Background: Emergence of panresistant superbugs is a serious threat to global public health security. Panresistant infections are associated with prolonged hospitalization, delayed recovery, disseminated infections and mortality. Antimicrobial misuse leading to selection pressure, transfer of resistance, inadequate detection of panresistance and inadequate infection control measures may contribute to a “Microbial Holocaust” by panresistant microbes. Detection and characterization of panresistance requires specialized testing modalities which may be beyond the scope of routine diagnostic laboratories. The present study was undertaken to confirm and characterize panresistant phenotypes.

Material/methods: Two hundred isolates identified by conventional methods and found resistant to all empirical antimicrobials by Kirby Bauer method were included. Identification was confirmed by Vitek 2 compact and susceptibility tested via microbroth dilution for Beta-lactams (BL), BL with beta-lactamase inhibitors (BL/BLI), carbapenems, aminoglycosides, quinolones, sulfamethoxazole-trimethoprim, nitrofurantoin, tigecycline and colistin using Vitek AST-N280 card. E-test were used to confirm Extended Spectrum β-Lactamase (ESBL), Metallo-β-Lactamase (MBL); and MICs of Tigecycline and Colistin. Phenotypic combined disk tests (PCDT) for ESBL + AmpC; and Klebsiella pneumoniae carbapenemase (KPC) + MBL (both Rosco Diagnostica, Denmark) were interpreted as per CLSI and/or European Committee on Antimicrobial susceptibility testing (EUCAST) guidelines.

Results: Panresistant Klebsiella pneumoniae, Klebsiella oxytoca, Burkholderia cepacia, Pseudomonas aeruginosa, Acinetobacter baumanii and Escherichia coli were isolated from blood samples from oncology patients and transplant recipients; multiple injuries and burn wounds; and endotracheal aspirates from critical care patients. 27 (54%) were found resistant to all drugs while 23 (46%) were found susceptible to tigecycline and colistin. Multiple resistant phenotypes such as ESBL+ carbapenemases (metallo or KPC), resistance to carbapenemases (impermeability) and aminoglycoside resistance (AAC 6’ +) were frequently seen in Klebsiella pneumoniae and Klebsiella oxytoca. Carbapenemase (metallo or oxa), acquired penicillinase + resistance to carbapenems (impermeability) and high level resistance + resistant carbapenems (impermeability) were also seen in Pseudomonas aeruginosa. Resistant carbapenems (impermeability), carbapenemase (metallo or oxa) and high level resistance + resistant carbapenems (impermeability) were seen in Acinetobacter baumanii. ESBL+ carbapenemases (metallo or KPC) were seen in E. coli. Coexistent ESBL, AmpC and MBL alongwith resistance to polymyxins and tigecycline were observed by PCDT.

Conclusions: Emergence of panresistant superbugs in cancer, burns, wounds, critical care and transplant recipients can cause significant morbidity and mortality. Multiple resistance phenotypes may surpass routine identification and result in treatment failures. Coexistent ESBL, AmpC and MBL along with resistance to Tigecycline and Colistin leaves no viable options. While antimicrobial stewardship, stringent infection control practices and rapid response to outbreaks may help control their emergence and transmission, grave issue of their exploitation as bioweapons remain at large.