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Dialysis

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Application of a Physical Antimicrobial Spray Dressing for Reducing Postoperative Peritoneal Dialysis Catheter Exit Site Infection in End-stage Renal Failure Patients: A Prospective Cohort Study

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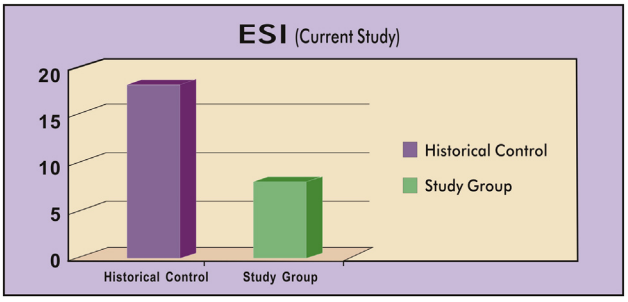
Objective: Exit site infection (ESI) in the early stage of peritoneal dialysis (PD) catheter insertion period which will disrupt healthy sinus tract formation and may lead to peritonitis and catheter removal. None of the current strategies is considered a successful measure in preventing early ESI. The aim of this study was to evaluate the effectiveness of JUC® non-pharmacological, nano-technology physical antimicrobial spray dressing (JUC®) on postoperative PD exit site care with respect to reduction of infection risk and patient comfort.
Methods: A prospective study was conducted from December 2012 to July 2014 and the results compared with a historical cohort data obtained between 2010 to 2011 in the same Renal unit (see table). JUC® was administered to exit sites of consenting subjects. The incidence rate of ESI, the causative microorganisms and demographic data were compared between the study and the retrospective control group. Pain was assessed by a numeric pain score. An exit site swab culture was performed two weeks after PD catheter insertion to determine presence of colonizing organisms. The patients were observed for ESI for 4 weeks.
Results: Of the 95 subjects completing the study, eight developed PD catheter ESI. ESI incidence (within 4 weeks after PD catheter insertion) in the study group was only 8.4% compared with 19% in the control group, representing a significant decrease in ESI by 56% (P = 0.038, Chi-squared test). The causative microorganisms for ESI in the study group were coagulase-negative *Staphylococci* (12.5%), *Corynebacterium* species (12.5%), *Staphylococcus aureus* (62.5%) and *Pseudomonas aeruginosa* (12.5%). For the numeric pain score (0 to 10), 93% of the subjects reported from 0 to 2. There were no adverse effects, skin discomfort nor delayed sinus epithelialization for wound healing reported.
Conclusion: JUC® physical non-pharmacological nano-technology antimicrobial spray dressing is effective and safe in preventing early postoperative PD catheter ESI.



		Historical Control (N = 209)	Study Group (N = 95)	P value
Sex	Male	122 (58.37%)	50 (52.63%)	0.66
	Female	87 (41.63%)	45 (47.36%)	
Age	Median	60	59	0.37545
Diabetes history	Yes	119 (56.94%)	50 (52.63%)	0.5656
	No	90 (43.06%)	45 (47.37%)	
Immuno-suppressant drug	Yes	8 (3.83%)	3 (3.16%)	0.5
	No	201 (96.17%)	92 (96.84%)	

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Effects of Astragaloside IV Against TGF-β-induced Epithelial-to-Mesenchymal Transition in Peritoneal Mesothelial Cells by Promoting Smad 7 Expression

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Objective: To investigate the effect of astragaloside IV (AS-IV) on the regulation of the TGF-β1 Smad signaling pathway in mesothelial cells with an epithelial-to-mesenchymal transition (EMT).
Methods: EMT of human peritoneal mesothelial cells (HMrSV5) was induced using 2 ng/ml TGF-β1. Cells were randomly divided into a vehicle group, a vehicle group with AS-IV, a TGF-β1 treated group, and a TGF-β1 treated group receiving varied doses of AS-IV or NAC. Real-time quantitative PCR and western blot were used to detect the expression of genes and proteins associated with the TGF-β1/Smad signaling pathway and EMT. DCFH-DA was used to detect the generation of ROS in HMrSV5 cells, and a transwell migration assay was used to verify the capacity of AS-IV to inhibit EMT in HMrSV5 cells. Lentiviruses were used as carriers for the overexpression or knockdown of the Smad7 gene.
Results: Expression levels of E-cadherin (epithelial marker) were decreased and vimentin, α-SMA (EMT markers) and collagen I (extracellular matrix protein) phospho-Smad2/3, Snail1 and Snail2 was increased significantly in the TGF-β1-treated HMrSV5 cells. AS-IV was associated with downregulated expression of vimentin and phospho-Smad2/3 in a dose-dependent manner, while the expression of Smad7 increased. Silenced or forced expression of Smad7 verified its role in the inhibitory effect of AS-IV on TGF-β1-induced EMT in HMrSV5 cells.
Conclusion: AS-IV effectively promotes the upregulation of Smad7 in the TGF-β1/Smad signaling pathway during the EMT of HMrSV5 cells, indicating its potential therapeutic effect for the control of PF.