidemiological alert system and overall improvement of hygiene standards. The availability of efficient antibiotic treatments has also reduced the impact of food-borne diseases. As most food safety hazards related to poultry come from the immediate health risks of ingesting foods contaminated with zoonotic bacteria, regulation and testing efforts have focused on reducing the incidence of this type of contamination. Over recent decades, the food chain approach has been recognized as a valuable step forward in ensuring food safety from production to consumption. Such a system can also control contamination with pesticides and veterinary drugs along the production and marketing chains. The many and varied routes of contamination mean that many actors have a role in reducing risk, including feed mill operators, farmers, chicken processors, retailers, supermarkets, restaurants, takeaway establishments, health authorities, legislators, governments and consumers. Flock health, the structure of the poultry food chain (short or chilled), the quality of control procedures during production and supply processes and on the final product - all contribute to the marketing of safe poultry meat and eggs.

WHO has set up a Food-borne Disease Burden Epidemiology Reference Group (FERG), which harmonizes international efforts to estimate and reduce the global importance of food-borne diseases.

This will help countries to estimate the magnitude of foodborne illnesses and to evaluate progress in their control. An international network of laboratories, alert systems and collaboration among authorities assist in solving food safety problems.

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Test Yourself - 43

Correct Answers :

1.	c	2.	c	3.	a	4.	b	5.	b	6.	c

CONGRATULATIONS!

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Test Yourself - 44

1. The followings are true for "Poultry & Human Health" except:

a. Poultry meats and eggs can be contaminated by infectious agents that are harmful to humans.

b. Campylobacter and Salmonella infections are among the most important food safety hazards.

c. Risks of the contamination of poultry products by residues and bacteria exit everywhere.

d. The pattern of food-borne disease outbreaks has not changed during the last two decades.

2. All the followings are correct for "Postmenopausal Mood Disorder" except:

a. For major depression, hormone replacement therapy alone may be appropriate.

b. Selective Serotonin Reuptake inhibitors (SSRIs) are most commonly used in the treatment of perimenopausal depression.

c. Clonidine and gabapentin have been shown to reduce hot flashes.

d. Depression may be a risk factor for osteoporosis in postmenopausal women.

3. All the below are true for "Contact Dermatitis" except:

a. It is not caused by infectious microorganisms and is not contagious.

b. The evaluation of allergic contact dermatitis may require patch testing.

c. Most cases of contact dermatitis are effectively managed with the use of systemic corticosteroids.

d. Topical corticosteroids are the mainstay of allergic contact dermatitis therapy.

4. All the followings are correct for "Chikungunya" except:

a. Bangladesh found the first case in 2008.

b. Both Chikungunya and Dengue fever can often be seen simultaneously in the same patient.

c. The onset is more acute and the duration of fever is much shorter in Dengue fever.

d. The virus may be isolated from the blood during the first few days of infection.

5. The followings are right for "Postmenopausal Mood Disorder" except:

a. An estimated 20% of the women have depression at some point during menopause.

b. Insomnia occurs in 10% to 20% of women during the menopausal transition.

c. Thyroid disease is an independent risk factor for depression in menopausal women.

d. In most cases schizophrenia first manifests in young adulthood.

6. All the followings are correct for "Chikungunya" except:

a. The roots of this viral illness date back to 1953.

b. It is characterized by a gradual onset of fever frequently accompanied by the joint pain.

c. Most patients recover fully, but in some cases joint pain may persist for several months or even years.

d. After the bite of an infected mosquito, onset of illness occurs usually between 4 to 8 days but can ranges from 2 to 12 days.

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