GASTRIC CANCER DEVELOPMENT MECHANISM FROM INTESTINAL METAPLASIA USING METABOLOMIC PROFILING

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A metabolomic study using LC-MS/MS identified distinct metabolic profiles between intestinal metaplasia and gastric cancer tissues in 37 patients, highlighting steroid hormone biosynthesis and lysine degradation pathways as significantly altered during carcinogenesis.

KEY POINTS

- Tissues from 37 gastric cancer patients were analyzed using PCA and PLS-DA, which significantly separated gastric cancer from intestinal metaplasia based on metabolic profiles
- Steroid hormone biosynthesis was the most significant pathway, with most related metabolites decreased in gastric cancer compared to intestinal metaplasia
- Metabolites promoting cancer cell growth including s-Adenosyl-L-Methionine, S-adenosylmethioninamine, nopaline, and N-acetylputrescine were increased in gastric cancer
- Stage-dependent analysis showed sulphate and indole-3-acetaldehyde were altered in early gastric cancer, while creatinine and L-lysine were altered in late-stage disease
- The authors suggest these metabolites could serve as potential biomarkers for gastric cancer detection pending further validation research

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