Decidual T lymphocyte activation in hydatidiform mole

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Abstract

Aim—To quantify and compare decidual leucocyte subpopulations in complete and partial hydatidiform molar pregnancy with those in normal early pregnancy.

Methods—Decidual leucocytes were characterised using an avidin-biotin technique and a panel of monoclonal antibodies in formalin fixed, paraffin embedded decidual tissues from 10 normal first trimester pregnancy terminations and from 13 partial and 13 complete hydatidiform moles. Immunostained cells were fully quantitated and differences between subject groups were analysed using the Mann-Whitney test. T lymphocyte populations were further characterised using double immunohistochemical labelling.

Results—The numbers and percentages of CD3⁺ and CD4⁺ T cells were significantly increased in complete hydatidiform moles compared with partial moles and normal early pregnancy decidua. No differences were found in the number or percentage of CD8⁺ T cells. The CD8⁺ to CD4⁺ T cell ratio decreased significantly in complete mole compared with partial mole and normal decidua. The numbers and percentages of CD45RO⁺ cells increased significantly in both partial and complete hydatidiform mole compared with normal first trimester decidua. Double labelling confirmed that 50% of CD3⁺ T cells in complete and partial molar pregnancy coexpressed CD45RO, compared with 30% in normal pregnancy. Other leucocyte populations (eGLs, macrophages, B cells, and classical natural killer cells) did not differ between complete and partial mole and normal pregnancy decidua.

Conclusions—The presence of an increased population of activated decidual CD45RO⁺ T cells in complete and partial molar pregnancy suggests altered maternal immune responses against molar trophoblast.

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Keywords: decidua; T lymphocyte; hydatidiform mole

In normal pregnancy around 30% of stromal cells in decidualised endometrium are leucocytes.¹ Of these, up to 70% are phenotypically unusual endometrial granulated lymphocytes (eGL)¹; eGLs display natural killer (NK) cell cytotoxicity^{2–4} and produce various cytokines,^{5–9} but their in vivo function remains uncertain. T lymphocytes comprise approximately 10–20% of decidual leucocytes with a

CD8⁺ to CD4⁺ ratio of around 3:1¹⁰; these cells are also a potential source of cytokines in uteroplacental tissues. It has been proposed that a local T helper (Th) 2-type cytokine response favours fetal survival and successful pregnancy, whereas a Th1-type cytokine response may be detrimental.¹¹ Macrophages constitute approximately 20% of first trimester decidual leucocytes and may play a role in intrauterine immunoregulation and cytokine production.^{12–14} It has been suggested that cytokines produced locally by different leucocyte populations at the uteroplacental interface regulate normal trophoblast proliferation and invasion, although the precise mechanisms are unknown.^{11, 15–18}

Hydatidiform mole is a gestational trophoblastic disease characterised by abnormal trophoblast proliferation. The incidence of molar pregnancy varies widely from 1/85 pregnancies in Indonesia¹⁹ to 0.6–1/1000 pregnancies in North America and Europe.²⁰ Complete hydatidiform moles are usually diploid, with a 46,XX karyotype, and are of androgenetic origin, the majority resulting from the fertilisation of an anuclear ovum by a haploid sperm which then duplicates its own chromosome.^{21, 22} In contrast, partial hydatidiform moles are generally triploid, resulting from the fertilisation of a normal ovum by two spermatozoa.²³ Both partial and, more commonly, complete hydatidiform moles are associated with persistent trophoblastic disease. Since all the chromosomes are paternal in origin, a complete hydatidiform mole is an intrauterine allograft within the mother and may therefore be expected to stimulate maternal immune responses leading to fetal rejection. To date, there have been few and limited studies of maternal uterine immune responses in molar pregnancy.^{24–26} The present study was designed to characterise, quantify, and compare decidual leucocyte subpopulations and particularly T lymphocytes in complete and partial molar pregnancy with those in normal pregnancy to serve as a basis for the analysis of cytokine profiles in molar pregnancy. As well as analysing T lymphocyte subsets, expression of the CD45 isoforms CD45RA and CD45RO were analysed. CD45RA is expressed by naive or virgin T cells, whereas activated and memory T cells express CD45RO.²⁷

Methods

TISSUES

Formalin fixed, paraffin embedded decidual tissues from 10 normal first trimester pregnancy terminations and from 12 partial and eight complete hydatidiform moles were re-

