Partial hydatidiform mole

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Abstract Objective: To identify the cytogenetic origin, clinical characteristics and natural history of partial hydatidiform moles and to outline their management.

Design: Literature review on partial hydatidiform mole with special emphasis on partial moles with coexistent living fetuses.

Setting: Department of Obstetrics and Gynaecology, King Khalid University Hospital, Riyadh, Saudi Arabia.

Results: Partial hydatidiform mole is more common than the complete mole, often presenting with spontaneous or missed abortion. The incidence of persistent trophoblastic tumour following partial mole was found to be 3.6% Seventy-three point eight percent of patients with partial moles have a triploid chromosomal configuration. However 25 live births with diploid karyotypes and normal phenotypes have been delivered of mothers with coexisting molar pregnancy.

Conclusions: The diagnosis of partial mole is often missed as most patients present with abortions. The partial mole is considered a less virulent form of molar pregnancy, however after evacuation or delivery patients must be monitored by regular HCG measurements, since there is a possibility of developing persistent trophoblastic tumours.

In managing patients with partial hydatidiform mole with a coexistent living fetus, the pregnancy should be continued until fetal maturity is reached provided the fetal karyotype obtained on amniocentesis is normal, there is no evidence of fetal abnormalities on ultrasonography and the clinical course is stable.

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Keywords: Partial hydatidiform mole, molar pregnancy with coexistent fetus, trophoblastic disease, vesicular mole

The trophoblast is a unique tissue originating from the outer cell mass of the preimplantation embryo. It is devoid of transplantation antigens (HLA and ABO) which renders it invulnerable to maternal immune rejection. It thus invades with impunity the maternal decidua and its vessels as well as the connective tissue of the adjacent myometrium. This fact contributes to the difficulties in distinguishing between the benign and malignant phases. Trophoblastic disease encompasses complete and partial hydatidiform moles, choriocarcinoma and placental site tumour.

Of the various kinds of trophoblastic diseases, the hydatidiform moles are the most common. They have been classified into two groups: complete and partial molar syndromes. It is important to distinguish between complete and partial moles, for they are genetically programed and committed to separate modes of behaviour, the complete mole being associated with a significantly higher malignant sequelae.

Incidence Partial moles are much more common than the complete moles. Triploidy is found in 1 to 2 percent of clinically apparent abortions and 80 percent of triploids are diandric partial moles. However, the partial mole is a grossly under-diagnosed form of trophoblastic disease.1

A recent study by Kodama has shown that the incidence of hydatidiform mole was annually decreasing with an almost constant ratio to the number of pregnancies. The number of partial hydatidiform moles was rising with an inverse decrease in complete moles.2

The occurrence of a hydatidiform mole with a coexisting fetus is rare, the reported incidence being 1:10,000 to 1:100,000.34 In most of these cases the pregnancy is terminated in fetal death in utero with abortion during its early to middle stage. Molar pregnancy with a coexisting fetus progressing to a viable infant is an extreme rarity. The principal reported instances which resulted in delivery of a live infant between 1938 and 1992 are

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Table 1 - Reports of coexistent living newborn with hydatidiform mole

