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A review on canine transmissible venereal tumour (CTVT)

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Abstract

Dogs who socialize closely or strays and wild dogs that engage in unrestricted sexual behavior are more likely to have the infectious canine transmissible venereal tumour (CTVT).

CTVT is a distinct, naturally contagious tumour in which the altered tumour cell itself acts as the causal agent and persists in the host as a parasitic allograft. The clinical history, signs, symptoms and cytological markers are frequently sufficient for making a diagnosis, although biopsy and a histological investigation may be required in rare circumstances. With three weekly vincristine sulphate IV injections, the majority of patients are curable. It is challenging to control and necessitates continuing animal birth control in stray dogs due to the disease's connection to wild and stray dogs. The various advancements around this intriguing tumour, including its biology, diagnostic techniques, and treatment options, are covered in this review.

Keywords: Canine, transmissible venereal tumour, treatment, Vincristine, cancer

Introduction

Considering the fact that tumour can develop in a wide range of tissues and can be brought on by a variety of different factors, such as radiation, mutagenic chemicals, and viruses, damage to the cellular genome is a characteristic shared by almost all neoplasms (Donald, 2002) [5]. A histiocytic tumour of the dog's and other canine's external genitalia is known as a canine transmissible venereal tumour (CTVT), also known as a transmissible venereal tumour, a canine transmissible venereal sarcoma (CTVS), a sticker tumour, sticker tumour and an contagious sarcoma, is passed from one canine to another during mating.

Geographic distribution

With the exception of Antarctica, CTVT has been documented from every continent (Boscos and Ververidis, 2004) [2]. Because of effective management of stray dog populations, rigorous prebreeding inspections, and efficient clinical case management, it is not frequently recorded from Northern and Central European countries and from North America. Except for a few places, such as Puerto Rico, Papua New Guinea, and France, CTVT is still an enzootic disease throughout the rest of the world (Moulton JE, 1990) [13]. Particularly in the Southern United States, CTVT is more prevalent in tropical and subtropical areas. It is the most widespread canine tumour in the Bahamas, Japan, and India (Singh *et al.*, 1991) [18]. The neoplasm was evenly distributed throughout India's various geo-climatic zones (Das and Das, 2000) [3].

Etiology

There is no one factor or circumstance that causes cancer. Instead, a combination of factors that interact over time ultimately lead to cancer. The finding that cancer is more common in older individuals lends credence to the idea that the course of time permits the chain of events necessary for a typical cell to undergo cancer cell transformation (Donald M, 2000) [5]. A variety of transmissible tumour types in humans and different animals have been linked to infectious viral agents, but in TVT, the disease causing agent is the cancer cell itself, and the tumour is clonal in genesis.

M.A. Novinsky initially described the canine TVTs in 1876 after showing that the tumour could be spread from one dog to a different one by infecting them with tumour cells (Martins *et al.*, 2005) [11].

Symptoms and signs

In dogs of both sexes, CTVT frequently affects the external genitalia. Males typically have the tumour in the glans, the caudal portion of the penis, or the foreskin. This tumour frequently develops in females at the intersection of the vestibule and the back of the vagina, and it sometimes develops at the urethral orifice (Madewell, 2001) ^[10]. Rarely are the nose or mouth affected (Morrison, 1998) ^[12]. The tumour frequently resembles a cauliflower. The prepuce discharge and, in certain cases, retention in urine brought on by urethral obstruction are symptoms of genital TVT (Hasler and Weber, 2000) ^[8]. Nasal fistulas, nosebleeds, other discharge from nostrils, facial edema, and swelling of the submandibular lymph nodes are all indications of a nasal TVT (Papazoglou *et al.*, 2001) ^[15].