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Table 1 Primary monoclonal antibodies used on paraffin embedded sections

Monoclonal antibody	Source	Specificity	Dilution	Pretreatment
CD3 (NCL-CD3-PS1)	Novocastra†	T cells	1 : 100	Pressure cooker (citrate buffer, pH 6.0)
CD4 (NCL-CD4-1F6)	Novocastra	Helper T cells, macrophages	1 : 50	Pressure cooker (EDTA buffer, pH 8.0)
CD8 (NCL-CD8-295)	Novocastra	Cytotoxic/suppressor T cells, NK cells, thymocytes	1 : 2000	Pressure cooker (EDTA buffer, pH 8.0)
CD20 (L26)	Dako‡	B cells	1 : 400	Pressure cooker (citrate buffer, pH 6.0)
CD45 (NCL-LCA-RP)	Novocastra	Lymphocytes, monocytes, eosinophils	1 : 100	Trypsin 5 min
CD45 RA (NCL-MB1)	Novocastra	B cells, monocytes and a small proportion of T cells	1 : 60	No pretreatment
CD45 RO (NCL-UCHL1)	Novocastra	Reactive T cells, monocytes, macrophages	1 : 400	Pressure cooker (citrate buffer, pH 6.0)
CD56 (NCL-CD56-1B6)	Novocastra	NK cells, a subset of activated T cells	1 : 500	Pressure cooker (citrate buffer, pH 6.0)
CD57 (NCL-NK1)	Novocastra	NK cells	1 : 10	Trypsin 10 min
CD68 (NCL-CD68)	Novocastra	Macrophages	1 : 50	Trypsin 10 min
CD79a (CD79 ; JCB 117)	Dako	B cells	1 : 50	Pressure cooker (citrate buffer, pH 6.0)
Cytokeratin (8/18) (NCL-5D3)	Novocastra	Simple and glandular epithelium	1 : 20	Trypsin 10 min
CD34 (QBEND/10, NCL-END)	Novocastra	Haematopoietic progenitor cells, vascular endothelium	1 : 25	Trypsin 10 min

†Novocastra Laboratories, Newcastle upon Tyne, UK.

‡Dako, High Wycombe, UK.

trieved from archive files of the department of pathology, Royal Victoria Infirmary, Newcastle upon Tyne. One partial and five complete hydatidiform moles were retrieved from archive files of the department of obstetrics and gynaecology, Siriraj Hospital, Mahidol University, Bangkok. All tissue blocks were sectioned at 3 µm and mounted on 3-aminopropyl-triethoxysilane (APES; Sigma) coated slides. At least one section of each tissue was stained with haematoxylin and eosin (H&E) to allow morphological assessment. Cases which showed histological evidence of decidual necrosis or inflammation identified by the presence of neutrophil polymorphs or plasma cells were not included in the study.

Figure 1 Decidual leucocyte populations in normal first trimester pregnancy and in partial and complete hydatidiform moles. Each bar represents the number of positive cells per ×400 field. Error bars = SEM. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

ANTIBODIES

A panel of 11 murine monoclonal antibodies was employed to characterise leucocyte subsets. Antibodies directed against CD34 (Qbend10) and cytokeratin 8/18 (5D3) were used to identify endothelial and trophoblast cells, respectively. All antibody specificities, dilutions, and pretreatments are detailed in table 1.

IMMUNOHISTOCHEMISTRY

Single labelling

Sections were labelled using a streptavidin-biotin complex immunohistochemical technique with no pretreatment, trypsin pretreatment, or pressure cooking in citrate buffer, pH 6.0, or EDTA buffer, pH 8.0. For each antibody the optimal dilution, incubation time, and pretreatment were established using similarly fixed and processed positive control tonsil and neuroblastoma tissues, as appropriate.

