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### MEDICAL HISTORY

- Stomach Ache
- Fungal Infection
- Itching
- Vaginal Discharge
- Loss of body weight
- Urinary Tract Infection
- Irritation
- Arthritis
- Inflammation
- Viral Infection
- Back Ache
- Immune Deficiency
- Other Complaints
### CLINICAL DETAILS

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### PATIENT CONSENT:

I hereby declare that I am aware of the nature and purpose of the above tests and the data provided by me is given with my own willingness.

Date:

Place:                  Sign:
**CLINICAL INFORMATION**

**STAGE OF THE DISEASE**

**CLASSIFICATION HISTOPATHOLOGICAL OBSERVATION:**  
- [ ] Border line  
- [ ] invasive

**EPITHELIAL TYPE**  
- [ ] Serous  
- [ ] Mucinous  
- [ ] Anaplastic  
- [ ] Endometriods  
- [ ] Other epithelial

**GERMLINE TYPE**  
- [ ] Yes  
- [ ] No

**OTHER COMPLAINTS**

**GENERAL EXAMINATION**

**BIOCHEMICAL MARKERS IDENTIFIED:**

**MODE OF TREATMENT**  
- Surgery  
- Chemotherapy  
- Radiation  
- **OTHERS:**
in a 68 year old woman. The FIGO stage was IIIC. The rare event coupled with her age suggests that she was less likely to be treated with standard therapy [14-16] and she was less likely to tolerate these treatments when received [17]. Taking into account her age factor she was less likely to be optimally debulked and also necessitate a post operative intensive care stay [18].

Thus in a view point not to worsen the prognosis for patient with such advanced ovarian cancer, neoadjuvant chemotherapy followed by interval cytoreduction was suggested. Thus caring for older women with advanced ovarian cancers presents unique challenges. Even after the achievement of optimal cytoreduction the survival rates are found to be very low. However careful selection of primary cytoreduction versus neoadjuvant chemotherapy can result in higher rates of optimal cytoreduction, low rates of post-operative death. Thus in patients where optimal debulking is not possible at presentation, neoadjuvant chemotherapy will be the best choice of treatment.

Consent
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Acknowledgements
The authors greatly acknowledge Dr. VJ Senthil, the Director of GVN Hospital, Tiruchirapalli, TamilNadu, India.

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4. Medical Microbiology, School of Life Sciences, Bharathidasan University, Tiruchirapalli, Tamil Nadu, India.

Authors’ contributions
SS and NSK designed the study and drafted the MS, SKG managed the treatment strategy, BK carried out the histopathological findings, VN and VK revised the manuscript critically. All authors have read and approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Received: 10 September 2010 Accepted: 3 June 2011 Published: 3 June 2011

References

Pre-publication history
The pre-publication history for this paper can be accessed here: http://www.biomedcentral.com/1471-2407/11/218/prepub
examination of the ascitic fluid showed cellular smear consisting of mixed inflammatory cells admixed with papillary and acinar clusters of eosinophilic cells with pleomorphic hyperchromatic nuclei thus suggesting a metastatic carcinoma.

Pelvic sonogram revealed a large tumour mass with solid and cystic components. Minimal ascites was noted. A subsequent computerized tomography (CT) scan of the abdomen and pelvis revealed heterogeneously enhancing mass lesion measuring 9 × 7.1 cm with solid and cystic areas and calcification in the retrovesical region (Figure 1A). The mass was found to be compressing on the right lower ureter leading to right hydrodeuteronephrosis. Multiple enlarged peritoneal nodules with a largest one measuring 10 × 6.3 cm were observed. Moderate free fluid in abdomen and pelvis with moderate right pleural effusion was observed. The diagnosis of the malignant transformation was suggested by the invasive growth of soft tissue components through the teratoma wall by CT scan images. Finally based on the clinical manifestations she was diagnosed as having FIGO stage IIIC of immature teratoma.

Due to unresectable bulky tumours and poor performance status the patient underwent neoadjuvant chemotherapy (NAC) followed by Interval Cytoreductive Surgery (ICS). Four cycles of combination of paclitaxel and carboplatin were administered every 3 weeks. ICS was performed in the 5th week after administration of the 4th cycle of NAC. Standard procedures of ICS consisting of total abdominal hysterectomy, bilateral salpingo-oophorectomy, omentectomy and maximal debulking of metastatic tumour was adopted. Following the procedure there was no residual macroscopic disease and she was transferred to high dependency.

