dissemination patterns. Tumour biopsies were collected (range 4–15, median 9), placed in short-term cultures, treated with cisplatin (25μM overnight) and apoptosis/viability assayed. When relapsed, patients also had paired biopsies collected for genomic and phenotypic analysis. DNA was extracted from tumours (5 per patient, n=49 patients plus relapse samples) and Illumina Human OmniExpress genotyping performed. Allele-specific copy number (CN) was quantified using ASCAT. Genomic heterogeneity was quantified as the estimated number of CN aberration events distinct between each pair of tumour deposits. Clonal diversity within a patient's deposits was calculated using the difference between within-patient and between-patient heterogeneity.

Results Broad heterogeneity was detected in response to platinum treatment across cases at the phenotypic level in vitro (n=49), with higher variances in apoptosis induction observed in patients with platinum resistant disease. Genomic analysis revealed widespread variations in patterns of evolution for different patients' tumours, including the relationship between primary tumours and relapsed disease. Extensive variations in CCNE1, MYC and PTEN CN were observed across multiple tumours in the same patients, and overall higher CCNE1 CN associated with poorer patient outcome (p=0.038).

Conclusion Vast intra-tumoural heterogeneity is observed at the phenotypic and genomic level in HGSOC patients. Extensive copy number variations in genes such as CCNE1, MYC and PTEN across multiple disseminated samples within patients, indicates that sampling of a single tumour site does not accurately represent overall disseminated HGSOC biology and has implications for overinterpretation of studies relating to outcome and platinum-resistance.

Disclosures CF: advisory boards and honoraria from Roche, Tesaro, Sequana, Olympus, Astra Zeneca. Other authors have no disclosures to declare.

550 NOVEL 3D MODEL SYSTEMS TO ASSESS HETEROGENEITY IN RESPONSE TO PLATINUM THERAPY IN HIGH GRADE SEROUS OVARIAN CANCER

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Introduction/Background High-grade serous ovarian cancer (HGSOC) is the most common subtype of ovarian cancer, characterised by vast genomic instability and heterogeneity and acquired resistance to platinum-based chemotherapy. However, matching the most beneficial treatment options to patients is difficult to predict due to different platinum resistance mechanisms and limited effective predictive biomarkers. A study characterising intra-tumoural heterogeneity in HGSOC has identified variations in phenotypic responses to platinum treatment between different metastatic sites. In this study, we aim to develop novel clinically-relevant 3D ex-vivo models of HGSOC to investigate the effect of the local microenvironment on metastatic tumour cells' response to treatment, and potential use as a screening tool to predict drug responses.

Methodology Three different ex-vivo models were developed: organotypic, organoid and tumour slice. For organotypic and organoid models, tumour cells were extracted from metastatic deposits obtained from defined anatomical regions during upfront radical debulking surgery of advanced stage HGSOC patients. Organotypic models were assembled using normal omental stromal cells embedded in Collagen-1 and tumour cells were added. Organoid models were propagated from tumour cells and embedded in basement membrane extract. For slice culture models, tumours were sliced into 350μm sections using a vibratome and cultured on cell culture inserts. All models were treated with cisplatin and assessed for apoptosis and viability read-outs.

Results Organotypic models showed that tumour cells cultured in 3D showed heterogeneity in response to cisplatin treatment, data showed a trend towards reduced response to treatment within 3D models compared to 2D (n=8). Changes in patterns of response to treatment between samples from 2D to 3D within the same patient was also demonstrated (n=5). Organoid models were successfully propagated from different metastatic sites and maintained long term growth (>15 passages). Histological read-outs for slice culture models demonstrated slices from different metastatic sites maintained viability in culture for up to 5 days.

Conclusion We have established growth, drug treatment conditions and assay read-outs for 3 different ex-vivo models of metastatic HGSOC. We have established that organoid culture must be generated within 24hours of tumour cell extraction. Furthermore, both fresh and viably frozen tumours can be used to generate organotypic and organoid models. The broader implication of establishing clinically-relevant complex tumour models as routine methodologies for screening novel therapeutics and capturing the complex heterogeneity of individual patients, may lead to better development of therapeutic strategies including tumour/microenvironment combination strategies and also better personalisation of therapy for patients with HGSOC.

Disclosures CF: advisory boards and honoraria from Roche, Tesaro, Sequana, Olympus, Astra Zeneca. Other authors have no disclosures to declare.

Trophoblastic diseases

393 A CLINICAL AUDIT OF MOLAR PREGNANCIES AND GESTATIONAL TROPHOBLASTIC NEOPLASIA CASES OVER 1YR IN A TERTIARY CARE HOSPITAL OF EASTERN INDIA WITH RESPECT TO THE INCIDENCE OF DISEASE, FACTORS RELATED TO ETIOPATHOGENESIS, DIAGNOSIS AND MANAGEMENT

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Introduction/Background A Clinical Audit of molar pregnancies and gestational trophoblastic neoplasia cases over 1yr was conducted at Kolkata Medical College & Hospital, a tertiary care hospital of Eastern India with respect to the incidence of disease, factors related to etiopathogenesis, diagnosis and management and effects on maternal morbidity and mortality.

Methodology A total of 10000 patients attended this institution during 2017 june to 2018 june for pregnancy or its complication in department of gynaecology & obstetrics.85 molar

pregnancies and their sequelae were followed up in medical oncology. The data was collected from out patient & in patient tickets & admission registrars and was analysed by descriptive statistics.

