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# Central nervous system vascular complications associated with the acute form of steroid-responsive meningitis-arteritis

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ABSTRACT

This retrospective study aims to describe the vascular events in the central nervous system (CNS) associated with the acute form of steroid-responsive meningitis-arteritis (SRMA), to compare the clinical features of dogs with and without such complications and to potentially identify predisposing factors for these events. Dogs with a presumptive diagnosis of SRMA visited between 2018 and 2023 with full medical records that underwent neurological examination, blood testing, cervical computed tomography or magnetic resonance imaging and cerebrospinal fluid (CSF) analysis were included.

Thirty-three dogs were included and divided in two groups. Group 1 included 7 (21,2 %) dogs with vascular complications secondary to SRMA (spinal cord ischemic and/or hemorrhagic infarcts, spinal cord subdural hematomas, intracranial subarachnoid hemorrhages), and group 2 included 26 (78,8 %) dogs with non-complicated SRMA. Age, breed, sex, presence of neurological deficits, CSF abnormalities and presence of relapses were factors evaluated for potential association with vascular complications of SRMA.

Six dogs in group 1 were Golden Retrievers (85,7 %), and there was a significant association between this breed and the occurrence of vascular complications ($P = 0.017$). Presence of neurological deficits ($P = 0.001$) and xanthochromic CSF ($P = 0.001$) were also associated with vascular complications in dogs with SRMA.

Hemorrhagic or ischemic lesions in the CNS can be a complication of the acute form of SRMA, and Golden Retrievers appear to be more affected. Dogs with vascular complications show often neurological deficits and CSF xanthochromia.

## Introduction

Steroid-responsive meningitis-arteritis (SRMA) is the most common neurological inflammatory disorder in young, large breed dogs. Two forms have been described: an acute and a protracted form (Tipold and Jaggy, 1994; Cizinauskas et al., 2000; Lowrie et al., 2009; Wrzosek et al., 2009; Tipold and Schatzberg, 2010; Lau et al., 2019). Dogs with the acute form show cervical pain and pyrexia without concurrent neurological deficits and have a marked neutrophilic pleocytosis in the cerebrospinal fluid (CSF) analysis. Dogs with the chronic protracted form exhibit waxing and waning signs of spinal pain and cervical rigidity with evident neurological abnormalities on examination and a mild to moderate mononuclear pleocytosis in the CSF analysis (Maiolini et al., 2013; Remelli et al., 2022; Spitzbarth et al., 2012; Wohlsein and Tipold, 2023).

Several immunological studies have demonstrated that SRMA is a systemic immune-mediated disease. Systemic involvement is supported by the fact that IgA concentrations are consistently increased in both serum and CSF of dogs with SRMA and this increase is higher than in any other central nervous system (CNS) inflammatory disease (Tipold et al., 1995). Additionally, C-reactive protein (CRP) is increased in dogs with SRMA both, in serum and CSF (Bathen-Noethen et al., 2008; Eckersall and Bell, 2010; Biedermann et al., 2016; Andersen-Ranberg et al., 2021; Wohlsein and Tipold, 2023). These characteristics, together with an increased B cell/T cell ratio in peripheral blood, CSF and meningeal lesions are typical features of a generalized humoral immune response (Schwartz et al., 2008, 2011; Spitzbarth et al., 2012; Andersen-Ranberg et al., 2021).

Despite being a systemic disorder, SRMA lesions have a clear

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predilection for the meninges and subarachnoid arteries of the cervical spinal cord (Burgener et al., 1998; Spitzbarth et al., 2012). However, any artery in the body can be affected. In a study including nine Beagles with SRMA histopathological findings revealed panarteritis affecting small and medium-sized blood vessels of the cervical spinal cord, mediastinum, and heart (Scott-Moncrieff et al., 1992). Typical histopathological findings of SRMA include necrotizing arteritis and proliferative lesions of the vascular intima causing blood vessel stenosis and fibrosis with occasional intravascular thrombi (Tipold and Stein, 2010).

Treatment with immunosuppressive doses of corticosteroids often leads to a favorable outcome although relapses are possible and occur in over 30 % of cases (Lowrie et al., 2009). Overall SRMA has a good prognosis if treatment is instituted early in the development of the disease (Tipold and Schatzberg, 2010; Lau et al., 2019).

SRMA has been associated with vascular complications such as spontaneous bleeding within the subarachnoid space in the brain or the spinal cord (Wrzosek et al., 2009; Hughes et al., 2015; Wang-Leandro et al., 2017; Brocal et al., 2017; Zilli et al., 2021). However, the pathophysiology of these events remains uncertain, and no studies have been performed to identify predisposing factors.

