Himachal Journal of Agricultural Research
Volume 52, Issue 1, June 2026; pp. 214–219; DOI: 10.18311/HJAR/2026/27064

INFORMATICS JOURNALS
Veterinary Sciences

Emergency Vascular Access using Mini-cutdown, Intraosseous and Central Venous Catheterisation in Critically Ill Dogs: Clinical and Survival Outcomes

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Abstract

Establishing vascular access in critically ill dogs is challenging when peripheral catheterisation is not feasible due to severe dehydration or circulatory compromise. This study evaluated mini-cutdown, intraosseous access and central venous catheterisation in ten critically ill dogs and assessed associated clinical, haematological, biochemical and survival outcomes. At admission, dogs exhibited significant tachycardia and prolonged capillary refill time, indicating poor perfusion. Haematological findings included leukocytosis with neutrophilia and monocytosis, while erythrocytic indices were reduced. Blood urea nitrogen and creatinine concentrations were markedly elevated at presentation and decreased following therapy, suggesting reversible renal hypoperfusion. C-reactive protein and lactate dehydrogenase levels were significantly increased, reflecting systemic inflammation and tissue injury. Electrolytes showed minimal variation. The overall survival rate was 30%, with most deaths occurring within 48 hours. These findings highlight the clinical utility of alternative vascular access techniques in hemodynamically unstable dogs, though prognosis remains dependent on the severity of systemic dysfunction.

Keywords: Central venous catheterisation, C-reactive protein, Critical care, Intraosseous access, Mini-cutdown

Establishment of rapid and reliable vascular access is a critical component in the management of emergency and critically ill canine patients. Intravenous access enables prompt administration of fluids, drugs, and blood products and facilitates monitoring of hemodynamic status. However, in severely compromised patients, particularly those presenting with hypovolemia, shock, dehydration, sepsis or cardiovascular instability, peripheral venous access may be difficult or impossible due to circulatory collapse or venous constriction.

In such circumstances, alternative vascular access techniques become essential for timely therapeutic intervention. Venous cutdown (mini-cutdown) provides direct visualisation and cannulation of peripheral veins when routine catheterisation fails. Intraosseous (IO) access offers a rapid and effective route for fluid and drug administration through the medullary cavity, especially in emergency settings. Central venous catheterisation (CVC), on the other hand, allows reliable long-term vascular access, central venous pressure monitoring and administration of hyperosmolar solutions[1].

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Manuscript Received: 04.04.2026; Accepted: 25.05.2026

Although these techniques are well described in veterinary critical care practice, comparative clinical evaluation of their use in dogs with failed peripheral venous access remains limited under field conditions. Assessment of associated clinico-pathological alterations, inflammatory markers and survival outcomes following the use of these alternative techniques is important to determine their feasibility, safety and prognostic relevance.

Therefore, the present study was undertaken to clinically evaluate the use of mini-cutdown, intraosseous and central venous catheterisation techniques in critically ill dogs where peripheral venous access was not feasible, and to assess the associated haematological, biochemical, electrolyte and survival outcomes. The study was not intended to compare the efficacy of individual vascular access techniques, but rather to evaluate their clinical utility as alternative emergency vascular access methods in critically ill dogs.

MATERIALS AND METHODS

The present clinical study was conducted on ten critically ill dogs presented for emergency management in which peripheral venous catheterisation was not achievable after routine attempts due to severe dehydration, circulatory compromise or collapsed veins. The enrolled dogs were suffering from various critical illnesses, including severe dehydration, septic conditions, gastrointestinal disease, renal compromise and circulatory shock requiring intensive



Figure 2. **(A)** Dog after catheter placement; **(B)** Catheter was secured to the skin using stay sutures; **(C)** Administration of therapy via intraosseous route; **(D)** Small incision to facilitate entry of catheter.

support therapy. A detailed clinical examination was performed at admission, including assessment of general demeanour, mucous membrane colour, capillary refill time (CRT), heart rate, respiratory rate, rectal temperature and systolic blood pressure. Clinical parameters were recorded at presentation (Day-0) and monitored during the treatment period (Day-5). Age, sex and survival outcome during hospitalisation were documented.

Based on clinical condition and urgency for vascular access, advanced techniques including mini-cutdown (venous cutdown), intraosseous access and central venous catheterisation were performed under strict aseptic precautions. Mini-cutdown was carried out by surgically exposing a peripheral vein (cephalic or saphenous) for direct catheter placement (Figure 1D). Intraosseous access was established in the proximal femur by inserting a sterile needle into the medullary cavity for fluid and drug administration (Figure 1C). Central venous catheterisation was performed via the jugular vein using a triple-lumen catheter (7 Fr, 16 cm) employing the Seldinger technique (Figure 1A) and (Figure 1B). Blood samples were collected for complete blood count, serum biochemistry and electrolyte estimation. Inflammatory markers, including C-reactive protein (CRP) and lactate dehydrogenase (LDH), were also assessed at admission and during monitoring. Data were analysed using GraphPad InStat statistical software, results were expressed as mean ± standard error (SE), and Student's t-test was used for comparison with $p < 0.05$ considered statistically significant.