Sections were deparaffinised and rehydrated before quenching of endogenous peroxidase activity by incubation for 10 minutes with 1.6% hydrogen peroxide in methanol. For trypsinisation sections were incubated with 2.5% trypsin (Difco Laboratories) in distilled water, pH 7.8, containing 2.5% calcium chloride for five or 10 minutes. For pressure cooking sections were heated for one minute in citrate buffer, pH 6.0, or EDTA buffer, pH 8.0, as appropriate. For anti-CD4, CD8, and CD45RO, pretreatment by pressure cooking was followed by the use of an avidin/biotin blocking kit (Vector Laboratories) to block endogenous avidin and biotin activity. All sections were then overlain with 10% normal rabbit serum (NRS) in 0.1 M Tris/0.05 M saline (TBS), pH 7.6, for 10 minutes and then incubated with the primary antibody appropriately diluted in NRS for 60 minutes or overnight (anti-CD45). After washing twice in TBS, sections were incubated with biotinylated rabbit antimouse immunoglobulins (Dako), diluted 1:500 in NRS, for 30 minutes followed by streptavidin-biotin peroxidase complex (1:100 in NRS, Dako) for 30 minutes. The reaction was developed with 3,3'-diaminobenzidine (DAB; Sigma) containing 0.02% hydrogen peroxide for five minutes. Sections were lightly counterstained with Mayer's haematoxylin, dehydrated, cleared in xylene, and mounted in DPX synthetic resin (Raymond A Lamb Co). Tonsil sections were used as a posi-

Figure 2 Decidual leucocyte populations in normal first trimester pregnancy and in partial and complete hydatidiform moles. Each bar represents the percentage of the CD45⁺ (LCA⁺) population. Error bars = SEM. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

tive control for all antibodies except CD56 for which neuroblastoma sections were the positive control. Replacement of the primary antibody by normal serum was performed as a negative control for all specimens.

Double labelling

To assess expression of CD45RO by CD3⁺ and CD56⁺ cells, a sequential double immunohistochemical labelling technique was applied to selected normal and molar pregnancy samples. After the appropriate pretreatment, the sections were incubated with normal horse blocking serum (NHS) for 10 minutes and then sequentially with either anti-CD3 or anti-CD56 for 60 minutes, biotinylated horse anti-mouse immunoglobulins for 30 minutes, and

avidin-biotin peroxidase complex for 30 minutes (Vectastain Elite; Vector Laboratories). This reaction was developed with DAB as described above to produce a brown reaction product. After thorough washing in running water and a 10 minute incubation in NHS, the sections were incubated with anti-CD45RO for 60 minutes, with biotinylated antimouse immunoglobulins for 30 minutes, and finally with ABC alkaline phosphatase for 30 minutes (Vectastain alkaline phosphatase kit; Vector Laboratories). The reaction was developed with alkaline phosphatase substrate kit III (Vecta Blue; Vector Laboratories) at room temperature until the appropriate blue colour developed, usually after approximately 10 minutes. Double labelled sections were not counterstained and were mounted with BDH aquamount (BDH Co). Positive controls were performed for anti-CD3, anti-CD56, and anti-CD45RO antibodies and for CD45RO/CD3 and CD45RO/CD56 double labelling using normal tonsil. Single labelling for CD3, CD56, and CD45RO was performed on adjacent sections for each sample in order to rule out spurious double labelling. Negative controls were performed at each level of the double labelling procedure.

QUANTIFICATION

Positive cells, identified by the presence of brown membrane reactivity, were quantified at $\times 400$ magnification using a 10×10 mm graticule. At least 200 CD45 positive (LCA⁺) decidual stromal cells were counted in comparable areas of consecutive sections and the results were expressed as a percentage of LCA⁺ cells or as the number of positive cells per $\times 400$ field. For the anti-CD4 and anti-CD45RO antibodies which also stained macrophages, only small rounded lymphocytic cells were counted. Similarly, anti-CD3 also stained some eGLs and again care was taken to count only small rounded CD3⁺ lymphocytes with little cytoplasm. Double labelled sections were assessed by counting the number of single (brown or blue) and double (brown and blue) labelled positive cells in each field. Statistical analysis was performed using the Mann-Whitney test with the conventional $p < 0.05$ significance level.

Results

QUANTITATION OF DECIDUAL LEUCOCYTES

Normal pregnancy

The numbers and proportions of decidual leucocytes in normal first trimester pregnancy confirmed previous reports^{1,2} and are summarised in figs 1 and 2 and tables 2 and 3. In normal early pregnancy decidua, most LCA⁺ cells were CD56⁺ eGLs (mean (SEM), 51.2 (3.9)%), CD68⁺ macrophages (28.5 (5.5)%), and CD3⁺ T cells (27.6 (5.6)%).