Results Most cases were seen amongst the second gravida 40%, Hindus 53.3%, low socio-economic strata 72%.Predominant Blood group was B 53%. Hemoglobin below 10 mg/dl was seen in 94%. 21.33% of patients had haemoglobing was below 6 gm/dl.Most of the patients of Hydatiform Mole (50%) were diagnosed within a period of ammenorhea of 8–12 weeks with 70% of cases diagnosed with amenorrhoea of less than 16 weeks. 18% of patients were diagnosed after a period of amenorrhoea of greater than 20 weeks. The most common presenting symptom in cases of Hydatiform Mole was Bleeding per vagina 74%. Features of Hyperthyroidism & respiratory distress were seen in 5% of patients. The most common signs were pallor 65%, pre-eclampsia were seen in 17.33%% of patients.

Suction & Evacuation 58.66%% with Oxytocin infusion was the predominant mode of management in cases of Hydatiform Mole. Ligation was done in one patient considering the risk of repeat molar pregnancy in future conception.

Modes of diagnosis were clinical (74%), & USG in 68%.

Persistent Gestational Trophoblastic Disease and Choriocarcinoma were diagnosed during follow up by symptoms of irregular bleeding P/V, elevated beta HCG titre and abnormal USG pelvis and chest X-Ray.

Chemotherapy was the predominant mode of treatment of GTT. hysterectomy was done in 2 patients of Invasive mole.

Single agent chemotherapy with Methotrexate in 20 patients 83.33% i.e low risk GTT. EMA-Co regimen was the preferred multiagent chemotherapy used in 4 patients 18% (upfront) and in 2 patients progressing on methotrexate, surgery in 1patient not responding to EMACO or EMA-EP.

Toxicity of chemotherapy was predominantly, Nausea & vomiting (38.89%) mucousitis (27.78%).Hepatotoxicity and infection was seen in 11.11% of patients. Grade3/Grade4 toxcity was nil.

Conclusion Though the proportion of molar pregnancies & gestational trophoblastic neoplasia is not much in comparison to the heavy attendees in the gynaecology and obstetrics opd but they represent a highly curable one with minimally intense chemotherapy thus avoiding unnecessary hospital stay due to chemotoxicity.

Disclosure I do not have any conflict of interestwith any person or organization.

422 NEW CHALLENGES IN THE MANAGEMENT AND FOLLOW-UP OF MOLAR PREGNANCY

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Introduction/Background Hydatidiform mole (HM) is the premalignant form of gestational trophoblastic neoplasia. This entity is of clinical and epidemiological interest because of its potential for significant consequences for women's reproductive health.

Methodology This is a retrospective study of all cases of molar pregnancy managed in County Emergency Clinical Hospital of Oradea from 1st January 2019 to 30th August 2020. The details of maternal characteristics, clinical presentation, tumor type and management were studied.

Results We diagnosed 17 cases of molar pregnancy during this period of 20 months and wemanaged 5364 deliveries. We also recorded 614 miscarriages and histopathological exams performed. The mean age of the patients with HM was 27,17 years old, with the highest incidence in patients between 15–20 years (29,41%). From allthe cases of HM, 94,12% were diagnosed in first trimester of pregnancy and we had only one case in second trimester pregnancy. Amenorrhea followed by vaginal bleeding was the common symptom in 14 cases (82,3%). A number of 12 patients were admitted because of exaggerated forms of hyperemesis gravidarum. The ultrasound exam showed the size of the uterus larger than the amenorrhea and ovarian lutein cysts were present in almost half of cases. All the patients have had higher then normal values of HCG. In our department all the cases were managed with dilation, suctionand mild curettage when necessary, except one case, finalized with hysterectomy, because of the molar type and the patient's age. Histopathological exam was performed in all cases. In 11 cases (64,7%) partial hydatidiform mole was diagnosed and in 6 cases complete HM (25,3%). A serial determination of HCG until normal values was always recommended, but we could not do the correct monitoring up to 6–12 months in 7 cases, related to the migration of the population in the region.

Conclusion Molar pregnancy has remained an important cause of maternal morbidity and mortality. There is need for early diagnosis, for proper treatment and follow-up of this condition. Due to the frequent use of ultrasound scanning, the diagnosis of hydatidiform mole could be made early in pregnancy. If hydatidiform mole is suspected, the quantitative estimation of serum level ofHCG should be done. After an appropriate treatment, it is always necessary to follow-up the patient and in present this is a new challenge because the population migration due to new socio-economic conditions and modern life.

Disclosures I have nothing to disclose.

434 METASTATIC POSTMOLAR CHORIOCARCINOMA OF THE SKIN

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Introduction/Background Gestational choriocarcinoma is a malignant tumor arising from trophoblastic cells with the lung and the vagina as its common sites of metastasis. Skin metastasis is known to be extremely unusual.1 This paper outlines the case of a 45-year-old multigravida who manifested with occasional nonproductive cough; multiple cutaneous lesions in left flank, right triceps area, upper back, and infraumbilical areas associated with neurologic symptoms, two years after undergoing hysterectomy for a molar pregnancy. Skin biopsy of the left flank masses showed metastatic gestational choriocarcinoma; and she had elevated B-hCG (309,245 mIU/mL), and lung, brain, liver, and right adrenal metastases on imaging studies. She achieved remission after treatment with Etoposide Cisplatin induction chemotherapy, high-dose EMACO with concurrent whole brain irradiation, and ten cycles of EMACO.