RESULTS AND DISCUSSION

A total of ten critically ill dogs requiring advanced vascular access were evaluated. The cases included dogs suffering from severe dehydration, gastrointestinal disorders, septic conditions, renal dysfunction and circulatory compromise. Heart rate was significantly elevated on Day-0 (135.3 ± 4.78 bpm) compared to healthy controls (75.6 ± 2.13 bpm; $p<0.05$) and showed partial reduction by Day-5 (129 ± 11.15 bpm), indicating initial circulatory compromise with subsequent stabilisation. Capillary refill time was significantly prolonged at presentation (2.8 ± 0.38 sec *vs* 1.3 ± 0.15 sec in controls; $p<0.05$), and it improved following therapy, reflecting restoration of peripheral perfusion as shown in Table 1. Haematological analysis revealed marked leukocytosis (25.85 ± 7.6 $\times 10^3/\mu L$ *vs* 12.27 ± 0.53 $\times 10^3/\mu L$), neutrophilia and elevated neutrophil-to-lymphocyte ratio (16.6 ± 5.2 *vs* 3.01 ± 0.17), consistent with inflammatory leukogram patterns reported in acute gastrointestinal and systemic inflammatory conditions[2]. Reduced erythrocytic indices and increased RDW suggest inflammatory stress and possible anaemia secondary to systemic

Table 1. Clinical observations in diseased dogs (Mean ± S.E.)Means bearing different superscripts (a,b) within a row differ significantly ($p < 0.05$)

Sr. No.	Parameter	Control Group (n = 10)	Diseased Dogs (n = 10) Day = 0	Diseased Dogs (n = 3) Day = 5
1	R.T. (°F)	100.54 ± 0.23[a]	99.33 ± 0.49[a]	99.53± 0.66[a]
2	H.R. (bpm)	75.6 ± 2.13[a]	135.3 ± 4.78[b]	129± 11.15[ab]
3	R.R. (per min)	27.4 ± 2.1[a]	28.5 ± 1.31[a]	24 ± 2.08[a]
4	CRT (Secs.)	1.3 ± 0.15[a]	2.8 ± 0.38[b]	1.33 ± 0.33[ab]
		Control group (n=10)	Group-4 (n=5)Day=0	
5	Systolic Blood Pressure(mm of Hg)	129.12 ± 11.4	92 ± 8.60 (70-120)	

Means bearing different superscripts (a,b) within a row differ significantly ($p < 0.05$)

Table 2. Alteration in haematological parameters in diseased dogs (Mean ± S.E.)

Sr. No.	Parameters	Healthy (n = 10)	Diseased Dogs (n = 10) Day = 0	Diseased Dogs (n = 3) Day = 5
1	TLC ($\times 10^3$ /µL)	12.27 ± 0.53[a]	25.85 ± 7.6[a]	19.92 ± 2.91[a]
2	Neutrophils ($\times 10^3$ /µL)	8.73 ± 2.52a	21.14± 7.06[a]	15.11 ± 4.05[a]
3	Lymphocytes ($\times 10^3$ /µL)	2.51 ± 0.47[a]	2.615 ± 1.03[a]	3.69 ± 1.59[a]
4	Monocytes ($\times 10^3$ /µL)	0.17 ± 0.1[a]	1.877 ± 0.6[b]	1.3 ± 0.40[ab]
5	Eosinophils ($\times 10^3$ /µL)	0.3 ± 0.10[a]	0.176 ± 0.86[a]	0.029 ± 0.19[a]
6	Neutrophils (%)	73.5 ± 2.12[a]	65.2 ± 12.6[a]	70.13 ± 11.88[a]
7	Lymphocytes (%)	9.67± 0.05[b]	11.24 ± 3.8[ab]	22.5 ± 6.06[a]
8	Monocytes (%)	8.43 ± 1.67[a]	7.6 ± 2.09[a]	6.63 ± 2.11[a]
9	Eosinophils (%)	1.23 ± 0.7[a]	1.26 ± 0.69[a]	2.6 ± 1.12[a]
10	TEC ($\times 10^{12}$ /L)	7.89 ± 1.86[a]	5.49 ± 0.95[a]	5.69±0.58[a]
11	Hb (g/dL)	13.45 ± 1.29[a]	12.4 ± 2.08[a]	12.93±0.74[a]
12	PCV (%)	42.1 ± 2.25[a]	33.5 ± 5.3[a]	34.8±22.2[a]
13	MCV (fL)	59.22 ± 0.81[a]	61.8 ± 2.5[a]	62.56±4.03[a]
14	MCH (pg)	22.3 ± 0.41[a]	22.7 ± 1.21[a]	22.37±1.44[a]
15	MCHC (g/dL)	36.6 ± 0.9[a]	30.8 ± 5.62[a]	32.03±3.42[a]
16	RDW (%)	13.6 ± 0.86[a]	17.3 ± 1.115[b]	16.16±0.88[ab]
17	PLT ($\times 10^9$ /L)	238 ± 2.40[a]	204.6 ± 48.76[b]	218±33.50[a]
18	NLR	3.01 ± 0.17	16.6 ± 5.2[b]	6.18±2.42[ab]

Means bearing different superscripts (a,b) within a row differ significantly ($p < 0.05$)

disease (Table 2). Biochemical evaluation (Table 3) demonstrated significantly elevated BUN (144.1 ± 44.2 mg/dL) and creatinine (7.21 ± 2.74 mg/dL) at admission compared to controls (23.46 ± 1.42 and 0.68 ± 0.23 mg/dL; $p<0.05$), which markedly declined by Day-5, suggesting reversible renal hypoperfusion or prerenal azotemia. ALT and ALP elevations likely reflected metabolic and inflammatory stress. Electrolyte concentrations remained within physiological limits, indicating compensated systemic status.