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>No. of Live Births</th>
<th>Gestational Age (mo)</th>
<th>Birth Weight (gm)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thaisz</td>
<td>1958</td>
<td>1</td>
<td>37</td>
<td>2050</td>
<td>Female baby with webbed fingers</td>
</tr>
<tr>
<td>Chacko</td>
<td>1964</td>
<td>1</td>
<td>30</td>
<td>1586</td>
<td>Female baby, No congenital anomalies</td>
</tr>
<tr>
<td>Bowles</td>
<td>1963</td>
<td>1</td>
<td>32</td>
<td>1960</td>
<td>Female baby, No congenital anomalies</td>
</tr>
<tr>
<td>Wieruszank</td>
<td>1963</td>
<td>1</td>
<td>36</td>
<td>2150</td>
<td>Female baby, No congenital anomalies</td>
</tr>
<tr>
<td>Muller &amp; Lapp</td>
<td>1950</td>
<td>1</td>
<td>35</td>
<td>1803</td>
<td>Female baby, No congenital anomalies</td>
</tr>
<tr>
<td>Ruse</td>
<td>1952</td>
<td>1</td>
<td>35</td>
<td>2840</td>
<td>Male baby, No congenital anomalies</td>
</tr>
<tr>
<td>Krone</td>
<td>1955</td>
<td>1</td>
<td>40</td>
<td>3200</td>
<td>Female baby, No congenital anomalies</td>
</tr>
<tr>
<td>Goldard</td>
<td>1960</td>
<td>1</td>
<td>33</td>
<td>1475</td>
<td>Male baby, No congenital anomalies</td>
</tr>
<tr>
<td>Sigristana &amp; Lapp</td>
<td>1960</td>
<td>1</td>
<td>36</td>
<td>1814</td>
<td>Female baby, Hemangiomata</td>
</tr>
<tr>
<td>Beischer</td>
<td>1861</td>
<td>5</td>
<td>23</td>
<td>-</td>
<td>Lived 10 minutes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>26</td>
<td>-</td>
<td>Lived 12 hours</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>31</td>
<td>1505</td>
<td>Male baby survived the neonatal No follow-up</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>36</td>
<td>2550</td>
<td>Male baby, No congenital anomalies</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>39</td>
<td>2950</td>
<td>Female baby</td>
</tr>
<tr>
<td>Beischer</td>
<td>1966</td>
<td>1</td>
<td>37</td>
<td>2980</td>
<td>Normal development until 9 months of age, No further follow-up</td>
</tr>
<tr>
<td>Jones &amp; Laasssen</td>
<td>1975</td>
<td>1</td>
<td>40</td>
<td>2900</td>
<td>Normal female</td>
</tr>
<tr>
<td>Fukuda</td>
<td>1976</td>
<td>1</td>
<td>38</td>
<td>1500</td>
<td>Normal female</td>
</tr>
<tr>
<td>Tomoda</td>
<td>1976</td>
<td>1</td>
<td>37</td>
<td>2400</td>
<td>Normal female</td>
</tr>
<tr>
<td>Suzuki</td>
<td>1980</td>
<td>1</td>
<td>32</td>
<td>1435</td>
<td>Normal female, Normal development until 12 months age, No further follow-up</td>
</tr>
<tr>
<td>Block &amp; Merrill</td>
<td>1982</td>
<td>3</td>
<td>36</td>
<td>1850</td>
<td>Normal male baby</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>35</td>
<td>2020</td>
<td>Normal male baby</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>24</td>
<td>900</td>
<td>Female baby with multiple congenital anomalies</td>
</tr>
<tr>
<td>Crooij</td>
<td>1985</td>
<td>1</td>
<td>27</td>
<td>980</td>
<td>Female baby 46 XX, Died after 87 days</td>
</tr>
<tr>
<td>Feinsberg</td>
<td>1988</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>Normal female baby 46 XX</td>
</tr>
<tr>
<td>Poi</td>
<td>1989</td>
<td>1</td>
<td>38</td>
<td>3450</td>
<td>Normal male baby, No congenital anomalies</td>
</tr>
</tbody>
</table>

Total no. of live births = 25

Genetic background The partial mole is a product of fertilization error. An apparently normal egg, with an intact 23, X haploid set is fertilized by two spermatozoa, the resulting karyotype showing 69 chromosomes. Partial moles are produced when two paternal sets combine with a single maternal set of 23, X (diandric triploidy), whereas no mole results if a diploid egg of 46, XX is combined with one paternal set (digenic triploidy).

