XXIII WORLD CONGRESS ON GESTATIONAL TROPHOBLASTIC DISEASE

Capecitabine as an Alternative Chemotherapy in a Case of Low-Risk Gestational Trophoblastic Neoplasia with Methotrexate Resistance and Actinomycin-D Induced Hypersensitivity Reaction

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Introduction:

Third-Line Dilemma

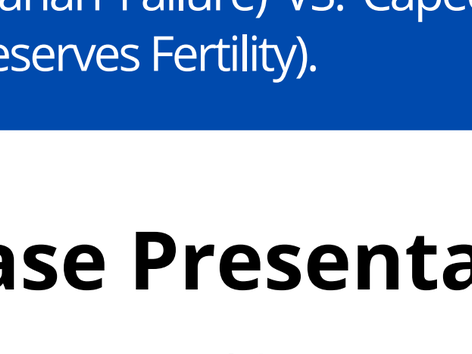
1st Line: Methotrexate (MTX)
Result: Resistance.

2nd Line: Actinomycin-D (Act-D)
Result: Severe Hypersensitivity Reaction.

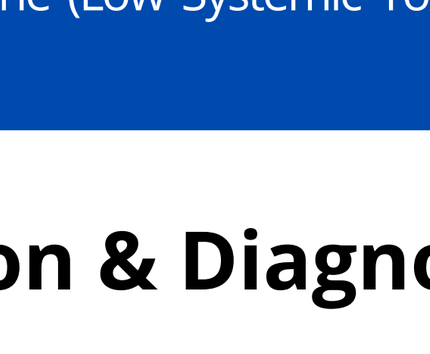
3rd Line: Multiagent Chemo (EMA/CO) (High Toxicity, Ovarian Failure) VS. Capecitabine (Low Systemic Toxicity, Preserves Fertility).

Case Presentation & Diagnosis

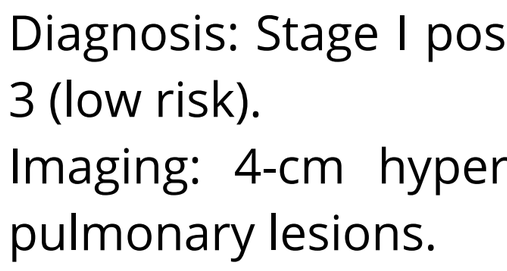
- 28-year-old primigravida.
- Diagnosis: Stage I postmolar GTN, FIGO risk score 3 (low risk).
- Imaging: 4-cm hypervascular uterine mass; no pulmonary lesions.



A. Color doppler



B. Spectral Doppler



C. Right ovarian mass

Mechanism of Action:

How Capecitabine Targets GTN

Oral Capecitabine (Prodrug)

↓ Activated by Thymidine Phosphorylase (TP)