The surface of the tumour appeared rough and congested. On cutting, the ovarian mass was full of sebaceous material and hair densely adherent to surrounding structures. Histopathological examination confirmed bilateral teratoma complicated with carcinosarcoma (Figure 1B) with heterogeneous rhabdoid elements (Figure 1C) Microscopically the left ovarian tumour displayed variable size cyst lined by multilayered malignant squamous cells (Figure 1D) with rhabdoid spindle cells, cytoplasmic clearing, mature atypical cartilage (Figure 1E), malignant tubules, small round cells with rosettes, bone marrow and neural bundle. In addition the focal area showed atypical giant bizarre cells. The observation of the right ovarian tumour displayed admixture of malignant, epithelial and mesenchymal elements. The epithelial layers showed variable sized islands of squamoid and polygonal mesenchymal cells and rarely showed tubular papillary structure. The stroma appeared to be a mixture of rhabdoid spindle cells, primitive mesenchymal cells, neural elements, adipocytic elements. Focal area showed pigmented cells.

The patient recovered well from surgery and was referred for oncological follow up and post-surgical chemotherapy (same regime as NAC). Given her age and performance status a surveillance approach was taken with regular clinical examinations, serial tumour markers and routine CT scans. The follow up studies showed no evidence of recurrence, regional or distant metastasis.

Conclusion
The prognosis of immature teratoma heavily depends on the FIGO stage. The other prognostic factors include tumour grade, growth pattern, capsular rupture and vascular invasion [6,7]. Age alone has been shown to be an independent predictor of survival in those with ovarian cancer [8]. The 2-year disease free survival for grade 1, grade 2 and grade 3 is 83%, 50% and 33% respectively [9]. The general consensus in the treatment of stage I immature teratoma is unilateral salpingo-oophorectomy for grade I disease, followed by adjuvant chemotherapy if tumour is grade 2 or 3 [10,11]. Several controversial arise regarding the standard treatment of immature teratoma among pediatric/adolescent population [12,13].

In spite of the controversial in treatment strategy, in the present study we have presented a rare event of bilateral ovarian teratoma complicated with carcinosarcoma...
Bilateral ovarian teratoma complicated with carcinosarcoma in a 68 year old woman: a case report

S Shanmughapriya¹,⁴, G Senthilkumar², K Balakrishnan³, N Vasanthi¹, K Vinodhini⁴ and K Natarajaseenivasan⁴*

Abstract

Background: Composing of less than 1% of all ovarian cancers, immature teratoma is a malignancy that mainly affects the young, and they present with advanced disease. The treatment of immature teratoma is conservative primary surgery usually involving unilateral salpingo-oophorectomy followed by combination chemotherapy.

Case presentation: Here we present a case of a 68 year old woman with bilateral ovarian teratoma complicated with carcinosarcoma. The patient was diagnosed as FIGO stage IIIC. She underwent neoadjuvant chemotherapy and interval cytoreduction followed by optimal cytoreduction. The post operative management strategies and gynaecological follow up studies revealed no evidence of regional or distant metastasis.

Conclusion: Thus the choice of initial treatment should be decided in a selective fashion depending on various prognostic factors in order to increase the survival of the patients.

Background

The term teratoma was derived from the Greek root teratos which means Monster [1]. Teratomas are the most common germ cell tumours (GCTs) composing of two or more germ layers (ectoderm, mesoderm or endoderm), derived from a pluripotent malignant precursor cell. Mature teratomas consist of adult-type differentiated components such as cartilage and glandular epithelium and immature teratomas contain tissues with partial somatic differentiation similar to that in foetal tissues [2]. Composing of less than 1% of all ovarian cancers, immature teratoma is rapidly progressing without treatment. The average age at diagnosis of this non-dysgerminatous tumour is 19 years. The symptoms are often non-specific, usually consisting of mass effect inflicting abdominal/pelvic discomfort [3]. The foundations of treatment for immature teratoma have been steadfast throughout decades: conservative primary surgery usually involving unilateral salpingo-oophorectomy followed by combination chemotherapy. We present here a patient with bilateral ovarian teratoma complicated with carcinosarcoma at the age of 68. Women at this age group are less likely to be optimally debulked, more likely to have high rates of chemotherapeutic toxicity and high rate of medical co-morbidities [4,5]. Taking into account all these considerations the present study was formulated to determine the impact and success of neoadjuvant chemotherapy and interval cytoreduction in the treatment of ovarian teratoma in an old age woman.