Animal experiments suggest the hypothesis that the maternal genome is necessary for the development of the embryo, whereas the paternal pronucleus is associated with development of the trophoblast. In view of the constant persistence of the maternal pronucleus in the partial mole, it is tempting to speculate that its presence brings genes activated to take care of the embryo proper and it may also be responsible for the dilution of the full molar syndrome. 1 The extra set of paternal chromosomes may contribute to the development of focal trophoblastic hyperplasia. The central theme of the molar disease appears to be an excessive amount of paternal chromosomes, which induces trophoblastic hyperplasia.

This literature review of 405 cases of partial hydatidiform mole showed that triploidy was the most common 73.8 percent; 17.3 percent were normal diploid and 1.7 percent were tetraploid (Table 2).

The most likely explanation of a diploid partial mole is a twin gestation in which only the normal cell line was identified after cell culture. Without marker analysis twinning cannot be excluded, even when a diploid karyotype is revealed in both fetal and extra fetal cells. 7 It is suggested that twin pregnancy with hydatidiform mole is more frequent than its description in the literature would suggest. 8

The other explanation of a diploid partial mole is the existence of a further distinct entity, the diploid, bipaternal hydatidiform mole with partial cystic appearance of the placenta. This was suggested by Vejerslev et al. Cytogenic investigation, analysis of restriction fragment length polymorphisms, and flow cytometry in three pregnancies were consistent with diploid, bipaternal conception as the origin of fetal tissue and molar and non-molar villi. This seems to constitute a separate group within hydatidiform mole, with implications for counselling and management of pregnancy. 7 Rare cases of tetraploid (XXXXY) partial moles have been reported. Two cases resulted from a combination of a haploid ovum with three haploid sets of paternal...
Table 2 - Chromosomal configuration of partial hydatidiform moles

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>No. of cases with partial mole</th>
<th>Chromosomal Configuration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasiilakos</td>
<td>1977</td>
<td>22</td>
<td>9</td>
</tr>
<tr>
<td>Sridharan &amp; Nurti</td>
<td>1978</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td>Jacob</td>
<td>1982</td>
<td>80</td>
<td>75</td>
</tr>
<tr>
<td>Lawler</td>
<td>1982</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Suri</td>
<td>1986</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Obama</td>
<td>1986</td>
<td>56</td>
<td>9</td>
</tr>
<tr>
<td>Edward</td>
<td>1987</td>
<td>19</td>
<td>13</td>
</tr>
<tr>
<td>Lawler &amp; Fischer</td>
<td>1987</td>
<td>35</td>
<td>38</td>
</tr>
<tr>
<td>Davis</td>
<td>1987</td>
<td>32</td>
<td>29</td>
</tr>
<tr>
<td>Hemming</td>
<td>1987</td>
<td>10</td>
<td>22</td>
</tr>
<tr>
<td>Lage</td>
<td>1988</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Lage</td>
<td>1989</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Lage</td>
<td>1991</td>
<td>51</td>
<td>41</td>
</tr>
<tr>
<td>Lawler</td>
<td>1991</td>
<td>51</td>
<td>41</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>405</td>
<td>299</td>
</tr>
<tr>
<td>Percentage</td>
<td></td>
<td>17.3%</td>
<td>73.8%</td>
</tr>
</tbody>
</table>

Chromosomes either by the mechanisms of tripermy involving three separate haploid spermatozoa or through dispermy, involving one haploid and one diploid sperm. Other reported cases of tetraploidy, of 92, XXXX or 92, XXXY karyotype, resulted from a failure of the first mitotic division of a normal zygote.^{22,23}

The important factor in the evolution of partial or complete moles appears to be the ratio of maternal to paternal chromosomes and not the ploidy of the tissue. Partial hydatidiform moles develop from those conceptions in which the number of paternal sets of chromosomes exceeds the number of maternally derived sets.^{40}

The histopathologic profile of the partial hydatidiform mole The syndrome of partial mole has an ascertainable fetus (alive or dead), commonly gives a triploid karyotype, and exhibits a slowly progressing hydatidiform swelling in the presence of functioning villous capillaries that spares many villi; trophoblastic immaturity is constant and focal hyperplasia is inconspicuous but present.^{38}