Hydatidiform mole

In complete hydatidiform moles (CHM), both the number and percentage of CD3⁺ T cells were significantly increased compared with normal early pregnancy decidua (NP): number, NP 12.7 (2.0), CHM 20.9 (1.8),

Table 2 Decidual leucocyte counts in normal first trimester pregnancy and in partial and complete hydatidiform moles

Decidual leucocyte populations	Normal pregnancy	Partial hydatidiform mole	Complete hydatidiform mole
CD45 (LCA)	47 (37.5 to 73.3)	53.5 (29.6 to 80.4)	42.7 (27.4 to 86.0)
CD3	11.9 (5.8 to 27.2)	20.0 (9.0 to 35.7)	20.0 (8.7 to 31.0)
CD4	2.4 (1.2 to 5.3)	2.8 (1.2 to 12.0)	4.2 (1.8 to 18.3)
CD8	7.5 (2.7 to 9.8)	8.5 (2.3 to 18.0)	7.6 (2.8 to 16.4)
CD20	0.5 (0.0 to 1.5)	0.2 (0.0 to 1.8)	0.0 (0.0 to 1.7)
CD56	27.2 (12.0 to 37.3)	23.8 (14.1 to 41.7)	19.7 (11.4 to 46.0)
CD57	0.0 (0.0 to 0.8)	0.0 (0.0 to 0.3)	0.0 (0.0 to 0.0)
CD68	12.6 (6.7 to 28.0)	13.5 (7.0 to 25.8)	16.5 (8.7 to 21.0)
CD79a	0.0 (0.0 to 1.0)	0.2 (0.0 to 2.0)	0.2 (0.0 to 1.7)
CD45RA	0.0 (0.0 to 0.3)	0.0 (0.0 to 0.7)	0.0 (0.0 to 0.0)
CD45RO	3.9 (2.5 to 8.5)	13.3 (5.8 to 30.0)	13.3 (4.0 to 39.0)

The median and range of the number of positive cells per $\times 400$ field of each leucocyte population are shown.

Table 3 Decidual leucocyte counts in normal first trimester pregnancy and in partial and complete hydatidiform moles

Decidual leucocyte populations	Normal pregnancy	Partial hydatidiform mole	Complete hydatidiform mole
CD3	25.2 (8.9 to 72.1)	39.1 (21.2 to 50.0)	48.3 (23.4 to 59.1)
CD4	3.9 (2.5 to 12.4)	6.8 (3.5 to 15.0)	12.3 (3.9 to 29.2)
CD8	16.3 (4.0 to 23.5)	15.8 (4.2 to 32.4)	15.6 (6.6 to 32.3)
CD20	1.0 (0.0 to 2.5)	0.5 (0.0 to 4.5)	0.0 (0.0 to 5.5)
CD56	50.2 (31.7 to 79.2)	52.5 (21.9 to 60.7)	43.0 (36.4 to 64.3)
CD57	0.0 (0.0 to 2.2)	0.0 (0.0 to 0.6)	0.0 (0.0 to 0.0)
CD68	24.9 (9.9 to 74.3)	25.1 (11.4 to 47.2)	31.8 (3.3 to 46.4)
CD79a	0.0 (0.0 to 1.9)	0.4 (0.0 to 4.5)	0.4 (0.0 to 5.0)
CD45RA	0.0 (0.0 to 0.5)	0.0 (0.0 to 1.4)	0.0 (0.0 to 0.0)
CD45RO	7.6 (4.0 to 15.7)	24.8 (12.0 to 40.3)	38.6 (8.2 to 67.1)

The median and range of the percentage of LCA positive cells of each leucocyte population are shown.

$p = 0.012$; percentage, NP 27.6 (5.6)%, CHM 45.6 (3.1)%, $p = 0.0058$) (figs 1, 2, and 3; tables 2 and 3). The percentage of CD3⁺ T cells in complete hydatidiform moles was also increased significantly compared with partial hydatidiform moles, at 36.6 (3.0)%, $p = 0.0355$. Similarly, the number and percentage of CD4⁺ T cells were significantly increased in complete hydatidiform moles compared with normal decidua: number, NP 2.7 (0.5), CHM 6.5 (1.3), $p = 0.0058$; percentage, NP 5.6 (1.1)%, CHM 13.8 (2.1)%, $p = 0.0032$ (figs 1, 2, and 4; tables 2 and 3). Both the number and percentage of CD4⁺ T cells in complete moles also increased compared with partial moles (number, 4.5 (0.9), percentage, 8.0 (1.1)%) but only the percentage of CD4⁺ T cells increased significantly ($p = 0.0274$) (fig 2; table 3).