Case presentation

A 68 year old lady was referred to oncology outpatients in January 2010, with a month history of severe abdominal pain. Her past clinical history included no tubal ligation or hormone replacement therapy. She had previously given birth to a female child at her age of 21. The patient was fit and well with no significant past medical history apart from hypertension and diabetes. There was no family history of breast or ovarian carcinoma.

Physical examination revealed an abdominal pelvic mass with ascites and omental deposits. Blood analysis showed haemoglobin concentration of 11.3 g/dl while the rest of the analysis were normal including the carcinoembryonic antigen (CEA), alphafetoprotein and Cancer Antigen-125 (CA-125) (1.25 U/ml). The cytological
Viral and bacterial aetiologies of epithelial ovarian cancer

S. Shanmughapriya, G. SenthilKumar, K. Vinodhini, B. C. Das, N. Vasanthi & K. Natarajaseenivasan
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ARTICLE

Viral and bacterial aetiologies of epithelial ovarian cancer

S. Shanmughapriya · G. SenthilKumar · K. Vinodhini · B. C. Das · N. Vasanthi · K. Natarajaseenivasan

Received: 15 December 2011 / Accepted: 26 January 2012 © Springer-Verlag 2012

Abstract We sought to analyse the presence of human papillomavirus (HPV), Chlamydia trachomatis and cytomegalovirus (CMV) infection in women with epithelial ovarian carcinomas. Polymerase chain reaction (PCR)-based detection of microbial infections was carried out. A total of 39 tissue samples were analysed with consensus and type-specific primers for HPV, primers specific for the cryptic plasmid of Chlamydia and primers for glycoprotein B of CMV. The samples analysed showed 40%, 80% and 50% positivity for HPV, Chlamydia and CMV infection, respectively, in cancerous ovarian tissues. The HPV type detected was HPV 6, with its genome integrated to the host genome in case of both invasive and borderline tumours and existed episomally in healthy controls. The patients with Chlamydia (odds ratio [OR] 32; 95% confidence interval [CI] 3.33, 307.65) and CMV infection (OR 8; 95% CI 0.888, 72.10) are at significantly higher risk of development of ovarian tumours. The present study validates the theory of chronic infections and inflammation in the pathogenesis of epithelial ovarian cancer. Further seroepidemiological studies and large fresh tissue sampling may represent the real prevalence of infections among ovarian carcinoma patients. This study is the first of its kind in detecting the bacterial and viral aetiologies in the development of ovarian carcinoma among Indian women.

Introduction

Over 90% of ovarian cancer is described as epithelial in origin. Epithelial ovarian cancer (EOC) is a highly heterogeneous group of cancers demonstrating the fourth most common cause of death from cancer among women and has the highest mortality rate of the gynaecological cancers [1]. The 5-year survival rate is less than 40% because of the presentation of the majority of cases at an advanced stage. The aetiology and precursor lesions of even the major histotypes are poorly understood and have been predicted to be multifactorial. Several epidemiological and clinical risk factors are known to influence a woman’s lifetime risk for ovarian cancer. Reproductive behaviours and the use of hormonal therapies are the main clinical risk factors for ovarian cancer. In addition to this, 5–10% are attributable to genetic predisposition [2].

Microorganisms causing chronic inflammatory disease have become increasingly investigated in the last decade as possible cancer initiators/promoters [3]. Helicobacter pylori has been linked to gastric cancer, human papillomavirus (HPV) to cervical cancer, hepatitis B and C to liver cancer [4] and Chlamydia trachomatis as a co-factor primarily associated with squamous cell carcinoma of the cervix [5, 6]. The...
role of persistent infection leading to chronic inflammation in the pathogenesis of ovarian cancer has received very little consideration, although a history of pelvic inflammatory disease (PID) was correlated to a higher risk of ovarian cancer in a case–control study [7].

Given the incomplete biological explanations for the aetiology of ovarian cancer and the hypothesis of chronic infection and inflammation as a part of ovarian tumour pathogenesis, we report here the status of HPV, *Chlamydia* and cytomegalovirus (CMV) infection among ovarian carcinoma patients from the Indian population.

**Materials and methods**

The study was undertaken to investigate the presence of microorganisms in the ovarian tissues of women undergoing surgery due to suspected ovarian pathology. The study was approved by the human ethics committee of Bharathidasan University, Tiruchirappalli, Tamilnadu, India (Approved IEC No: DM/2010/101/21). Informed written consent was obtained from patients undergoing surgery.