Pathology

Histiocytic tumours known as “canine transmissible venereal tumours” can spread from dog to dog by mating, licking, biting, and sniffing tumour-affected regions. Three significant observations led to the hypothesis that the tumour is an allograft that is naturally transmissible. First, deceased cells or cell filtrates cannot be used to artificially create CTVTs; only transferring living tumour cells may do so. Second, all tumours gathered from various geographical locations have aneuploid tumour karyotypes, but they also have distinctive marker chromosomes. Third, all tumours analyzed thus far include a long interspersed nuclear element insertion near c-myc, which can be utilized as a diagnostic marker to determine whether a tumour is a CTVT (Murgia *et al.*, 2006; Dingli and Nowak, 2006) ^[14, 4].

The first progressive phase, which typically persists for a few weeks, is distinguished by a 50% cell loss estimate and a fast growth in tumour volume with a duration of doubling period of 4–7 days. The tumour cells use a variety of strategies to avoid immune detection during the progression phase. Transforming growth factor-1 (TGF-1) is produced by these tumour cells, and this cytokine suppresses the function of natural killer cells (NK), the infiltration of cytotoxic lymphocytes, and the expression of MHC. Additionally, chemicals secreted kill B-cells and stop dendritic cells from differentiating and activating (Hsiao *et al.*, 2004) ^[9].

Although limited is known about the specifics of spread, canine transmissible venereal tumours are most frequently observed in sexually active dogs in tropical, and temperate areas where there are high numbers of stray dogs (Vonholdt and Ostrander, 2006) ^[21]. The tumour may spontaneously shrink, most likely as a consequence of an immune system reaction (Stettner *et al.*, 2005) ^[19].

The CTVT goes through a cycle that is predictable: a four to six-month initial growth phase (P phase), a stable period, and a regression phase (R phase), albeit not every CTVTs will regress. Except in young and immunocompromised dogs, the tumour seldom metastasizes (less than 5% of cases), and this is true of most tumours. In addition to local lymph nodes, the epidermis, nervous system, eye, liver, spleen, testis, rectum, and musculature can all show signs of metastasis (Rogers *et al.*, 1998) ^[16].

It takes a biopsy to make a diagnosis. The tumour's process of spread in a particular host system is responsible for this single cell lineage's success, which is thought to be the greatest continuously propagating cell lineage in the world. CTVTs take advantages of the well-known sire effect of domesticated canines, even though direct contact is typically not a very

effective mode of contamination. The tumour can harm many more females than it might if the host were a species that was monogamous since a male can have dozens of litters over the period of his life.

Genetics

Compared to typical dog cells, CTVT cells have a less number of chromosomes. Dogs typically have 78 chromosomes, whereas CTVT cells have 57–64 chromosomes (Martins *et al.*, 2005) ^[11] that look considerably different from dog chromosomes in general. While chromosomes of the CTVT cells are metacentric or sub metacentric, all dog chromosomes aside from X and Y are acrocentric (Hasler and Weber, 2000) ^[8]. This particular cancer's tumour cells all have an incredibly identical genetic makeup that is frequently, but not always, unconnected to the DNA of their host (Murgia *et al.*, 2006) ^[14]. A few other putative driver mutations have been found in addition to the previously described c-myc insertion (Belov *et al.*, 2015) ^[1].

Treatment

Vincristine is a chemotherapeutic treatment that causes the host immune system to become active, which causes the tumour to retreat. CCL5 might be crucial for the immunological response (Frampton *et al.*, 2018) ^[7].

The placement of these tumours may make surgery challenging. Recurrence is frequently caused by surgery alone. For TVTs, chemotherapy is particularly successful. Complete remission following chemotherapy has great prognosis (Ettinger *et al.*, 1995) ^[6]. Vincristine, vinblastine, and doxorubicin are the most frequently utilised chemotherapeutic drugs (Stettner *et al.*, 2005) ^[19]. If chemotherapy is unsuccessful, radiotherapy can be necessary (Rogers *et al.*, 1998) ^[16].

Conclusion

CTVT is the earliest known malignant cell in continuous replication with a single malignant clone of the cells colonizing canines all over the world. CTVT is generally curable with three cycles of intravenous vincristine sulphate injection at a dose rate of 0.025mg per kg body weight. Prevention from the disease will require proper evaluation of the breeding dogs before mating. There should be government policy to control stray dog population to reduce the transmission.

Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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