HERPES SIMPLEX VIRUS OF GENITAL TRACT

ESSAY

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Of The Master Degree In

Gynaecology and Obstetrics

BY

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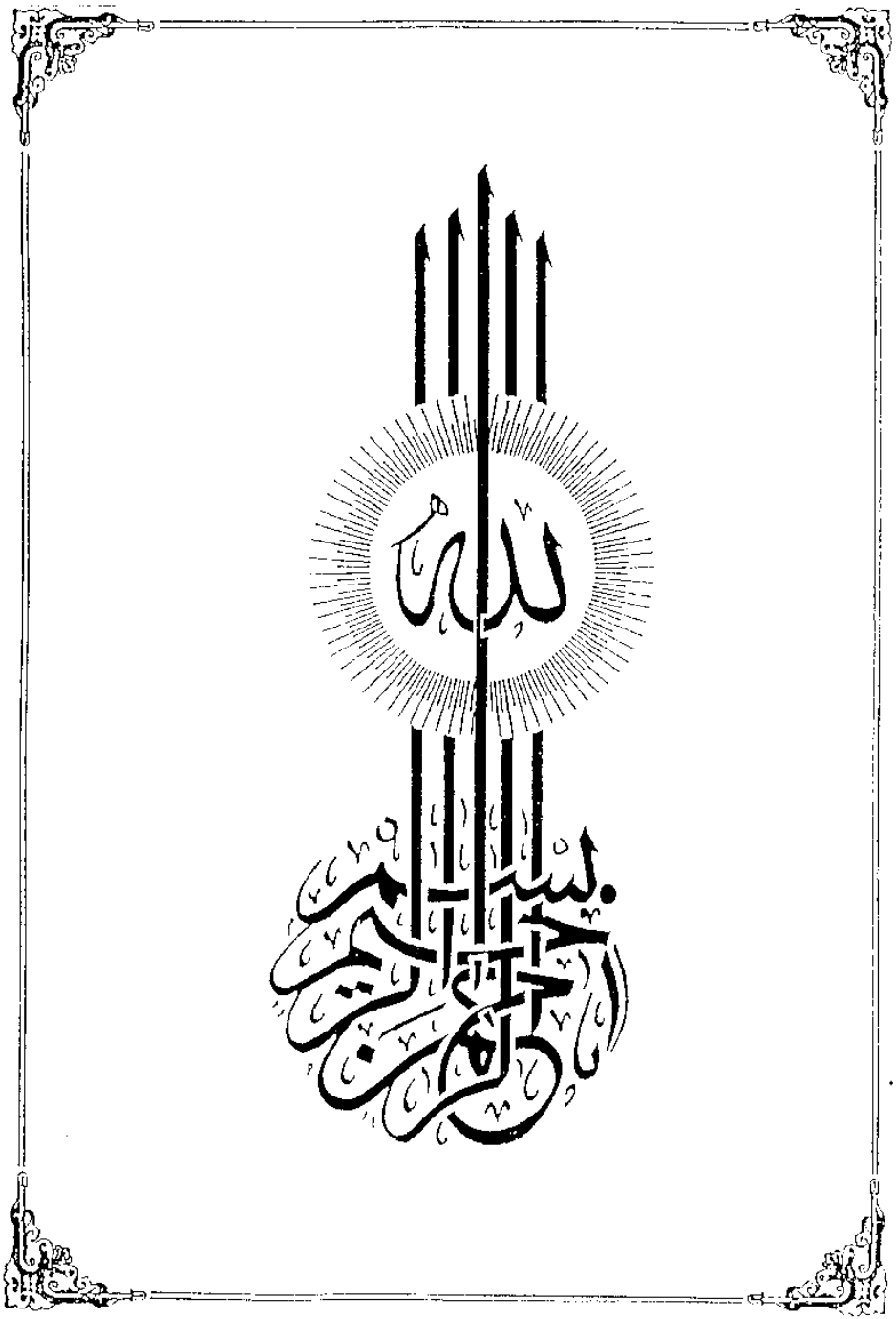
TO MY PARENTS



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SUMMARY

HERPES SIMPLEX VIRUS OF GENITAL TRACT

The term herpes is derived from the Greek word " to creeps", was first applied by Hippocrates.

Herpes simplex is caused by herpes virus hominis which is DNA containing virus, there are two antigenic strains of H. S. V. Type I and Type II.

H. S. V. is composed of four subvirons:

1. Core 2. Capsid 3. Envelope 4. Tegument

H. S. V. infection is one of the commonest infection in man throughout the world, frequently subclinical. Following infection clinical or subclinical the virus becomes occult and the host is latently infected.

Clinically H. S. V. infection occurs in two forms:

1. Primary infection which is manifested as:
   a) Acute gingivostomatitis.
   b) Acute keratoconjunctivitis.
   c) Herpes genitalis.
   d) Acute meningioencephalitis.
   e) Cutaneous herpes.
   f) Generalized H. S.

2. Recurrent infection.

Primary infection occur in exposed individuales who have no circulating antibodies either in childhood or in later life, usually the infection is severer than the recurrence and showing regional lymphadenitis recurrence tend to be in the same region with small sized vesicles, close grouping and without constitutional symptoms.

