***Review Article***

**TRADITIONAL WISDOM AND MODERN NEEDS: ETHNOVETERINARY APPROACHES TO SNAKEBITE MANAGEMENT IN LIVESTOCK**

**ABSTRACT**

Snakebite envenomation remains a critical public and veterinary health concern, particularly in rural and agrarian communities of developing countries. Each year, millions of humans and animals fall victim to venomous snakebites, with livestock such as cattle, goats, sheep, and working animals like oxen and horses at considerable risk due to their constant exposure to snake prone environments like fields and forests. The effects of snakebite in animals can range from localized swelling and tissue necrosis to systemic complications such as respiratory failure or internal bleeding, often resulting in severe economic losses for rural farmers. Conventional antivenoms, while effective, are frequently inaccessible in remote areas due to high cost, limited availability, need for refrigeration, and potential for adverse reactions. In this context, **ethnoveterinary medicine** - the traditional knowledge and practices used to treat animal ailments has emerged as an alternative or complementary approach to snakebite management. Across India and other parts of Asia and Africa, various plant based remedies have been traditionally employed to treat snakebite in animals which are believed to possess anti-venom, anti-inflammatory, and wound healing properties. These treatments are typically administered orally, topically, or both, often combined with ritualistic or cultural practices. While scientific validation of many of these remedies is still ongoing, ethnoveterinary treatments are valued for their accessibility, affordability, and lower risk of side effects. Promoting research into these traditional practices can offer sustainable solutions to snakebite management in regions where conventional treatment is out of reach.

Key words: Snakebite**,** antivenoms**, ethnoveterinary,** traditional knowledge, **animal ailments.**

**INTRODUCTION**

Snakebite envenomation affects approximately 5.4 million people worldwide each year, resulting in 1.8 to 2.7 million envenomation cases and an estimated 81,410 to 137,880 deaths. In addition to fatalities, snakebites often lead to significant morbidity, including amputations and lifelong disabilities. The burden is especially severe in developing countries, with India accounting for the highest number of snakebite cases globally - about one million bites annually, resulting in approximately 600,000 envenomations and 58,000 deaths (Suraweera et al., 2020). The primary treatment for snakebite is antiserum (antivenom), which is conventionally produced by injecting a non lethal dose of venom into mammals such as horses or rabbits to stimulate the production of immunoglobulins. These antibodies are then harvested from the animal's blood (Makhija and Khamar, 2010). However, antivenom therapy faces several critical limitations. It is most effective only when administered promptly, ideally before venom toxins exert systemic effects. In many rural regions, transporting victims to distant urban hospitals where antivenom is available often proves ineffective due to time delays. Furthermore, antivenoms are expensive and thus inaccessible to many impoverished victims. They may also cause adverse reactions such as anaphylactic shock, pyrogenic responses, and serum sickness (Morais, 2018). Moreover, conventional antivenoms often offer limited protection against specific venom induced toxicities like necrosis, hemorrhage, cytotoxicity, and nephrotoxicity (Sani et al., 2019). In India, snakebites are also a significant and frequently overlooked threat to animals, especially in rural and semi urban regions where interactions between humans, animals, and snakes are common. Livestock such as cattle, buffaloes, goats, sheep, and working animals like oxen and horses, along with domestic pets like dogs and cats, are at considerable risk. This risk intensifies during the monsoon and post monsoon seasons, when snake activity increases due to higher humidity and prey availability. Grazing livestock or animals resting in snake inhabited areas are particularly vulnerable, and bites often go unnoticed until symptoms manifest - delaying treatment and increasing the risk of fatal outcomes. Globally, among the approximately 3,848 snake species identified, around 750 are venomous (Uetz et al., 2020). In India, four species - **Bungarus caeruleus** (common krait), **Naja naja** (spectacled cobra), **Echis carinatus** (saw scaled viper), and **Daboia russelii** (Russell’s viper) are primarily responsible for the majority of bites and related morbidity, despite the presence of over 60 venomous species in the country (Sulabh and Shivahre, 2018; Senji Laxme et al., 2019).