The presence of small, unenlarged immature villi is characteristic. These may be interpreted as representing the more recently formed elements of the growing placenta that have not yet begun to undergo hydatidiform changes, and it must significantly contribute to fetal survival.^{24} One of the essential differences between partial and complete moles is the retention of the functioning fetal vessels in the villi as the latter accumulates fluid.^{46} The functioning of the villous capillaries is proved by the presence of fetal erythrocytes with the same proportion of nucleated cells as in the embryo proper.^{24}

The trophoblast is strikingly immature and maintains its Langhans layer well into the second trimester. There is hyperplasia, albeit much less dramatic than in classical moles. It varies from mild to moderate and appears as small foci of vaculating syncytium that vary in number from case to case.^{34}

A further characteristic of the partial moles with triploidy is the "trophoblastic inclusions", which are invaginations of the surface trophoblast into the stroma of the villi. Another special feature found in the enlarged villi is the formation of complex, maze-like central cisterns which are sharply demarcated by connective tissue without any traces of endothelium and contain no blood. These usually appear after 19 weeks of gestation.^{24}

The partial mole, 46 XX or 46 XY, partakes of morphologic characteristics of both main syndromes and may represent an unusual syndrome of its own.^{77}

In summary the histopathologic characteristics of partial moles are:

1. Chronic villi with focal hydropic swelling and cavitation or cistern formation.
2. Focal mild to moderate trophoblastic hyperplasia confined to the syncytiotubular.
3. Scallop of the chorionic villi and stromal trophoblastic inclusions.
4. Identifiable fetal or embryonic structures.

Clinical presentation Partial mole has been considered a less virulent form of molar pregnancy. The clinical characteristics and natural history are not entirely dissimilar from the complete mole. However, they do not exhibit the dramatic clinical features characteristic of complete mole.

The commonest clinical presentation of the partial mole remains "spontaneous or inevitable abortion" that takes place most often in the second trimester. Partial moles in the guise of missed abortions constitute the other large group, they...
present in the latter half of the second trimester with a long-dead fetus, fibrosis of villi and scanty hydatidiform vesicles. The uterus is invariably small for dates and HCG only slightly raised. In this group, as in that of spontaneous abortion, uterine retention after embryo-fetal demise tends to be perceptibly longer than in the common end-of-first trimester spontaneous abortions, pregnancy most likely being prolonged by the sustained syncytiotrophoblastic CG secretion characterizing the partial mole.

Between 1979 and 1984, 81 patients with partial hydatidiform mole were followed up at the New England Trophoblastic Disease Center. The presenting clinical diagnosis was either incomplete or missed abortion in 91 percent of patients. The uterine size was either small or appropriate for gestational age in 96.3% of patients. Only 6.2% of patients presented with excessive uterine size or toxemia and were thought to have a molar pregnancy. No patient had prominent theca lutein cysts, hyperthyroidism or respiratory insufficiency.

In another review from Hawaii, 55 cases were collected. Their mean age was 25.6%. The most common presenting symptom was vaginal bleeding in 68% of cases. Hypertension was present in 68%. Excessive uterine size was found in 44%. This was different from the above mentioned study where only 3.7% presented with a uterus large for gestational age and only 2.5% showed toxemia. Similarly Szulman and Surti described only 11% of their patients with partial moles presenting with excessive uterine growth. The gestation age at diagnosis of partial mole was 23.8 weeks. The average fetal weight recorded was 1155 grams with a range of 50-1980 grams. Four women delivered at term and six others delivered in the third trimester.

The clinico-pathological profile of 86 patients with partial hydatidiform mole was studied in comparison with the more familiar syndrome of the classic complete mole (115 cases). Nearly half of the patients with partial molar disease presented with spontaneous abortion whereas the other large group, 43%, was that of missed abortion. Forty percent of the patients with complete hydatidiform moles presented spontaneous abortions and only 12% had missed abortions.

