SUMMARY OF CONSENSUS RECOMMENDATIONS

Detecting Concurrent Carcinoma

Gynecologists should attempt to exclude concurrent carcinoma in individuals with a working diagnosis of EIN–AEH. Hysteroscopic examination with further sampling of the endometrium is the most accurate method for detecting a concurrent carcinoma.

Surgical Management

Hysterectomy is the definitive treatment for EIN–AEH. Gynecologists should not perform supracervical hysterectomy for the treatment of EIN–AEH.

Gynecologists should not perform endometrial ablation (thermal or electrocautery) for EIN–AEH due to high persistence and recurrence rates, as well as potential difficulty in evaluating future bleeding episodes.

Nonsurgical Management

Clinicians should recommend progestational agents as treatment for EIN–AEH for patients in whom hysterectomy is not an option.

Data on the superiority of either oral or intrauterine progestational agents are lacking, though limited data suggest that intrauterine progestational administration may be associated with a higher rate of disease regression when compared with oral administration alone in patients with EIN–AEH.

There is insufficient evidence to recommend any one formulation of oral progestational agent over another.

Follow-up

For those initially treated with progestational agents, gynecologists should perform repeat histologic assessment for response to treatment for EIN–AEH within 3–6 months.

After initial progestin treatment, gynecologists may consider long-term maintenance therapy with progestational agents for patients with continuing risk factors for endometrial cancer.

Counseling Patients on Lifestyle Modifications

Gynecologists and other clinicians should counsel patients that lifestyle modification resulting in weight loss and glycemic control can improve overall health and may decrease the risk of EIN–AEH and endometrial cancer.

EIN–AEH, endometrial intraepithelial neoplasia or atypical endometrial hyperplasia.

for atypical hyperplasia or EIN include crowded glandular architecture and altered epithelial cytology distinct from the surrounding endometrium or entrapped nonneoplastic glands or both. Desirable criteria include the following: loss of immunoreactivity for PTEN, PAX2, or mismatch repair proteins. In the EIN schema developed by the International Endometrial Collaborative Group, endometrial precancer is termed endometrial intraepithelial neoplasia, and pathologic criteria were used to develop three disease categories: 1) endometrial hyperplasia, 2) endometrial intraepithelial neoplasia, and 3) adenocarcinoma (7). The American College of Obstetricians and Gynecologists (ACOG) does not recommend one terminology schema over another. This topic has been updated to reflect newer data on the management of EIN–AEH. This guidance applies to those patients who have already received a diagnosis of EIN–AEH.

Epidemiology

Estrogenic stimulation of the endometrium, unopposed by progestins, causes proliferative glandular epithelial changes or hyperplasia. Hyperplasia, due to prolonged exposure to estrogens, is biologically distinct from the precancerous lesion EIN–AEH. The precursor lesion of type I endometrioid adenocarcinoma is EIN–AEH. Making the distinction between hyperplasia and EIN–AEH is important, because each condition has differing cancer risks and requires different interventions to avoid undertreatment or overtreatment.

Cancer of the endometrium is rising in the United States, with an estimated incidence of 66,200 cases and 13,030 deaths in 2023 (8). Disparities in care linked to differences in race, ethnicity, geography, and socioeconomic status have been associated with poorer