The **Common Krait** (Bungarus caeruleus), a highly venomous snake belonging to the family Elapidae, is widely distributed across rural and agricultural regions of the Indian subcontinent, including India, Pakistan, Nepal, and Sri Lanka (Warrell, 2010). Its venom is predominantly neurotoxic, leading to respiratory paralysis. The presence of presynaptic neurotoxins further intensifies its lethality by disrupting neurotransmitter release at the nerve terminals (Damm, 2023). Similarly, the **Indian Cobra** (Naja naja), also a member of the Elapidae family, is commonly found across South Asia. Its venom comprises a potent mix of neurotoxins, cytotoxins, and cardiotoxins, posing serious health risks (Whitaker et al., 2004). Neurotoxins induce paralysis by impairing nerve signal transmission, cytotoxins cause local tissue necrosis, and cardiotoxins affect heart muscles and the circulatory system, making envenomation by Naja naja particularly dangerous (Rathnayaka et al., 2017). The **Russell’s Viper** (Daboia russelii), a member of the Viperidae family, inhabits diverse environments such as grasslands and woodlands throughout South Asia, extending into Southeast Asia and southern China (Whitaker, 2004). Its venom contains a complex mix of hemotoxins and cytotoxins, leading to severe local tissue damage and systemic effects, including coagulopathy and spontaneous bleeding due to disrupted blood clotting mechanisms (Ismail et al., 2023). The **Saw scaled Viper** (Echis carinatus), also of the Viperidae family, is highly adaptable and thrives in a range of habitats, including deserts, grasslands, and rocky terrains across the Indian subcontinent, the Middle East, and parts of Africa (Warrell, 2010). Its venom contains potent hemotoxic and cytotoxic components that interfere with blood coagulation and cause substantial local tissue injury (Ismail et al., 2023). Despite the availability of antivenoms, their clinical utility is limited by several factors. These include the requirement for stringent cold chain storage, high cost, limited availability in rural regions, and narrow venom specificity, which may render them ineffective against bites from certain snake species. Moreover, conventional antivenoms often induce adverse effects such as anaphylaxis, pyrogenic reactions, and serum sickness. In response to these limitations, there has been increasing interest in **natural products and plant based remedies** with antivenom potential. Researchers are investigating the anti-myotoxic, anti-hemorrhagic, and anti-inflammatory properties of various plant extracts as complementary or alternative therapies. Ethnomedicinal plants have a long history of use in traditional snakebite treatments and show promise in neutralizing venom toxins. These natural remedies could serve as accessible, affordable, and well tolerated adjuncts or alternatives to serum based antivenoms, particularly in underserved and remote regions (Lima et al., 2022).