Maternal age tends to be slightly higher in patients with partial mole (mean 24.7 years). Gravidity was significantly lower in women with partial molar syndrome. Four patients (8%) with partial moles presented with pre-eclampsia and the same number of cases of pre-eclampsia were seen in complete moles, however, its occurrence in pregnancies with partial mole was comparatively late, the corresponding time ranges being 13-16.5 weeks for a completed mole and 17-22 weeks for a partial mole.

In partial moles the predominance of small for dates uterus (65%) is striking, while large for dates uterus was substantially lower (11%). Uterine enlargement was found in 33% of all complete mole cases.

Virtually all characteristics, morphologic and clinicopathologic features, of the class complete mole appear in the partial mole syndrome. Comparison of the two syndromes suggests that the partial moles may be regarded as an attenuated version of the classic syndrome.

**Diagnosis** Few partial moles (approximately 10%) have been diagnosed pre-operatively. The bedside diagnosis is often delayed because the uterus usually remains small or normal for gestational dates, and bleeding is often late and slight. Because the fetus dies early, differentiation from "missed abortion" is often impossible, and the diagnosis rests on gross and microscopic pathology.

I) **Ultrasound** The proportion of partial hydatidiform cases diagnosed pre-operatively may be slowly rising with the application of sonography. Even so, because the hydatidiform vesicles are often few and small and increase in size slowly, there remain many cases that evade diagnosis. Ultrasound can detect partial molar disease in about 26% of patients. Infrequently, ultrasound also identifies a fetus with multiple anomalies suggestive of triploidy.

Two sonographic findings were significantly associated with the diagnosis of partial mole: cystic changes in the placenta due to swollen villi that produce a snowstorm-like pattern and ratio of transverse to anteroposterior dimension of the gestational sac more than 1.5. Changes in the shape of the gestational sac may be part of the embryoopathy of triploidy. When both criteria were present, the positive predictive value for partial mole was 87%.

II) **HCG** (Human chorionic gonadotrophin) The measurement of HCG is an integral part of the diagnosis and evaluation of the patient suspected of having gestational trophoblastic disease. The levels
in normal pregnancy rarely exceed 100,000 mIU/ml. The HCG levels tend to be elevated above normal pregnancy values in complete mole, whereas partial mole tends to produce lower levels. The HCG serum beta-HCG was found to be greater than 100.00 mIU/ml in only 6% of patients with partial mole.42

Partial moles have a higher serum level of percent free alpha-HCG than do complete moles (0.85 vs 0.17, P<0.005). The mean ratios of beta-hCG to alpha-HCG in complete and partial moles are 20.9 and 2.4 respectively (P<0.005).35

The trophoblastic cells in complete and partial mole differ significantly in the manner in which they secrete the free subunits of HCG. Trophoblastic cells secrete higher levels of free beta-HCG with increasing cellular atypia and proliferation. Percent free beta-HCG increases progressively from normal pregnancy after five weeks gestation (mean, 0.4), to partial mole (mean, 2.4), to gestational choriocarcinoma (mean, 9.2).46

III) Histopathologic diagnosis, chromosomal ploidy and parental origin of partial hydatidiform mole. The differential pathologic diagnosis of the partial mole depends on a constellation of several variable morphologic features, none of which is unique for the mole. Irrespective of the gross morphology, the final diagnostic features must be assessed through the microscope and the diagnosis of a partial mole rests with the pathologist, as it is based on a characteristic constellation of histologic criteria (described earlier on page 11).

