## **Online suppl. Table 1**: Clinicopathological features of individual tumors (age, gender, locus, size of mucosal lesion, pT, pN, and excision type) and immunohistochemical and in situ hybridization results (p53, MMR enzymes, EBV, mucin markers) of individual samples. (xlsx28KB)

## **Online suppl. Table 2**: Frequencies of copy-number gains and amplifications of receptor tyrosine kinases and representative growth-related genes on chromosome 7 in clusters A and B. (xlsx15KB)

## **Online suppl. Fig. 1**: Penetrance plots of individual samples. The sample orders in clusters A and B and LOM corresponds to the sample order in Fig. 1. LOM: loss of mismatch repair enzyme expression; ROM: retention of mismatch repair enzyme expression. (pdf282KB)

## **Online suppl. Fig. 2**: Comparison of mucin phenotype composition between mucosal (M) and invasive/metastatic (I + LN) samples in the cluster A and the cluster B tumors. Based on the online suppl. Table 1, mucin phenotype was classified into gastric predominant phenotype (G, G≥I), intestinal dominant phenotype (I, I>G), and unclassifiable phenotype (N). **a.** mucin phenotype composition of M and I + LN parts in clusters A and B. **b, c**. Statistical analyses of the differences in frequency of gastric predominant expression (b) or intestinal predominant expression (c) between M and I + LN parts and between clusters A and B. (pdf389KB)

## **Online suppl. Fig. 3**: Outline of 32 genes that showed significantly different copy-numbers between clusters A and B. The mean copy number alterations (CNAs) are expresses as Tumor/Reference (T/R) signal intensity ratio. In the gene function column, tumor suppressor genes and proto-oncogenes are marked with green and pink background, respectively. In the mean T/R ratio columns, copy-number gains and losses are marked with pink and green background, respectively. Concordant pairs of CNA and gene function are marked with black frame. (xlsx15KB)