**List 1 : Traditionally used plants for Snakebite Management**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Plant species**  | **Family**  | **Common name**  | **Part used**  | **Direction of uses** | **Administration**  | **References** |
| Abrus precatorius | Leguminosae  | Kundumani  | Roots  | Unknown  | Orally for 5 days | Makhija, Khamar, 2010 |
| Abutilon indicum  | Malvaceae | Atibala | Leaf | Leaf juice Mixed with jiggery Fruits | Oral (2days) | (Uawonggul *et**al*., 2006) |
| AcaciaLeucophloea | Mimosaceae | White bark | Bark | Paste | External (1 Week) | (Samy *et al*., 2008) |
| AchilleaMillefolium | Asteraceae | Mountain yarrow | Whole plant | Paste | Oral (6 days) | (Makhija,Khamar, 2010) |
| AchyranthesAspera | Amaranthaceae | Nayuruvi | Leaf | Paste | External (3Weeks) | (Butt *et al*.,2015) |
| Acorus calamus | Araceae | Vasambo | Rhizome | Paste | External (7 days) | (Verma, Singh, 2008) |
| Aeglemarmelos | Rutaceae | Vilvam | Root bark | AqueousDecoction | Oral (2 Weeks) | (Panghal *et al*., 2010) |
| Aerva lanata | Amaranthaceae | Poolapo | Rhizome | Unknown | Oral (11 days) | (Selvanayagam*et al.*, 1995) |
| Alangium salvifolium  | Alangiaceae | Alangi | Root bark | Decoction | Oral (twice a dayup to 4 days) | (Alagesaboopathi,2013) |
| Allium cepa | Liliaceae | Venkayam | Skin bulb | Paste | Externalapplication (5 days) | (Butt *et al*., 2015) |
| AmaranthusDubius | Amaranthaceae | Gusanito | Leaves, rootand seed | Ointment | Applied externally | (Félix-Silva *et al*., 2017) |
| Andrographispaniculata | Acanthaceae | Periyananghai | Whole plant | Decoction, Paste | External(5–14 days) | (Uawonggul *et**al*., 2006) |
| Argemonemexicana | Papaveraceae | Barahmathandu | Leaf, Seed | Decoction | Oral (7 days) | (Samy *et al.*, 2008) |
| AristolochiaIndica | Aristolochiaceae | Birthwort | Whole plant | Root extract | Paste External(1 Week) | (Meenatchisundaram,Parameswari,Michael, 2009) |
| AzadirachtaIndica | Meliaceae | Neem, Wimpu | Flower | Decoction | Oral (7 days) | (Dey, De, 2012) |
| Calotropisgigantea | Asclipiadaceae | Madar, Crownflower,Milkweed | Latex, Leaf,Root | Fresh latexPaste with ghee | Oral (3–7 days)and externally | (Félix-Silva *et al*., 2017) |
| Cassia alata | Caesalpiniaceae | Senna | Leaf | Paste | Oral (21 days) | (Rahmatullah *et**al*., 2009) |
| Cassia tora  | Caesalpiniaceae | Tagarai  | Leaf | Decoction  | Topical(14 days) |  (Samy *et al.,* 2008) |
| Citrus limon | Rutaceae | Elumichai | Ripe skin | Paste | External (3 days) | Gomes *et al.*, 2010) |
| Curcuma longa | Zingiberaceae | Haldi | Whole plant | Paste | Paste is takenorally and applied | (Kumar *et al*, 2016) |
| DalbergiaMelanoxylon | Fabaceae | Veelipruthi | Stem bark | Decoction | Oral (6 days) | (Kala, 2009) |
| DracontiumSpruceanum | Araceae | Chupa, Chupadera | Roots,Leaves | Direct heatingdecoction | Externally | (Félix-Silva *et al*., 2017) |
| DracontiumSpruceanum | Araceae | candelillachupadera | Rhizome,Stem, Leave | Extract, Poultice | Internally,externally | (Giovannini,Howes, 2017) |
| Ehretiabuxifolia | Ehretiaceae | Thelchedi | Root | Paste | External (7 days) | (Samy *et al.*, 2008) |
| FeronicaLimonia | Rutaceae | Elephant apple | Root | Juice | Oral (3 days) | (Makhija,Khamar, 2010) |
| GloriosaSuperb | Liliaceae | Kalappaih kilangu | Tuber | Paste | External (2–5 days) | Minu *et al.,* 2012 |
| Gymnemasylvestre | Asclepiadaceae | Gurmarbuti | Root | Tincture | Oral (4 days) | (Sajon, Sana, Rana, 2017) |
| HemidesmusIndicus | Asclepiadaceae | Anantamul | Root | Decoction | Oral (7 days) | (Dey, De, 2012) |
| Madhucalongifoila | Sapotaceae | Saathikkai | Nut | Paste | External (2–3 days) | (Minu *et al.,* 2012) |
| Mimosa pudica | Leguminosae | Touch-me-not,Chui mui | Creeper Root | Paste | Paste is mixed withraw rice water andgiven orally | (Kumar *et al*, 2016) |
| Momordicacharantia | Curcubitaceae | Karela | Leaf, Stem,Fruit | Extract | Oral | (Giovannini,Howes, 2017) |
| MoringaOleifera | Moringaceae | Murunghai | Bark, Root | Tincture | External (3 days) | (Minu *et al.*, 2012) |
| Morus alba | Moreaceae | Mulberry llai | Leaf | Juice | Oral (3 Weeks) | (Dey, De, 2012) |
| OcimumSanctum | Lamiaceae | Tulasi | Leaf | Juice | Oral (8 days) | (Panghal *et al.,* 2010) |
| Ophiorrhizamungos | Rubiaceae | Napali | Root | Juice | Oral (Twice aday for 6 days) | (Krishnan *et al.,*2014)) |
| Rauvolfiaserpentine | Apocynaceae | Sarpgandha | Root | Unknown | External (10 days) | (Makhija, Khamar,2010)) |
| Sansevieriatrifasciata | Asparagaceae | Lirio de tigre | Ariel parts | Decoction | External | (Félix-Silva *et al*., 2017) |
| SapindusEmargiatus | Sapindaceae | Puvam kottai | Bark | Paste | Bark Paste | (Minu *et al.*, 2012) |
| Strychnosnux-vomica | Loganiaceae | Visakkotai | Bark | Paste | External (12 days) | (Makhija,Khamar, 2010) |
| SyzygiumCumini | Myrtaceae | Naeralae | Stem bark | Decoction | Oral (14 days) | (Makhija, Khamar,2010)) |
| *Tapirira**guianensi*s | Anacardiaceae | Fresmo | Oil | Ointment | Applied externally | (Félix-Silva *et al*., 2017) |
| TerminaliaArjuna | Combretaceae | Marutham | Bark | Paste | External (5 days) | (Minu *et al.,* 2012) |
| TrichodemaZeylanicum | Boraginaceae | Camel bush | Root | Aqueous extract | Oral and External(3 days) | (Asad *et al.,* 2011) |
| Wedeliacalendulae | Asteraceae | Karisilangkanni | Leaf | Juice | Internally (14 days) | (Girish,Kemparaju, 2011) |