Specimen collection

Fresh neoplastic and normal ovaries were collected aseptically in ice-cold 1× phosphate buffered saline (PBS) pH 7.4 during laparotomies performed at Dr. G. Vishwanathan Hospital (Dr. GVN Hospital), Tiruchirappalli, Tamilnadu, India. The collected samples were transported on ice to the laboratory for further analysis. Histopathological reports were obtained for all specimens from the histopathological department of Dr. GVN Hospital. A portion of each sample was used to extract DNA.

DNA extraction

DNA was extracted from tissue samples according to the SDS–proteinase K–phenol chloroform method as described previously [8]. Briefly, the tissues were minced, suspended in 500 µl of extraction buffer (10 mM Tris HCl [pH 8.0], 2 mM EDTA and 400 mM NaCl, 0.5% SDS, 0.4 µg proteinase K) and incubated overnight at 55°C. The DNA was extracted with phenol:chloroform:isoamyl alcohol (25:24:1) and precipitated with ice-cold absolute ethanol. The precipitated DNA was washed twice with 70% ethanol and resuspended in 50 µl TE buffer (10 mM Tris HCl [pH 7.5] and 0.1 mM EDTA).

HPV detection and typing

The integrity of DNA specimens was verified by the amplification of a 408-bp region of the p53 gene using Ex5F and Ex6R primers [9]. DNA samples were tested by the MY09/11 polymerase chain reaction (PCR) protocol [10, 11]. The PCR products negative for the primary amplification with MY09/11 primers were further assessed by secondary amplification with the general GP5′/GP6′ PCR system [12]. For HPV typing, type-specific primers (HPV 16, 18, 52, 6 and 11, and HPV 11), consensus primers PU-1 M/PU-2R (specific for HR-HPV 31, 33, 52b and 58), PU-31B/PU-2R (specific for other LR-HPV), consensus primers for E6 and E7 proteins, and consensus primers specific for E2 proteins were used. The amplified PCR products were visualised on 1% agarose gel stained with ethidium bromide and documented using a gel documentation system (Bio-Rad, USA). The primers used in the present study and the corresponding product size are given in Table 1.

**Chlamydia trachomatis** infection

*C. trachomatis* infection was detected by a nested PCR-based assay using KL5/KL6 and KL1/KL2 primers complementary to the sequence of the *C. trachomatis* cryptic plasmid [13]. The amplified PCR products of 350bp were visualised on 2% agarose gel stained with ethidium bromide.

**Cytomegalovirus infection**

CMV infection was detected by a nested PCR-based assay using external and internal primers specific for the CMV glycoprotein B gene. The expected product size of 150 bp was visualised by 2% agarose gel [14].

**Statistical analysis**

Odds ratios (ORs) and their corresponding 95% confidence intervals (CIs) were calculated by unconditional logistic regression and maximum likelihood estimation. Tests of statistical significance were based on differences in the log likelihoods, and all *p*-values are two-sided.

**Results**

Fresh ovary tissue samples were collected from 39 women (mean age, 55±15 years; range 40 to 70 years) undergoing laparotomies between May 2010 to March 2011. Of these, 24 had malignant ovarian lesions (12 serous adenocarcinoma, six mucinous adenocarcinoma and six endometrioid adenocarcinoma). Six women had benign ovarian lesions, of which all were of serous histotype. Nine women (including three patients with cervical squamous cell carcinoma) had normal ovaries. The entire extracted DNA was of sufficient quality and integrity as evidenced by amplification with Ex5F and Ex6R primers for the human p53 gene.
No specific amplification product was detected with the use of the MY09/11 primer. The further processing of the samples with GP5+/GP6+ primers showed the presence of HPV DNA in 15 (38.5%) of the ovarian tissues, accounting for three positivities in serous adenocarcinoma, three in endometrioid adenocarcinoma, six in serous cystadenoma and three in healthy tissues (Fig. 1A). No positivity was observed in mucinous histotype. The prevalence of HPV infection among invasive and borderline tumours and between ovarian carcinoma cases and controls did not vary significantly \((p>0.05)\). The results from the univariate analysis indicated that the odds of being HPV positive was one time greater for cases with epithelial ovarian carcinoma than for those with normal ovaries (OR 1.33; 95% CI 0.28, 6.39) (Table 2). Among the nine healthy control cases, three ovary samples were from patients with cervical cancer. All three samples analysed were not positive for HPV infection.