In contrast, there were no significant differences in the number or percentage of CD8⁺ T cells between complete and partial molar and normal pregnancy decidua (figs 1, 2, and 5; tables 2 and 3). Although the numbers and

percentages of CD3⁺, CD4⁺ and CD8⁺ T cells also increased in partial hydatidiform moles compared with normal early pregnancy decidua, the differences were not significant. The ratio of CD8⁺ to CD4⁺ T cells was significantly decreased in complete hydatidiform mole (1.6:1) compared with normal early pregnancy decidua (3.2:1, $p = 0.02$) and partial mole (2.4:1, $p = 0.0378$).

The numbers and percentages of CD45RO⁺ cells were significantly increased in both partial (number, 15.1 (2.2), $p = 0.0001$; percentage, 27.7 (2.7)%, $p = 0.0002$) and complete (number, 17.4 (2.9), $p = 0.0004$; percentage, 34.3 (4.7)%, $p = 0.0002$) hydatidiform mole compared with normal first trimester decidua (number 4.2 (0.6), percentage 8.8 (1.3)%) (figs 1, 2, and 6; tables 2 and 3). As in normal pregnancy CD45RA⁺ cells were virtually absent from partial and complete molar pregnancy decidua.

The number and percentage of CD56⁺ cells (eGLs) in decidua from complete hydatidiform moles did not differ significantly from those in partial moles and normal first trimester decidua. Although CD68⁺ macrophages increased in complete hydatidiform moles compared with normal early pregnancy decidua and partial hydatidiform mole, this difference was not significant. As in normal decidua, CD79a⁺ and CD20⁺ B cells and CD57⁺ natural killer cells were rare in molar pregnancy decidua (figs 1 and 2; tables 2 and 3).

DOUBLE IMMUNOHISTOCHEMICAL LABELLING

Double immunohistochemical labelling confirmed the increase in the numbers of CD3⁺ cells in complete hydatidiform moles compared with normal decidua. The numbers

Figure 3 Formalin fixed, paraffin embedded sections of decidua from (A) normal first trimester pregnancy; (B) complete hydatidiform mole immunostained for CD3. Note the increased number of T cells in complete hydatidiform mole.

Figure 4 Decidua from (A) normal first trimester pregnancy; (B) complete hydatidiform mole immunostained for CD4 showing increased numbers of CD4⁺ cells in complete hydatidiform mole.

Figure 5 Decidua from (A) normal first trimester pregnancy; (B) complete hydatidiform mole immunostained for CD8. Numbers of positive cells are comparable.

Figure 6 Decidua from (A) normal first trimester pregnancy; (B) complete hydatidiform mole immunostained for CD45RO. Increased numbers of CD45RO+ cells are seen in complete hydatidiform mole.

of CD3⁺ cells that coexpressed CD45RO increased in both partial and complete hydatidiform moles (table 4); up to 50% of CD3⁺ cells in molar pregnancy decidua coexpressed CD45RO, compared with 30% in normal early pregnancy decidua. In contrast, the number of decidual CD56⁺ cells that coexpressed CD45RO was comparable in normal pregnancy and in partial and complete hydatidiform moles (data not shown).

Discussion

Our analysis of decidual leucocytes in normal first trimester pregnancy is in agreement with previous studies of frozen tissues.¹ T cells accounted for a lower proportion of the stromal leucocytes compared with the CD56⁺ granulated lymphocytes but, as reported previously, many decidual T cells were CD45RO⁺ memory cells.²⁸ Decidual T cells have also been reported to express various activation markers including HLA-DR, CD69, and interleukin (IL)-2 receptor α and β , although expression of the α IL-2 receptor subunit has been disputed.^{29–33} Despite this evidence of activation, to date there have been no functional studies of decidual T cells in normal pregnancy. It has been proposed that Th2-type cytokines are necessary for successful preg-

nancy, whereas Th1 type cytokines may be deleterious.¹¹ Although it has been reported that a wide range of cytokines has been produced in human uteroplacental tissues,^{6 7 10 13 14 34} the cytokine profile of decidual T cells has not been fully defined. However, recent studies of T cell clones from early pregnancy decidua have indicated defective production of Th2 type cytokines in unexplained recurrent miscarriage compared with normal first trimester pregnancy.³⁵ It can be hypothesised that the abnormal trophoblast proliferation and invasion in gestational trophoblastic neoplasia may be associated with an abnormal decidual leucocyte profile and hence an altered local cytokine milieu.