**CLINICAL EFFECTS AND SYMPTOMS IN ANIMALS**

Snakebite envenomation in animals presents a complex spectrum of clinical signs and systemic effects, largely determined by the type of venom injected, the snake species involved, the amount of venom delivered, and the size and species of the animal bitten. Understanding the distinct toxic actions of various venoms is crucial for accurate diagnosis and effective treatment. Snake venoms are generally classified into neurotoxic, hemotoxic, cytotoxic, and myotoxic types, though many snakes produce a mixture of these toxins, resulting in overlapping clinical presentations. **Neurotoxic venom**, typical of snakes like cobras, kraits, mambas, and some coral snakes, primarily affects the nervous system by interfering with nerve impulse transmission. This venom acts rapidly to induce paralysis by blocking neuromuscular junctions, leading to muscle weakness and respiratory failure. Animals bitten by neurotoxic snakes often show early signs such as muscle twitching or generalized weakness, which quickly progress to more specific symptoms including drooping eyelids (ptosis), difficulty swallowing (dysphagia), facial muscle paralysis, and loss of coordination (ataxia). Respiratory muscles can become paralyzed, causing breathing difficulties and respiratory failure, a life threatening emergency. Interestingly, neurotoxic envenomation may produce only minimal local signs such as swelling or pain at the bite site, making it harder to detect initially without systemic neurological symptoms. In contrast, **hemotoxic venom**, commonly found in vipers, rattlesnakes, and some pit vipers, predominantly targets the circulatory system. This venom damages blood vessel walls and disrupts the coagulation cascade, leading to increased vascular permeability and spontaneous bleeding. Locally, bites from hemotoxic snakes typically cause painful swelling, bruising, and bleeding at the bite site. Systemically, envenomed animals may exhibit widespread hemorrhages manifesting as bleeding from the gums, nostrils, or injection sites, and blood can be present in the urine (hematuria) or feces (melena). Prolonged clotting times and coagulopathy can cause internal bleeding into vital organs such as the lungs, brain, or gastrointestinal tract. Hemolysis induced by venom further worsens the animal’s condition by causing anemia, lethargy, and weakness. Without prompt treatment, hemotoxic bites can rapidly progress to circulatory collapse and death due to hemorrhagic shock. **Cytotoxic venom** often associated with many vipers and rattlesnake species, primarily causes local tissue damage and necrosis. Upon injection, cytotoxins destroy cell membranes, triggering intense inflammation, swelling, and pain localized around the bite site. As the venom’s destructive effects escalate, affected tissues may develop blistering and extensive necrosis, leading to skin sloughing and ulceration. Such tissue damage can result in secondary bacterial infections and slow wound healing. In severe cases, extensive necrosis requires surgical intervention such as debridement or amputation to prevent systemic spread. Cytotoxic venom usually causes less systemic toxicity compared to neurotoxic or hemotoxic venoms, but local effects can be devastating and lead to permanent disability or loss of the affected limb. **Myotoxic venom**, produced by some sea snakes and certain cobras, specifically targets muscle tissues. It causes direct muscle cell destruction (rhabdomyolysis), resulting in muscle pain, stiffness, swelling, and weakness. As muscle fibers break down, myoglobin is released into the bloodstream and filtered through the kidneys, potentially leading to myoglobinuria, which presents as dark or reddish urine. This condition poses a significant risk of acute kidney injury or renal failure if not promptly managed. Myotoxic venom may also contribute to systemic weakness and paralysis, overlapping with neurotoxic symptoms. Animals affected by myotoxic venom require aggressive supportive care to prevent renal complications. In reality, many snake species inject venom that contains a combination of these toxic effects, making clinical presentations variable and sometimes confusing. For example, many vipers have both hemotoxic and cytotoxic properties, leading to a mixture of bleeding tendencies and severe tissue necrosis. Similarly, some elapid snakes like cobras produce neurotoxic and myotoxic venom simultaneously, causing both paralysis and muscle damage. This complexity necessitates careful clinical evaluation and sometimes laboratory testing to determine the predominant venom effects and guide treatment choices. The **local clinical signs** of snakebite typically begin at the bite site and include swelling, redness, heat, pain, bruising, and sometimes blister formation. In cases involving cytotoxic venom, tissue necrosis and ulceration may develop over hours to days. Bite sites may reveal puncture wounds or fang marks, though these can be difficult to detect, especially in animals with thick fur or dark skin. The degree of local swelling can vary from mild to severe and may spread proximally along limbs or other body parts. **Systemic symptoms** usually manifest within hours and can be life threatening. Animals often exhibit lethargy, weakness, and anorexia. Vomiting, excessive salivation, and difficulty swallowing are common, especially with neurotoxic envenomation. Respiratory distress is a critical sign indicating paralysis of respiratory muscles and requires immediate veterinary attention. In hemotoxic cases, animals may show signs of shock such as pale or bluish mucous membranes, rapid heart rate, and weak pulses due to blood loss and vascular leakage. Urinary changes such as dark colored or decreased urine output can indicate myoglobinuria and kidney damage from myotoxic venom. Laboratory tests, when available, can assist in confirming snakebite envenomation and determining venom effects. These may reveal anemia, thrombocytopenia (low platelet count), prolonged clotting times, and elevated muscle enzymes (e.g., creatine kinase) indicative of muscle damage. If untreated, snakebite envenomation often leads to **progressive deterioration**, including circulatory collapse, multi organ failure, and death. The prognosis depends on the venom type, dose, bite location, and promptness of treatment. Early recognition of clinical signs and rapid initiation of appropriate therapy including administration of specific antivenom, supportive care with fluids, pain management, respiratory support, and wound care are essential for survival and recovery. In summary, snakebite in animals can produce diverse and severe clinical effects depending on the venom type. Neurotoxic venoms cause paralysis and respiratory failure, hemotoxic venoms disrupt blood clotting and cause hemorrhage, cytotoxic venoms lead to severe local tissue damage and necrosis, and myotoxic venoms result in muscle breakdown and kidney injury. Many snakes inject venom with mixed properties, further complicating the clinical picture. Effective management requires understanding these venom specific effects to guide timely diagnosis and targeted treatment, ultimately improving the chances of survival and reducing long term complications in envenomed animals.

