Background: Acute Myeloid Leukemia (AML) is most common in adults and is associated with rapid progression and high mortality with delayed treatment. Despite therapeutic advances, its prognosis remains poor in adults compared to the young. Mesenchymal Stem Cells (MSCs) are reported to have anti-cancer properties but their effects against AML are hitherto not reported. We in the present study evaluated the anticancer properties of human umbilical cord mesenchymal stem cells (hUC-MSCs) against an AML cell line (K562) in vitro using co-culture system.

Methods: Human umbilical cords were collected following Institutional Ethical Committee approval [a33-15/KAU]. hUC-MSCs were derived using explant culture method and K562 was obtained from ATCC. Both hUC-MSCs and AML cells were cultured under standard culture conditions and respective cell proliferation assessed. Derived hUC-MSCs were characterized for their stemness using cell morphology and MSCs related CD markers expression (FACS). Anti-cancer effects of hUC-MSCs were evaluated by co-culture with AML cells plated at equal seeding density (2×10^4 cells/well) in a 24-well plate followed by culture for 24h, 48h and 72h. Changes in cell morphology and cell proliferation (MTT assay) were assessed.

Result: Derived hUC-MSCs were plastic adherent and showed short fibroblastic morphology resembling MSCs. K562 cells showed spherical morphology like undifferentiated blast cells. hUC-MSCs were positive for CD73, CD105, CD29 and CD90 while they were negative for CD34, CD45. In co-culture, K562 cells clustered onto the hUC-MSCs and showed signs of cell death. K562 also demonstrated statistical decrease in cell proliferation by 2.03%, 36.92% and 16.38% at 24h, 48h and 72h, respectively compared to the untreated control.

Conclusion: hUC-MSCs induced inhibition of K562 cells in vitro. Inhibitory effect on cell proliferation indicates that hUC-MSCs have anti-cancer effects. Additional studies using cell free extracts of hUC-MSCs to evaluate AML inhibition will help to elucidate the underlying anticancer mechanism.

Biography
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