Herpes genitalis:

May be due to venerealor non-venereal contact but it can be considered as one of major sexually transmitted diseases, in young children the non-venereal contact is usual and is due to Type I strain of virus while in older children and adults the infection is usually due to a venereally induced Type 2 strain of virus, but despite increased incidence of genital herpes, proportion of the population infected by herpes simplex Type 2 remains small.

In the females primary infection of the vulva is shered by vesicles on the mucosa of the labia and sometimes on the adjoining skin which soon rupture to greyish-yellow plaque; there are regional lympha denopathy with local pain and low grade fever and the lesi on heals in 1-2 week's. Sometimes these painful ulcers may appear on the vagina and cervix as a large discrete punched out ulcer on a cervical lip or extensive tumour like mass resembling carcinoma

in conjugation with herpetic lesion s of the vulva and adjoining skin of perineum or thigh, sometimes pain and dysuria to cervical infection which result in ulcerative cervicitis.

The association of maternal genital with abortion, stillibirth and premature delivery has been reported and although secretions from the uterus have been shown to be inhibitory to the growth of herpes simplex virus in vitro recurrence of genital herpes is common.

In the male primary infection is manifested by urethritis with a burning on micturation and watery of purulent discharge and tiny vesicles about the urethral opening with inguinal lymphadenopathy. Penile infection is sometimes associated with acute herpetic stomatitis, commonly manifested as penile ulceration on the glans, prepuce or shaft of penis, the lesion is sore and last 2-6 weeks if untreated also recurrent herpes of the penis is common (herpes genitalis) and manifest by clusters of eroded vesicles on the glans.

genital herpes must be differentiated from :

1- Syphilitic primary lesion,

2- Chancroid (soft chancre, ulcus molle),

3- Gonococcal ulceration,

4- Granuloma inguinale, affects the genital and anal region and groins.

5- Gangrenous ulcer.

6- Ulcus valvuae acutum,

7- Erythema multiforme of the penis or vulva,

8- Bechets syndrome.

9- Ulcerations in the scrotal region that show no tendency to heal,

10- Candidasis of the genital tract,

11- Fixed drug eruption which may be herpetiform.

12- Lymphogranuloma venereum.

Neonatal Herpes:

Congenital herpes simplex virus infection was first recognized as a severe neonatal disease associated with cutaneous involvement,. The infection reaches the newborn either through the placenta or through direct contact in the birth canal, this intrauterine herpes simplex can be termed early when there is an evidence of disturbed embryogenesis or late when congenital malformations are less specific or in-apparent and usually women

delivering infected babies by herpes simplex virus were young nulliparous and infrequent aborters, infection was asymptomatic in 70 % of the mother s and usually in vertex deliveries, the scalp is common site for the development of the initial herpetic vesicles.

Whereas infants delivered by breach often have lesion on the buttocks and perianal area, lesions differ from those of adults in that they are grouped tense, arise on erythematous base. However individual vesicles progress to pustulate, at times larger bullous type lesion or zosterform can occur.

Herpes simplex is a common disease, also a typical forms are common-so diagnosis is sometimes difficult and require a laboratory assistance, when diagnosis can not be reached by the clinical picture alone:

1. Isolation of the virus from visible lesion, blood or organs at autopsy by inoculation of material into a susceptible laboratory host.
2. Demonstration of significant rise in specific antibodies in the serum taken in the acute stage of the disease preferably before the fifth day compared to the serum taken in the second and third week after the onset.
3. Demonstration of virus particles in the vesicular fluid under the electron microscope using phosphotangastic acid staining.
4. Demonstration of viral antigens in sections of infected tissues by the technique of fluorescent
5. Demonstration of the development of tuberculin like skin test after intra dermal inoculation of killed virus.
6. Demonstration of virus induced gaint cells in smears from the base of fresh vesciles.
7. Demonstration of characteristic inclusions in tissues suggesting herpetic infection.

H. S. V. infection is a self limited disease but chronic cutaneous H. S. can occur, it was found that cell-mediated rather than antibody mediated immunity play a role in recovery.

H. S. V. infection may precipitate Erythema Multiforme, and genital H. S. V. Type 2 infection can be considered as an etiological factor in cancer cervix.

Histopathologically:

Intraepidermal vesicles produced by ballooning and reticular degeneration, ballonning degeneration is specific for viral vesicles:

Management:

1. Prophylaxis through:
   a) German heat inactivated H. S. antigen.
   b) Interferon inducers.
   c) Human lymphocytes.
2. Tretament of H. S. V. infection:
   a) Silver nitrate paint.
   b) Gentain violet paint.
   c) Idoxuridine.
   d) Vidarabine (ara A) adenine arabinoside.
   e) Cytarabine (ara C) cytosine arabinoside.
   f) Acyclovir.

g) Levamisole.

h) Zinc sulphate solution.

i) Combined treatment by ultrasound and herpigon ointment.

j) Grenz rays.

k) Surgical conversion of H. S. from an epidermal to a dermal lesion.

N. B. :

Caesarean section may be considered as a prophylactic measure to prevent occurence of congenital H. S., if there is an actual lesion in the mother, primary or recurrent genital herpes simplex virus infection prior to the rupture of the membranes.

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