### ****ECONOMIC AND EMOTIONAL IMPACT OF SNAKEBITE IN ANIMALS****

Snakebite in animals is a serious and often underreported issue that affects both rural communities and urban pet owners around the world. The consequences of snakebite go far beyond the immediate physical effects on the animal; they ripple into economic loss and emotional distress for those who depend on or care for these animals. Whether it involves a beloved pet or a valuable livestock animal, a snakebite incident can be devastating. This essay explores the multifaceted economic and emotional impacts of snakebite in animals, highlighting the challenges faced by farmers, pet owners, and animal caregivers alike.

#### ****Economic Impact****

The economic burden of snakebite in animals is significant and multifactorial. One of the most direct forms of loss is the **death of the animal**. In the case of livestock, such as cattle, goats, sheep, or poultry, the animal often represents a considerable investment in terms of purchase cost, feeding, healthcare, and time. Losing a productive animal translates into not only the loss of that investment but also the loss of future income that the animal would have generated. For example, a dairy cow may provide milk daily and produce calves over its lifetime. Its sudden death due to snakebite can create an immediate income vacuum for a household or farm. Beyond the death of animals, **veterinary treatment costs** also place a heavy economic strain on owners. Treating snakebites often involves emergency care, including antivenom, which is both expensive and sometimes difficult to source in rural areas. In many countries, a single vial of antivenom can cost hundreds of dollars, and multiple vials may be required depending on the species and severity of envenomation. Additional costs include intravenous fluids, antibiotics to prevent secondary infections, wound care, pain relief, and hospitalization. Even when the animal survives, the cost of medical treatment can be overwhelming, especially for small scale farmers or low income families. In working animals such as oxen, horses, or donkeys, which are commonly used for plowing fields or transporting goods, a snakebite injury can render the animal **temporarily or permanently unfit for work**. This affects the productivity of farms and reduces income generating capacity. For instance, in subsistence farming communities, the loss of a working bull can significantly delay planting or harvesting, affecting food security. Similarly, in equestrian businesses or racing industries, even a non lethal snakebite that results in muscle damage or scarring can drastically reduce an animal’s value and utility. There is also the issue of **productivity loss** in animals that survive the bite. A lactating cow or goat may show a significant drop in milk production due to stress, systemic illness, or organ damage caused by the venom. Breeding animals may suffer reduced fertility or complications that make them unsuitable for reproduction. Meat producing animals may gain weight more slowly or require extended recovery time before they can be slaughtered or sold, delaying returns on investment. Furthermore, farmers and animal keepers may incur **opportunity costs**. When a valuable animal is bitten, the time and resources needed to care for it mean other duties are often neglected. In larger farms or herds, other animals may also be at risk, prompting the need for immediate changes in animal management or infrastructure (e.g., snake proof fencing, habitat control), all of which carry additional expenses. In some cases, repeated snakebite incidents on a farm or in a breeding facility can damage the **reputation of the operation**, leading to reduced sales or customer trust. Buyers may avoid purchasing animals from a location known to have snake problems, fearing poor biosecurity or increased risk to their own animals.