Chromosomal ploidy can be determined by flow cytometric analysis of nuclear DNA content which is a rapid, accurate and cost effective means for assaying nuclear ploidy.33,34 Other means of chromosome ploidy analysis using interphase cytogenetics by in situ hybridization or staining have been described.46,47

Parental origin or cell line can be determined by studies of chromosomal banding polymorphisms, human leukocyte antigen typing of molar and parental cells and restriction fragment length polymorphisms.48

With incomplete genetic information a hydatidiform mole with coexistent normal fetus is generally considered to result from dizygous twinning comprising an androgenetic complete mole and a normal conception.37 Analysis must distinguish between a partial mole plus a triploid fetus and abnormal fetus occurring with a partial or complete mole. The distinction is important for decisions made during pregnancy and may be of prognostic significance after termination.38

The diploid partial moles tend to have more complications. About 20% would develop persistent gestational trophoblastic disease, sometimes with clinical metastasis or overt choriocarcinoma.31 In addition, diploid partial moles with persistent tumour are less sensitive to single agent chemotherapy.9 A combined morphologic and genetic classification of partial moles is recommended for identification of patients at high risk of complications.

Natural history The incidence of persistent gestational trophoblastic disease after evacuation of partial mole was reported to be between 1.24-14% in different studies.

Table 3 summarizes all reported cases of gestational trophoblastic disease following partial hydatidiform mole between 1976 and 1992. The percentage of persistent trophoblastic disease after a partial mole in this review of 2,306 cases was 3.6%. This highlights the problem of differentiating between complete and partial hydatidiform mole, which is quite important from a prognostic standpoint, since partial moles are usually benign and regress spontaneously without requiring therapy, while about 20% of women with complete (or classic) moles will need chemotherapy for persistent disease.46

While persistent gestational trophoblastic tumours follow both partial and complete moles, there remains controversy as to whether the malignant choriocarcinoma can follow a partial hydatidiform mole. Gardner recently reported a case of choriocarcinoma following a partial hydatidiform mole.46 However, it is still uncertain whether the risk of gestational choriocarcinoma preceded by partial mole exceeds the risk related to non-molar abortions.26 While patients with persistent disease after partial mole usually have non-metastic tumours, Stone et al.,60 Wong et al.,60 and Kodama et al.,2 all reported patients with metastasis after partial mole.

The DNA content of most partial moles with persistent gestational trophoblastic tumour is triploid and most of them will achieve complete remission with one course of single agent chemotherapy.39

Patients with partial mole who develop persistent gestational trophoblastic tumour do not
have clinical or pathological characteristics that distinguish them from other patients with partial mole. Therefore all patients with partial mole should be followed up with measurement of HCG levels to detect persistent disease and affect prompt therapy.

### Table 3 - Gestational trophoblastic tumours following partial hydatidiform moles

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Total no. of cases with P.H.M.</th>
<th>No. of cases who developed PGTT after evacuation of P.H.M.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stone &amp; Bagshawe</td>
<td>[50]</td>
<td>1976</td>
<td>71</td>
<td>5</td>
</tr>
<tr>
<td>Szuliman &amp; Suri</td>
<td>[43]</td>
<td>1982</td>
<td>49</td>
<td>2</td>
</tr>
<tr>
<td>Berkowitz et al</td>
<td>[42]</td>
<td>1985</td>
<td>81</td>
<td>8</td>
</tr>
<tr>
<td>Giber et al</td>
<td>[51]</td>
<td>1986</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Obama et al</td>
<td>[28]</td>
<td>1986</td>
<td>56</td>
<td>0</td>
</tr>
<tr>
<td>Lowe &amp; Vint</td>
<td>[52]</td>
<td>1986</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Lawler &amp; Fisher</td>
<td>[30]</td>
<td>1987</td>
<td>38</td>
<td>0</td>
</tr>
<tr>
<td>Edward et al</td>
<td>[29]</td>
<td>1987</td>
<td>55</td>
<td>8</td>
</tr>
<tr>
<td>Bolis et al</td>
<td>[33]</td>
<td>1988</td>
<td>86</td>
<td>2</td>
</tr>
<tr>
<td>Ricet et al</td>
<td>[54]</td>
<td>1990</td>
<td>240</td>
<td>16</td>
</tr>
</tbody>
</table>