As complete hydatidiform mole is androgenetic, it appears likely that molar trophoblast may stimulate an altered maternal immune response. Our study extends previous immunohistochemical studies of T cell populations in complete molar pregnancy decidua.²⁵ CD3⁺ T cells accounted for a larger percentage of the leucocytes, with a dramatic increase in the number and proportion of CD45RO⁺ cells. Moreover, the CD4⁺ T cell population which accounts for only a small proportion of decidual T cells in normal first trimester decidua was increased significantly in complete molar pregnancy, with an altered CD8:CD4 ratio of 1.6:1 in complete moles compared with 3.2:1 in normal pregnancy. These findings conflict with a previous study which reported that, although CD4⁺ T cells increased in complete molar pregnancy decidua, CD8⁺ T cells formed only a minority decidual T cell population, comprising only one quarter to one third of the total number of T cells.²⁵ This may reflect the use of different techniques: the latter

Table 4 Number of decidual CD3⁺ cells that express CD45RO in normal early pregnancy decidua and in partial and complete hydatidiform moles

Samples	Single labelled CD3 ⁺ cells/field	Double labelled CD3 ⁺ CD45RO ⁺ cells/field	Total number of CD3 ⁺ cells/field
Normal decidua	5.80 (0.80)	2.33 (0.33)	8.17 (1.17)
Partial hydatidiform mole	10.13 (0.63)	8.25 (0.50)	18.13 (8.8)
Complete hydatidiform mole	11.09 (1.92)	10.42 (1.25)	21.50 (3.17)

Values are mean (SEM) number of cells per $\times 400$ field (three to six fields were counted for each case).

study used frozen tissues and an indirect immunoperoxidase method, whereas the present study used a large number of paraffin embedded tissues with heat mediated antigen retrieval and a more sensitive avidin-biotin peroxidase technique. Moreover, CD4⁺ macrophages may have been included in the assessment of CD4⁺ T cells.²⁵

Our findings of altered activated CD45RO⁺ T cells in molar pregnancy decidua suggest that an altered maternal immune response may be induced by molar trophoblast. The mechanisms of T cell activation in normal pregnancy decidua are unknown. Extravillous trophoblast in normal pregnancy expresses a non-classical class I MHC antigen, HLA-G, which shows limited polymorphism.³⁶ MHC antigen expression by molar trophoblast has been less extensively investigated but evidence indicates similar MHC antigen expression by molar trophoblast.³⁷ Moreover, expression of HLA-G by choriocarcinoma cell lines has been well documented.³⁸ It is possible therefore that the invasive trophoblast within decidua leads to activation of decidual T cells. Despite the similarities in MHC expression, the present results suggest that this response may differ for molar trophoblast.

The function of T cells in normal pregnancy decidua is unknown, although it is reasonable to suggest that they contribute to local cytokine production. It is not known whether the increased numbers of CD4⁺ and CD45RO⁺ T cells reflect altered cytokine production which could enhance either cell-mediated cytotoxicity or humoral immunity in response to molar trophoblast. It has been reported that cytotoxic trophoblast cells in normal pregnancy and complete hydatidiform mole express Fas ligand (FasL) which may induce apoptosis of decidual activated CD95(Fas)⁺ T cells.³⁹ The presence of increased numbers of activated T cells in molar pregnancy decidua suggests that there may be an increased turnover rate of these activated T cells in response to the molar trophoblast. The absence of B cells, immunoglobulin, and complement deposition at the molar implantation site²⁶ suggests that humoral immunity is not the primary immune response mechanism. It appears unlikely that the alterations in decidual leucocyte populations noted in the present study reflect decidual inflammation. B cells were not a feature of molar pregnancy decidua and, moreover, none of the samples showed evidence of decidual necrosis, acute inflammatory cells, or plasma cells.

Further studies are required to clarify the role of decidual leucocytes, particularly T cells, in molar pregnancy. We are currently performing an immunohistochemical study of decidual T cell cytokine expression in molar pregnancy with the aim of elucidating the in vivo role of these cells.

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