#### ****Emotional Impact****

While economic loss can often be quantified, the **emotional impact** of snakebite in animals is equally important and often deeper than anticipated. For **pet owners**, especially those who keep dogs, cats, or horses as companions, snakebite can be emotionally devastating. Pets are often considered family members, and their suffering or sudden death can lead to intense grief, trauma, and feelings of helplessness. Watching a pet die or suffer due to snakebite sometimes within hours can leave a lasting emotional scar. In rural settings, families often develop strong emotional bonds with their livestock. Animals are not only sources of income but also represent stability, tradition, and trust. The sudden loss of a familiar animal can be emotionally destabilizing, especially for children who may be attached to farm animals they helped raise. In addition, **working animals** such as oxen and horses often form strong bonds with their handlers, and their loss can be deeply felt. Another common emotional consequence is **guilt**. Owners may blame themselves for not preventing the bite, not recognizing symptoms earlier, or not being able to afford timely treatment. This guilt is especially pronounced in cases where the owner had to make the difficult decision of euthanasia due to financial constraints or medical futility. Many pet owners describe such situations as among the most painful choices they’ve ever faced. Beyond grief and guilt, there is often **chronic stress and anxiety** following a snakebite incident. Pet owners in snake prone areas may become hypervigilant, altering their daily routines and environments in an attempt to prevent recurrence. Farmers may struggle with ongoing worry over the safety of their herds, and in extreme cases, fear of snakes can become so intense that it interferes with daily life and agricultural activity. The **emotional toll can also extend to the broader family unit**. In households where children are involved in animal care or have formed attachments to pets or livestock, witnessing snakebite or its aftermath can be traumatic. Such experiences may lead to lasting fears, aversions, or even nightmares. In communities where animals are deeply embedded in cultural or spiritual life, the death of an animal may carry symbolic meanings that intensify the psychological impact. In animal focused businesses such as breeding farms, veterinary clinics, or sanctuaries, snakebites can lead to staff burnout, emotional fatigue, and moral distress. Veterinary professionals may experience psychological stress when treating animals with poor prognoses or when they are unable to save a life due to resource limitations.