No amplification was observed for the HR-HPV consensus primer, LR-HPV consensus primers and type-specific HPV 16, 18, 52 and 11 primers, whereas the samples allowed the amplification of a 68-bp fragment with HPV 6 and 11-specific primer, showing a probable HPV 6 infection in the ovarian samples (Fig. 1B). The PCR amplification with primers specific for HPV regions detected no amplification for E2 and detected positivity for E6 and E7 in tumour ovaries and none for samples from healthy controls (Fig. 1C, D).

At baseline, \(C.\) \textit{trachomatis} DNA was present in 24 (80%) of the 30 tissues from ovarian cancer cases (Fig. 1E). Of the 24 \(C.\) \textit{trachomatis}-positive cases, 21 developed invasive epithelial ovarian carcinoma (12 serous, six endometrioid and three mucinous) and three were typed histologically as serous borderline tumours. All the tissues from controls at the time of the case diagnosis were negative for \(C.\) \textit{trachomatis} DNA. The results from univariate analysis indicated that the odds of being \(C.\) \textit{trachomatis}-positive was 32 times greater for cases with epithelial ovarian carcinoma than for those with normal ovaries (OR 32; 95% CI 3.33, 307.65). The prevalence of \(C.\) \textit{trachomatis} infection differed significantly \((p<0.05)\) between the cases and controls.

Approximately 15 (50%) of cases with detectable CMV DNA in their tissue specimens had ovarian cancer (Fig. 1F). For the cases that were CMV-positive, 80% of the infections were noted in cases with invasive and 20% among borderline tumours. None of the healthy controls were positive for CMV DNA. The results from the univariate analysis indicated that the odds of being CMV-positive were eight times greater for cases with epithelial ovarian carcinoma than for those with normal ovaries (OR 8; 95% CI 0.88, 72.10). The prevalence of CMV infection differed significantly \((p<0.05)\) between the cases and controls.

### Discussion

HPVs play a causal role in cervical cancer. HPV infection is also detected in other cancers of the female lower genital tract, including cancers of the vulva, vagina and perineum [15–17]. However, the role of HPV infection in the development of cancers in the upper genital tract is less clear. There exist discrepancies in the status of HPV infection among ovarian carcinoma [18–21]. The differences might be due to various reasons: (i) detection methods employed in the studies; (ii) the tissue sample may not be a representative of the entire ovary/tumour; (iii) If oncogenic HPV's were involved in the initial transformation of the ovarian epithelial cell, viral DNA sequences may stay episomal [22] and may be lost before
being carried in the genome of the host in an integrated state during tumour progression [23]. This may be an explanation for HPV being undetectable in tumour tissues with L1 consen-

Table 2 Manifestations of human papillomavirus (HPV), Chlamydia trachomatis and cytomegalovirus (CMV) infections in human ovary tissues

<table>
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<th>No.</th>
<th>Histopathological findings</th>
<th>No. of cases (N=39)</th>
<th>HPV-positive (%) (N=15)</th>
<th>C. trachomatis-positive (%) (N=24)</th>
<th>CMV-positive (%) (N=15)</th>
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<tr>
<td>1</td>
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<td>24</td>
<td>6 (25)a</td>
<td>21 (87.5)b</td>
<td>12 (50)c</td>
</tr>
<tr>
<td></td>
<td>Serious</td>
<td>12</td>
<td>3 (25)</td>
<td>12 (100)</td>
<td>6 (50)</td>
</tr>
<tr>
<td></td>
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<td>6</td>
<td>0</td>
<td>6 (100)</td>
<td>3 (50)</td>
</tr>
<tr>
<td></td>
<td>Endometrioid</td>
<td>6</td>
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<td>3 (50)</td>
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<td>6 (100)</td>
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<tr>
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<td>No atypia</td>
<td>9</td>
<td>3 (33.3)</td>
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a Significant risk (p<0.05) among cases and controls
b Significant risk (p<0.05) for invasive carcinoma in comparison with borderline
c Risk for invasive carcinoma but not significant (p>0.05)
also showed negative amplification for L1 primers, but the nested PCR system detected 40% positivity for HPV infection.