*All non-metastatic GTT

*No choriocarcinoma

** baggage et al | [55] | 1990                          | 857                                                      | 10         | 1.2%       |
| Lage et al      | [35] | 1991                          | 310                                                      | 17         | 5.5%       |
| Lawler et al    | [36] | 1991                          | 51                                                       | 0          | 0%         |
| Verjuskev       | [56] | 1991                          | 1                                                        | 1          | 1%         |
| Kodama et al    | [2]  | 1991                          | 307                                                      | 6          | 1.6%       |
| Appelman et al  | [57] | 1992                          | 1                                                        | 1          | 1%         |
| Gardner & Lage  | [58] | 1992                          | 1                                                        | 1          | 1%         |

Total 2306 84 3.6%

P.H.M. = Partial hydatidiform mole
PGTT = Persistent gestational trophoblastic tumours

### Management

As mentioned earlier most of the patients with partial hydatidiform mole present with inevitable or missed abortions and the diagnosis is based on histopathologic features of the curettage specimens.

Only about 6% of partial moles are diagnosed pre-operatively and in those cases the management would be the same as for the classic or complete mole. Suction curettage is the preferred method of evacuation, followed by Sharpe curettage, to remove any residual molar tissue. For patients with complete mole, hysterectomy with mole in situ, is sometimes performed if the patient desires sterilization and prophylactic chemotherapy can be used. However, I think that these are not indicated for the management of patients with partial hydatidiform moles, since it is a less virulent form of trophoblastic disease and the incidence of persistent gestational trophoblastic tumour after evacuation is low.

The major difficulty in the management of partial hydatidiform mole lies in the case of a surviving coexistent fetus. Jones and Lauerse recommend immediate termination of pregnancy after the diagnosis of hydatidiform mole with a coexisting fetus, but they do not give any reasons. Some cases of molar pregnancy with a coexisting fetus are terminated because of severe pre-eclampsia. Suzuki et al, however, state that in the absence of pre-eclampsia or fetal abnormality the pregnancy can be allowed to continue, since there is no reported evidence suggesting that the likelihood of secondary molar growth or choriocarcinoma increases as the pregnancy advances.

The possibility of hydatidiform mole should be considered whenever a patient presents with bleeding, inappropriate uterine growth, and sudden hypertension or pre-eclampsia in the first and second trimesters. In such cases the evaluation should include ultrasound scanning for both placental pathology and fetal abnormalities. If hydatidiform mole is suggested, serum HCG may be important for confirmation of the diagnosis and as a baseline for subsequent follow-up. Absolute levels of HCG may not be solely diagnostic of mole.

Amniocentesis for chromosome analysis and identification of karyotype is suggested. If a fetal abnormality is identified by ultrasound or by chromosomal analysis, prompt termination of pregnancy seems appropriate, for continuation of the pregnancy presents unwarranted risk of maternal morbidity. With normal fetal karyotype in the presence of diffuse hydroptic change, continuation of the pregnancy may not be unreasonable when the clinical course is stable. The identification of a significant discrete mass of molar tissue however does seem to warrant pregnancy termination because this particular morphologic entity appears to have a potential for subsequent...
malignant trophoblastic disease. Watson et al confirm that normal karyotype is obtained on amniocentesis, and there is no evidence of fetal abnormality on ultrasonograph, the pregnancy should be allowed to continue until fetal maturity is reached.

Follow up After evacuation or delivery, the patient should be followed up with weekly HCG measurements until they are normal for 3 consecutive weeks and then with monthly HCG until measurements are normal for 6 months. Persistent gestational trophoblastic tumour is diagnosed if the HCG level re-elevated or plateaued for at least 3 consecutive weeks. The mild degree of trophoblastic hyperplasia in partial mole is generally associated with lower HCG levels and their rapid decline to normal (as compared to complete mole). In patients with partial moles and coexisting fetus, the initial HCG levels vary from 150,000 to 250,000 UI/liter urine and decrease to normal levels within an average of 13.5 weeks. For patients with missed and spontaneous abortion the levels return to normal within 6-8 weeks post-evacuation.