### ****CHALLENGES IN MANAGEMENT OF SNAKEBITE****

Managing snakebite in animals is a complex and demanding task that poses several significant challenges, particularly in rural, resource limited, and snake endemic areas. One of the foremost difficulties lies in the **early recognition and diagnosis** of snakebite. Unlike humans, animals cannot communicate pain or discomfort clearly, and snakebites often occur when animals are grazing, exploring, or sleeping times when owners are not typically present. As a result, bites often go unwitnessed. In many cases, the clinical signs are either subtle at first or easily confused with other medical conditions such as sprains, infections, insect stings, or trauma. Common early signs like swelling, lethargy, mild lameness, or salivation may not raise immediate concern, leading to delays in veterinary intervention. Moreover, the **bite site may be hidden**, particularly in animals with thick fur or in hard to examine areas like the inner thighs, abdomen, or oral cavity. By the time the bite is discovered or suspected, the venom may already have caused significant systemic damage. Access to **qualified veterinary care** is another substantial challenge. In many regions where snakebites are common, especially in developing countries, veterinary services are sparse or located far from rural households. Even where clinics exist, they may be inadequately staffed, poorly equipped, or closed outside normal business hours posing a problem since much snakebite occur at night or in the early morning. Emergency care, which is often crucial in the hours following envenomation, may simply not be an option. This lack of access results in poor outcomes, higher fatality rates, and greater suffering for affected animals. The **availability of antivenom**, the most critical treatment for venomous bites, is also a major issue. Antivenom is often prohibitively expensive, especially for livestock owners who may need to treat large animals requiring multiple vials. It is also commonly out of stock, only available in limited quantities, or stored at central locations far from the patient. Additionally, antivenoms require refrigeration and proper handling, conditions not always met in rural veterinary clinics. In some regions, the right type of antivenom for the specific snake species is not available, rendering treatment less effective or even dangerous. Another complicating factor is the **difficulty in identifying the snake species** responsible for the bite. Proper treatment, especially the administration of species specific or regionally appropriate antivenom, often depends on knowing whether the venom is primarily neurotoxic, hemotoxic, cytotoxic, or myotoxic. However, in most bite cases, the snake is not seen, or it escapes before it can be captured or identified. Even when it is seen, accurate identification requires knowledge of local snake species, which many owners or even veterinary personnel may lack. Misidentification can lead to the administration of inappropriate antivenom, delayed treatment, or failure to anticipate complications. In areas with multiple venomous species and no access to venom detection kits, this issue becomes particularly problematic. In addition to these issues, the **clinical presentation of snakebite is highly variable**, making management even more difficult. Different snake species produce different types of venom with distinct effects. Some animals may show severe neurological signs, while others may develop coagulopathies, organ failure, or extensive tissue necrosis. The location of the bite, the amount of venom injected, and the size, species, and age of the animal all influence the severity of the symptoms. This variability makes it difficult to apply standardized treatment protocols. For instance, a bite to the face may cause rapid swelling and airway obstruction, while a bite to a limb may lead to slow onset tissue necrosis. This unpredictability often forces veterinarians to rely on supportive therapy and symptomatic management, which may not be sufficient without antivenom. Supportive care itself poses further challenges, as it often requires **intensive monitoring and advanced veterinary infrastructure**. In moderate to severe envenomations, animals may need intravenous fluid therapy to manage shock, oxygen supplementation for respiratory distress, blood transfusions for severe hemorrhage or anemia, and medications to manage pain, vomiting, seizures, or coagulopathies. Additionally, some cases require surgical debridement of necrotic tissue or even amputation. Many rural clinics lack the equipment, medications, or staff needed to deliver such complex care. This not only limits the ability to save the animal’s life but also contributes to prolonged suffering and poor quality of life even if the animal survives. Finally, one of the most critical and widespread challenges in the management of snakebite in animals is **financial limitation**. Treating snakebite can be very expensive, particularly when antivenom, advanced diagnostics, prolonged hospitalization, or follow up care is required. Many animal owners, especially subsistence farmers or low income families, simply cannot afford such treatments. As a result, they may delay seeking veterinary attention, opt for traditional or home remedies, or abandon treatment altogether. In some cases, euthanasia is considered not because the animal’s condition is untreatable, but because the cost of care exceeds what the owner can bear. This adds a tragic dimension to the management challenge, where animals may die not from the bite itself, but from lack of affordable care.

**CONCLUSIONS**

Although numerous studies have documented ethnoveterinary practices for the treatment of snakebites in humans, to the best of our knowledge, this is the first scholarly report that specifically addresses the use of herbal remedies for managing snakebite cases in animals. The use of plants in treating snakebite cases in animals represents a valuable and often underutilized aspect of ethnoveterinary medicine. In many rural and resource limited settings where access to conventional antivenom is restricted or unaffordable, traditional plant based remedies provide a practical and culturally accepted alternative. Numerous medicinal plants have demonstrated potential antivenom properties, including anti-inflammatory, anti-hemorrhagic, and wound healing effects, which may help alleviate the symptoms of envenomation and support recovery. While these remedies have been used for generations with reported success, scientific validation and standardization are essential to ensure their safety, efficacy, and dosage accuracy. Integrating traditional knowledge with modern veterinary science through rigorous pharmacological and toxicological studies could lead to the development of effective, accessible, and affordable snakebite therapies for animals. Promoting such interdisciplinary research and supporting local knowledge systems can enhance veterinary healthcare, particularly in areas most burdened by snakebite incidents.

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