C-reactive protein levels were significantly elevated at admission (6.34 ± 0.44 mg/L *vs* 3.04 ± 0.90 mg/L; $p<0.05$), supporting activation of the acute-phase response, as CRP is a recognised marker of systemic inflammation and disease severity in critically ill dogs[3,4]. Similarly, lactate dehydrogenase concentrations (Table 4) were markedly increased (334.3 ± 21.18 U/L *vs* 121.82 ± 22.16 U/L; $p<0.05$), indicating tissue hypoxia and cellular injury, consistent with its documented role as a marker of systemic cellular damage[5]. The overall survival rate was 30%, with most fatalities occurring within 48 hours of admission, reflecting severe systemic compromise. The survival outcome observed in the study was

Table 3. Alterations in biochemical parameters in diseased dogs (Mean ± S.E.)

Sr. No.	Parameters	Healthy (n = 10)	Diseased Dogs (n = 10) Day = 0	Diseased dogs (n = 3) Day = 5
1	BUN (mg/dL)	23.46 ± 1.42[a]	144.1 ± 44.2[b]	30.25 ± 8.66[b]
2	Creatinine (mg/dL)	0.68 ± 0.23[a]	7.214 ± 2.74[b]	0.63 ± 0.09[b]
3	Total protein (g/dL)	6.85 ± 0.05[a]	6.84 ± 0.71[a]	6.22 ± 0.54[a]
4	Albumin (g/dL)	2.77 ± 0.06[b]	2.2 ± 0.169[a]	2.50 ± 0.23[ab]
5	Albumin - globulin ratio	1.3 ± 0.2[b]	0.543 ± 0.12[a]	0.67 ± 0.01[ab]
6	Total bilirubin (mg/dL)	0.13 ± 0.03[a]	0.7 ± 0.49[a]	0.11 ± 0.01[a]
7	AST (U/L)	26.48 ± 1.8[a]	260.8 ± 204.6[a]	37 ± 7.33[a]
8	Glucose (mg/dL)	105.7 ± 2.31[a]	95.82 ± 12.48[a]	93.76 ± 9.83[a]
9	ALT (U/L)	33.46 ± 1.19[a]	197.7 ± 74.4[b]	78.1 ± 1.09[ab]
10	ALP (U/L)	35.71 ± 0.52[a]	740.15 ± 436.9[a]	268.59 ± 96.98[a]

Means bearing different superscripts (a,b) within a row differ significantly ($p < 0.05$)

Table 4. Alteration in the value of CRP and LDH (Mean ± S.E.)

Parameter	Healthy (n = 10)	Day 0 (n = 10)
C-reactive Protein (mg/L)	3.04 ± 0.90	6.34 ± 0.44*
Lactate Dehydrogenase (U/L)	121.82 ± 22.16	470.4 ± 27.3*

Means bearing different superscripts (a,b) within a row differ significantly ($p < 0.05$)

likely influenced by the severity of the underlying disease condition, systemic inflammatory response and response to supportive treatment rather than the vascular access technique itself. The high inflammatory burden and SIRS positivity likely contributed to poor clinical outcomes, as previously reported in critically ill canine patients[4,6]. Collectively, these findings highlight that mini-cutdown, intraosseous access and central venous catheterisation are essential supportive interventions in hemodynamically unstable dogs by facilitating rapid vascular access and emergency therapeutic management. However, the present study did not compare the individual efficacy of these techniques. The prognosis remains largely dependent on the severity of underlying systemic dysfunction at presentation. A limitation of the present study was that serial hourly monitoring could not be performed, and evaluations were conducted primarily at Day-5 due to clinical and logistical constraints associated with emergency critical care cases.

CONCLUSION

The clinical utility of alternative vascular access techniques in hemodynamically unstable dogs, though prognosis remains dependent on the severity of systemic dysfunction.

ACKNOWLEDGEMENTS

The authors are thankful to the facilities provided by CSKHPKV Palampur to the Department of Veterinary Medicine, which helped in completing the above study.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

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AUTHORS CONTRIBUTION

IS: Conducted the clinical work, collected and analyzed the data, and prepared the original manuscript draft. AS: Conceived and supervised the research work, provided methodological guidance, and reviewed and edited the manuscript. AK: clinical case management and clinical support throughout the study. AKu: guidance about central venous catheterization and surgical procedures, and reviewed the manuscript. RK: guidance regarding intraosseous catheterization and reviewed the manuscript.