High TP concentrations in Trophoblastic Tissue

↓ Localized conversion

5-Fluorouracil released intratumorally

↓

Blocks DNA synthesis with minimal systemic toxicity

Clinical Course and Treatment Response

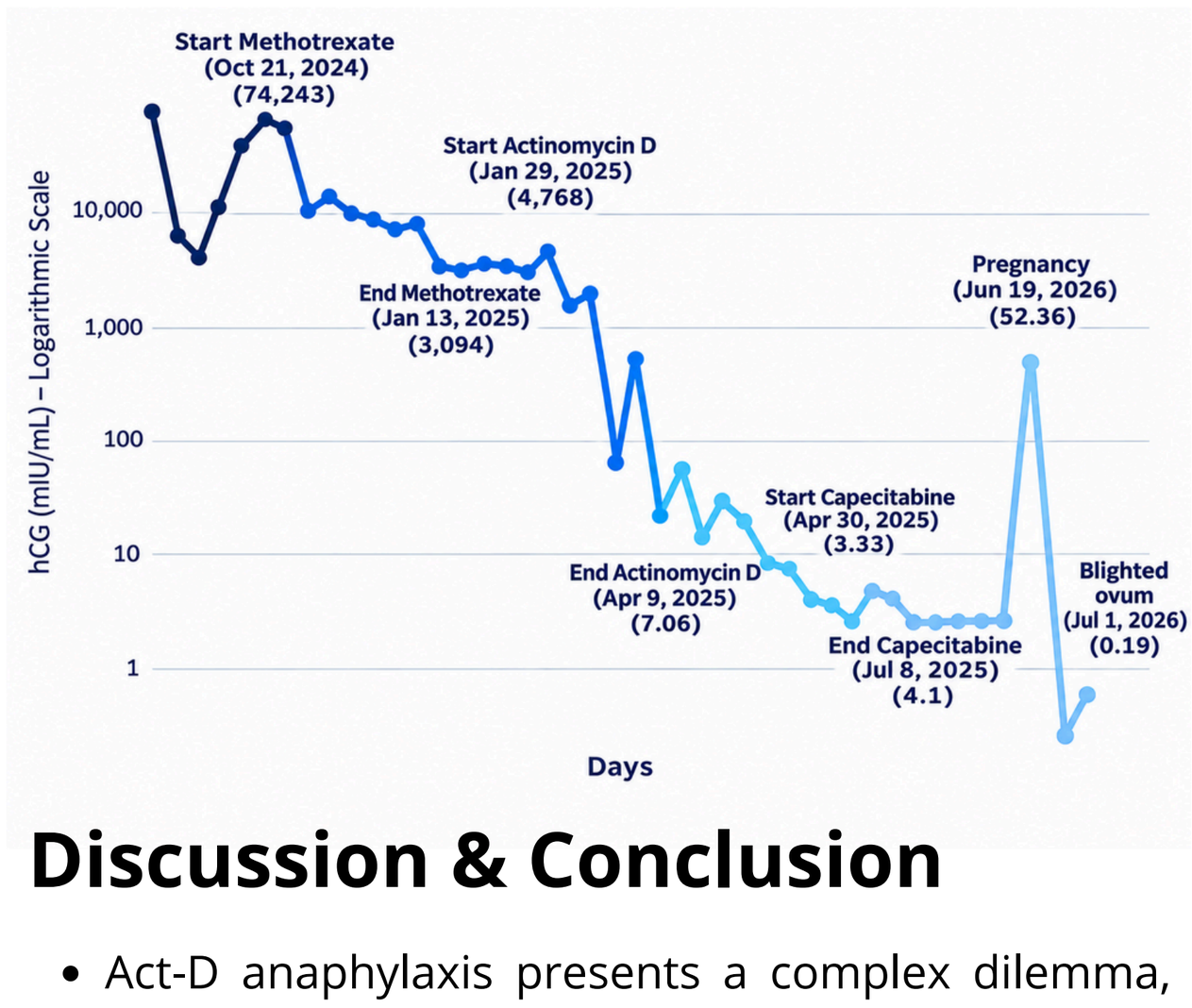
Start MTX: Resistance identified by hCG plateau (3,094 mIU/mL)

Start Act-D: hCG drops, but complicated by hypersensitivity reaction (hypotension, dyspnea, dysphagia) at cycle 8

Start Capecitabine: Oral administration (2,500 mg/m2/day)

Normalization: Normalising hCG below 5 mIU/mL during capecitabine treatment

Pregnancy: Spontaneous conception 1 year post-treatment, proving fertility preservation



Discussion & Conclusion

- Act-D anaphylaxis presents a complex dilemma, contraindicating EMA/CO regimens.
- Immunotherapy is effective but currently limited by high cost and low availability.
- Oral capecitabine is a highly effective, viable salvage option for low-risk GTN with MTX/Act-D resistance or intolerance.

Conclusion: It provides rapid biochemical response and sustained remission, offering minimal systemic toxicity while successfully maintaining future conception capacity.

Selected Literature