Intracranial hemorrhages and subarachnoid spinal bleeding can cause severe clinical impairment and represent a fatal complication of this otherwise benign disease. Such events occur during the acute phase of the disease and mask the typical clinical features of the acute form of SRMA (cervical pain without neurological deficits).

Thus, this study aims to describe the clinical presentation of patients with the acute form of SRMA and CNS vascular complications, as well as to evaluate clinical factors that may be associated with the occurrence of such complications.

## Materials and methods

This retrospective study included dogs that presented to the Hospital Clínic Veterinari of the Universitat Autònoma de Barcelona and had a presumptive diagnosis of SRMA during the period 2018 – 2023.

Inclusion criteria were dogs that: (a) showed consistent clinical signs such as cervical or diffuse spinal pain and pyrexia (rectal temperature >39.2 ºC following a standardized reference (Ramsey and Tasker, 2017)), with or without neurological deficits, (b) had complete medical records that included hematology and serum biochemistry, thoracic radiographs and abdominal ultrasound, and computed tomography or magnetic resonance imaging (MRI) of the spinal cord with compatible findings and/or presence of neutrophilic or mixed pleocytosis on CSF analysis (>5 WBCs/uL with neutrophil predominance) without evidence of microorganisms in the cytological examination or bacterial culture, and (c) that showed clinical response to glucocorticoid therapy. Cases in which extensive CSF blood contamination precluded WBC counting due to the presence of vascular events were included in the study if all other criteria were fulfilled satisfactorily.

Dogs were divided in two groups: group 1 included dogs that suffered vascular complications of acute SRMA, and group 2 included dogs with the acute form of SRMA without any associated complication. Clinical factors assessed for correlation with the development of vascular complications were breed, age, sex and neuter status, neurological signs at presentation, CSF total nucleated cell count (TNCC) and type of pleocytosis, presence of CSF xanthochromia and relapses.

All dogs diagnosed with SRMA at our institution had a complete blood cell count and serum biochemistry, thoracic radiographs, and abdominal ultrasound performed. Dogs in group 1 had specific tests performed to rule out coagulopathies that could contribute to the development of vascular complications, following the criteria of the clinician in each case.

### *Statistical analysis*

Statistical studies were performed using the IBM SPSS software (version 26.0). Descriptive analyses were performed for all variables, and all quantitative variables were tested for normality using the Saphiro-Wilk test and graphical methods. All quantitative variables were non-normal distributed and results are presented as a median (25th-75th quartile). Chi-Square tests were used in order to detect relationship between presence (group 1) or absence (group 2) of complications for breed and sex and type of pleocytosis. Fisher's exact tests were performed to detect relationship between group and presence of CSF xanthochromia, presence of neurological signs on admission and relapses. Comparison between groups for quantitative non normal distributed variables (age) were performed using a Mann-Whitney *U* test. Statistical significance was set at 5 % ($P < 0.05$).

## Results

Thirty-five dogs were diagnosed with SRMA over the five-year period, and two of them were excluded because of incomplete medical records. Thirty-three dogs were finally enrolled in the study, and there were 19 males (57,6 %) and 14 females (42,4 %). Crossbreed dogs were most commonly affected ($n = 9$, 27,3 %), followed by Golden Retrievers ($n = 8$, 24,2 %), Boxers ($n = 5$, 15,2 %), German shepherds ($n$=3, 9,1 %), Border collies ($n = 3$, 9,1 %), Weimaraners ($n = 2$, 6,1 %), and one of each of the following breeds: Rottweiler, Beagle and American Staffordshire ($n = 1$, 3,0 %).

Seven dogs were included in group 1 (21,2 %). These had different vascular lesions: one intramedullary ischemic infarct in the cervical spinal cord, three thoracolumbar subarachnoid hemorrhages, two intracranial subarachnoid hemorrhages, four multifocal intramedullary hemorrhages, and two cervical subdural hemorrhages. The clinical and diagnostic features of dogs in group 1 are detailed in Table 1.

When studying the association between breed and the presence of vascular complications, a significant association was found only with Golden Retrievers ($P = 0017$). Moreover, when considering the only two breeds that suffered vascular complications (Golden Retrievers and crossbreeds), Golden Retrievers were still significantly associated to vascular complications when compared with crossbreeds ($P = 0015$),

When including all dogs, median age at diagnosis was 9 months (6 – 13 interquartile range). Dogs in group 1 tended to be older (median age 11 months, 6 – 17 interquartile range) than those in group 2 (median age 8,5 months, 6–11 interquartile range), although this difference was not statistically significant ($P = 0214$). No significant differences in sex distribution or neuter status were observed between the two groups ($P = 1000$, and $P = 0538$, respectively).

