

Use Of Cytosorb Cartridges In A Case Of Systemic Leptospirosis

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Introduction

Leptospirosis presents with a wide clinical spectrum, ranging from subclinical infection to severe multi-organ involvement. Pathogenesis involves *Leptospira* binding to host TLR2 receptors through LipL32, activating proinflammatory pathways (e.g., TNF- α , NF- κ B), promoting immune evasion and leading to acute tubular necrosis and hepatic injury. In March 2025, a 51-year-old male farmer was hospitalized after a one-week history of fever with chills, jaundice, and anuria. Laboratory tests revealed severe hepatic and renal dysfunction, along with elevated inflammatory markers (Table 1). Given his occupational exposure and clinical presentation, Weil's syndrome—a severe manifestation of leptospirosis—was suspected and later confirmed via serological testing.

Methods

Empirical broad-spectrum antibiotic therapy (ceftriaxone, meropenem, linezolid) was initiated and subsequently de-escalated to a cephalosporin upon confirmation of the diagnosis.

Renal ultrasound showed findings consistent with acute tubular necrosis, including hypoechoic pyramids and a hyperechoic cortex, with normal kidney size.

The patient was admitted to the ICU and initiated on Continuous Kidney Replacement Therapy (CKRT), specifically Continuous Veno-Venous Hemodiafiltration (CVVHDF). Two sessions using a polycarbonate filter combined with Cytosorb[®] adsorber cartridges were performed to manage severe hyperbilirubinemia, resulting in an initial bilirubin reduction. Due to persistent anuria and hypertension, a new CKRT circuit with an Oxiris[®] (AN69 ST) endotoxin-adsorbing filter was initiated, and later a new Cytosorb[®] cartridge was added.

Results

The treatment achieved initial bilirubin reduction, and the addition of a new Cytosorb[®] cartridge led to a marked clinical and biochemical improvement.

After four CKRT circuits and three adsorber cartridges, the patient showed progressive clinical recovery, including resolution of jaundice and a transition to the polyuric phase. Two additional intermittent dialysis sessions were required.

Conclusion

The patient was discharged with improving renal function (creatinine 3.03 mg/dL, urea 62 mg/dL) and scheduled for outpatient nephrology follow-up.

	30/3	31/3	1/4	2/4 h 7:00	2/4 h 16:00	3/4	16/4	28/4
Blood purification modality/ membrane	CVVHDF/ PCTE +Cytosorb [®]	CVVHDF/ PCTE +Cytosorb [®]	CVVHDF/ PCTE	CVVHDF/ AN69 ST	CVVHDF/ AN69 ST +Cytosorb [®]	CVVHDF/ AN69 ST	-	-
WBC (x10E9/L)	14.40	12.62	14.61	19.32	21.84	28.12	5.97	6.79
Hb (g/dl)	10.4	9.5	9.9	10.0	9.6	9.3	7.8	10.1
PLT (x10E9/L)	48	39	41	65	80	89	260	338
Creatinine (mg/dl)	5.4	4.48	2.66	2.24	2.04	2.21	5.79	3.03
Urea (mg/dl)	icteric sample	icteric sample	icteric sample	icteric sample	icteric sample	icteric sample	190	62
Uric Acid (mg/dl)	6.30	5.65	3.44	-	-	2.98	10.5	7.83
CRP (mg/L)	156.0	113.0	59.0	27.9	22.1	16.8	8.4	16.9
PCT (mcg/L)	49.15	30.53	21.37	12.88	-	6.3	0.66	-
Total bilirubin (mg/dl)	32.93	28.62	26.13	33.73	39.78	35.26	4.96	4.31
Direct bilirubin (mg/dl)	25.7	24.1	22.8	25.1	30	24.0	2.4	1.7
AST (U/L)	200	139	116	111	-	76	-	-
ALT (U/L)	107	85	84	94	-	82	55	-

Table 1. From 30/3 initiation of CKRT with two Cytosorb cartridges. On 2/4, an attempt to pause Cytosorb was restart after blood tests, followed by improvement of hyperbilirubinemia. On 16/4, the patient was weaned off dialysis support during the polyuric phase.