

**Abstract:**

Extended-spectrum beta-lactamases (ESBLs) were present in high proportions of *Escherichia coli* (25% [9 of 36]) and *Klebsiella pneumoniae* isolates (17% [9 of 52]) causing pediatric septicemia at a tertiary hospital in Tanzania. Patients with septicemia due to ESBL-producing organisms had a significantly higher fatality rate than those with non-ESBL isolates (71% versus 39%,  $P = 0.039$ ). This is the first report of the CTX-M-15 genotype of ESBLs on the African continent and the first observation of SHV-12 genotype in an isolate of *Salmonella enterica* serotype Newport.

Resistance to beta-lactam antibiotics was demonstrated in *Escherichia coli* even before penicillin was released for clinical use. In the 1960s, the first plasmid-transferable beta-lactamase was discovered and named TEM-1 after Temoniera, the Greek girl who harbored the *E. coli* isolate from which the enzyme was obtained. Since the 1980s, a large number of plasmid-transferable extended-spectrum beta-lactamases (ESBLs) capable of inactivating extended-spectrum cephalosporins has been discovered (6). Most of the ESBLs are derived from TEM-1 and SHV-1 (sulfhydryl variable) by mutations. ESBLs have spread widely and have become a major cause of nosocomial infections associated with high mortality rates, particularly in serious infections such as septicemia. In Africa, ESBLs have been reported in Egypt, Tunisia, Morocco, Senegal, Nigeria, South Africa, and Kenya but not previously from Tanzania. In the present study, we investigated the prevalence and clinical implications of ESBL production in *E. coli*, *Klebsiella pneumoniae*, and *salmonellae* causing septicemia in infants and children admitted to a tertiary teaching hospital in Tanzania.