One of the hypothetical pathophysiological factors in the development of neoplasia of the ovarian tissues is the HPV infection ascending from the cervix to the fallopian tube and ovary. Interestingly, the ovary tissues from cervical carcinoma cases were found to be negative for HPV infection, thus, ruling out the hypothesis of ascending infection. The transmission of HPV types to the ovarian surfaces can be postulated that some of the papillomaviral DNA-coated sperm upon reaching the ovarian surface epithelium may be trapped into inclusion cysts and, thereby, give rise to ovarian cancer.

HPV types 6 and 11 are regarded as being of low risk for malignant conversion. Nevertheless, they have been shown to be associated with some invasively growing tumours [24–30]. The present study showed the infection of the ovary tissues with HPV 6. This can be due to alterations in sequences involving the long control region (LCR) in the HPV 6 genome that may result in such malignant transformations. Further, the positive amplification with E6 and E7 primers showed the selective pressure on HPV sequences that lead to retention of the E6 and E7 gene, although other regions of HPV including the E2 and L1 genes were being lost.

The results of the present study thus indicate the integration of HPV 6 into the host genome, resulting in the consequence of neoplastic transformation of ovarian epithelium and its probable existence as episomes in control tissues. The contribution of HPV 6 infection and integration to the development of ovarian carcinoma is not known. One possibility is that the episomal HPV 6 molecules may have undergone alteration, increasing their integration and, thus, their oncogenic potential. Another possibility is that integration events might have disrupted a cellular gene critical to normal cell proliferation.

There exists controversy in the role of *Chlamydia trachomatis* in ovarian malignancy. Earlier seroepidemiological studies found a positive correlation of plasma *Chlamydia trachomatis* IgG with ovarian tumours [3]. On the other hand, a previous study [21] reported all tumour specimens from ovarian carcinoma to be negative for the infection and, thus, reported it as a rare event. The present study has identified 70% of the tumour ovarian tissues with *Chlamydia* infection and none from healthy controls. In cervical carcinoma, *Chlamydia trachomatis* has been reported to cause severe inflammation of the cervix associated with metaplastic atypia of the transformation zone of the cervix [31, 32]. Taken altogether, the concept of chronic infections and inflammation as promoters/initiators of epithelial ovarian cancer development holds good.

Several mechanisms explain the involvement of *Chlamydia* in the development of cancer: (i) inhibits apoptosis by blocking the release of mitochondrial cytochrome C and caspase 3, which may then allow infected cells to escape CD8+ killer T-cell attack and are, thus, less likely to undergo the normal process of programmed cell death [33]; (ii) production of reactive oxygen species that may cause DNA damage and increase the risk of carcinogenesis [34, 35]; (iii) disrupts the normal structure of cadherin–catenin junctions, resulting in increased susceptibility to other infections [36].

Ovarian CMV infection is generally a rare finding and reported in autopsy studies of immunocompromised patients. The putative vascular/restrictive pathogenesis model describe the ovaries as plausible candidates to serve as CMV reservoirs [37–39]. Additionally, CMV DNA has been identified in arterial wall smooth muscle cells in both seropositive and seronegative healthy individuals [40]. This is because CMV enters the latent phase after a primary infection with its DNA incorporated into the host’s genome. Once infected, individuals probably carry the virus for life. Immunocompromised states like malignancies and chemotherapy can result in the reactivation of CMV. Furthermore, data on the role of ovaries in CMV latency is sparse. However, it is still unclear as to whether CMV reactivation in the ovaries is only a component of immunocompromised patients or the actual primary site of infection. To avoid misdiagnosis or underdiagnosis, ovarian involvement by CMV as a possibility of ovarian carcinoma in immunocompromised patients should be recognised.

### Conclusion

In conclusion, this study shows the presence of human papillomavirus (HPV), *Chlamydia trachomatis* and cytomegalovirus (CMV) infection in ovarian tissues. A prospective fresh tissue sampling and analysis of the presence of microbes followed by seroepidemiological associations can elucidate and authenticate the results of the present study. However, this study is the first of its kind to identify bacterial and viral aetiologies for ovarian carcinoma from the Indian population.

### Acknowledgement

K.N. is thankful to the Indian Council of Medical Research (ICMR; 5/13/88/06/NCD-III) for providing financial support to carry out the research work. S.S. is thankful to the ICMR for providing the Senior Research Fellowship (3/2/63/2011/NCD-III). The authors greatly acknowledge the Vice-Chancellor of Bharathidasan University and Dr. V.J. Senthil, the Director of GVN Hospital, Tiruchirappalli, Tamilnadu, India.

### Conflict of interest

The authors declare no conflict of interest.

### References


