Hypervirulent Klebsiella pneumoniae (hvKp)-associated pyogenic liver abscess is typically linked to K1/K2 capsule serotypes and canonical lineages such as ST23, ST65, and ST86. Cases outside these lineages are rare but pose significant public health concerns. We investigated an unusual liver abscess in an immunocompetent patient caused by hvKp sequence type 111 (ST111). Five isolates (KP1-KP5) were recovered, all belonging to the same clonal lineage. Genomic analysis revealed a 181 kb IncHI1B/IncFIB virulence plasmid encoding rmpADC and the salmochelin cluster (iroBCDN), but lacking aerobactin (iuc) and rmpA2 gene loci. Notably, this plasmid also carried the yersiniabactin locus (ybt), which is typically located on the chromosome. Comparative genomics indicated that similar ST111 strains have been reported only in China, suggesting clonal expansion of a previously unrecognized hvKp lineage. Functional assays demonstrated that plasmid curing abolished the hypermucoviscous phenotype, reduced capsule production, and completely attenuated virulence in murine models, confirming the plasmid as the major driver of hypervirulence. Interestingly, plasmid loss enhanced biofilm formation and relieved growth burden, highlighting fitness trade-offs associated with virulence. This study documents a rare case of hvKp ST111 liver abscess and identifies a novel plasmid conferring hypervirulence. Our findings expand the known diversity of hvKp and underscore that non-canonical lineages can acquire potent virulence determinants enabling severe disease. Continued clinical and genomic surveillance is essential to detect emerging hvKp clones before widespread dissemination.