The patients are advised to use reliable contraception. There has been some suggestion in the literature that birth control pills might increase the risk of persistent gestational trophoblastic disease but recent data suggests that birth control pills are safe and effective in this situation.

Treatment for persistent gestational trophoblastic disease following partial hydatidiform mole Generally the same lines of treatment, chemotherapy or surgical, are followed whether the persistent gestational trophoblastic disease follows a complete or partial mole. Triploid partial moles usually require only one course of single-agent chemotherapy, while diploid partial moles are less sensitive. Methotrexate, actinomycin D, and hysterectomy all have been used for treatment of women with persistent gestational trophoblastic disease following partial moles. The number of treatment courses range from 1 to 4 and all of them achieve remission.

Reproductive experience after partial hydatidiform mole A review of subsequent pregnancy in all cases with partial and complete mole and gestational trophoblastic tumours who were managed at the New England Trophoblastic Disease Center was performed between 1965 and 1989. It was concluded that such patients should be reassured that they can anticipate a normal reproductive outcome in the future.

References


الهدف:
معرفة الأصل الروائي الخلوي والمميزات السريرية والتطور الطبيعي للرحي العدارية المتجزئة، ومن ثم بيان طرق العلاج.

tصميم الدراسة:
مراجعة لمجموع ما تم نشره عن الرحي العدارية المتجزئة مع الاهتمام الخاص بالحالات المرضية التي تعاني من وجود حقيقي جنبًا إلى جنب مع حالة حمل مشوٌم.
المكان:
قسم النساء والولادة، مستشفى الملك خالد الجامعي، الرياض، المملكة العربية السعودية.

النتائج:
بعد انتشار الرحي العدارية المتجزئة أكثر شيوعًا من انتشار الرحي العدارية المكتملة، ومعظم المريضات يظهرن في حالة إفراز عفوي أو إفراز غافل. وجد أن نسبة انتشار ورم المشيمة الظهاري الذي يعقب حدوث الرحي العدارية المتجزئة تبلغ 7.2% وقد وجد أن تركيب شكل الصبغيات في حالة الرحي العدارية المتجزئة هي صبغيات دائرية وذلك في 8.73% ومع ذلك هناك 25 حالة كان فيها نمط النمو المزدوج سلبيًا، وقت ولادة أطفال سليمين من الناحية الظهرية ولدتهم أمهات كان يعانين في الوقت نفسه من وجود حالة حمل مشوٌم.

الاستنتاج:
غالبًا ما يحدث خطأ في تشخيص الرحي الداري المتجزئة؛ نظرًا لأن معظم المريضات يتعرضن في حالة إفراز. تعد الرحي العدارية المتجزئة صورة أقل في حدثها من صورة الحمل المشوٌم، أي أنه يمكن اعتبارها صورة مخففة عن السمات الخاصة لتشابهات الكتلة. ومع ذلك يجب متابعة المريضات بعد الإفراز أو الولادة، وذلك لاحتفال الإصابة بوجود المشيمة الظهاري الذي يعقب حدوث الرحي العدارية المتجزئة.

إن معالجة المريضات اللواتي يعانين من رحي عدارية متجزئة موجودة جنبًا إلى جنب مع حالة حمل حقيقية يكون الجنين فيها جيًا، تستند على شريطة أن يدل فحص نمط النواة المأخوذة عبر فحص السائل ما يدل على وجود تخلفات شاذة في الجنين عند إجراء الفحص بالمجيبات فوق الضوئية، وأن الحالة السريرية العامة حالة مستقرة. 

مفتاح الكلمات:
رحي عدارية جزئية، حمل حقيقي متزامن مع الحمل المشوٌم، مرض الأرمعة الغاذبة، الرحي الحويصلية.