All dogs diagnosed with SRMA had an elevated rectal temperature (>39.2 ºC) at presentation. Neurological deficits on examination were found in 7/26 dogs in group 2 (26,9 %) and in 7/7 dogs in group 1, and this difference was statistically significant ($P = 0001$).

Regarding blood test results, haematology revealed neutrophilic leukocytosis in 28 cases (6/7 dogs in group 1 and 22/26 in group 2). In two dogs the results of the hematological tests were not available for review (one of each group) and for the three remaining cases of group 2 leukocyte values were within normal range. The results of serum biochemistry, thoracic radiographies and abdominal ultrasound were unremarkable or did not show alterations that could be related to the clinical and neurological presentation of the dogs. Serum CRP was measured in 2/7 cases of group 1 and 9/26 cases of group 2, and it was above reference range in all cases.

CSF pleocytosis was found in all dogs in group 2 and in 5/7 dogs in group 1. The remaining two dogs in group 1 had evident macroscopic blood contamination that precluded WBC count. CSF cytology revealed neutrophilic pleocytosis in 87.1 % of cases, but mononuclear and mixed pleocytosis were also recorded ($n = 2$ each, 6,5 %). No significant differences were observed between the two groups regarding type of pleocytosis ($P = 0.428$). Xanthochromic CSF was found in 5/7 (83.3 %) dogs in group 1 and in two dogs in group 2 (8,3 %). In the 2 cases in group 1 in which TNCC was not performed due to excessive blood

**Table 1**
Clinical and diagnostic findings of dogs in group 1.

| Case | Neurological signs | MRI | CSF |
|---|---|---|---|
| Crossbreed, 15 mo, intact male | Ambulatory tetraparesis, severe cervical pain | C2-C3 ischemic infarct | Neutrophilic pleocytosis (2400 WBCs/uL), xanthochromia |
| Golden Retriever, 11 mo, intact male | Ambulatory paraparesis, proprioceptive ataxia of the pelvic limbs, thoracolumbar pain | Subarachnoid hemorrhage extending from T12-L2 | Neutrophilic pleocytosis (51 WBCs/uL) |
| Golden Retriever, 11 mo, intact male | Obtunded mental status, non-ambulatory tetraparesis. Progresses to deep pain negative paraplegia in 48 hours | Brain: subarachnoid hemorrhage causing compression over the brainstem SC: T7-T8 intramedullary hemorrhage and T10-L1 subarachnoid hemorrhage | TNCC NA due to severe blood contamination, xanthochromia |
| Golden Retriever, 18 mo, intact female | Obtunded mental status, non-ambulatory tetraparesis with deep pain negative paraplegia, bilaterally absent menace response, generalized tremors | Brain: subarachnoid hemorrhage in the caudal fossa extending over the foramen magnum SC: T8-T11 multifocal intramedullary hemorrhages, diffuse spinal subarachnoid hemorrhage | Neutrophilic pleocytosis (138 WBCs/uL), xanthochromia |
| Golden Retriever, 6 mo, neutered female | Non-ambulatory tetraparesis, cervical hyperesthesia | Intramedullary hemorrhage extending from C1-T5 | TNCC NA due to severe blood contamination, xanthochromia |
| Golden Retriever, 15 mo, intact male | Ambulatory tetraparesis and proprioceptive ataxia of all limbs, cervical hyperesthesia | C1-C3 subarachnoid hemorrhage | Neutrophilic pleocytosis (1500 WBCs/uL), |
| Golden Retriever, 6 mo, intact female | Proprioceptive ataxia of all limbs, cervical hyperesthesia | Subarachnoid hemorrhage at C3, intramedullary hemorrhage extending from C1-C3 | Neutrophilic pleocytosis (790 WBCs/uL), xanthochromia |

MRI, Magnetic resonance imaging; CSF, Cerebrospinal fluid; SC, Spinal cord; TNCC, Total nucleated cell count; NA, Not available;

contamination, xanthochromia was evident after CSF centrifugation. The presence of xanthochromia was significantly associated with presence of vascular complications ($P = 0001$). Serological and CSF infectious agent testing yielded negative results in all cases. The main clinical and diagnostic findings of both groups are summarized in Table 2.

Coagulation times (prothrombin time and partial thromboplastin time) were performed in 5/7 dogs. Specific infectious agent testing for *Leishmania infantum, Dirofilaria immitis, Borrelia burgdorferi, Ehrlichia* spp. and *Anaplasma* spp. were performed in three cases. Two animals were tested for *Bartonella henselae* infection and one for *Angyostrongylus vasorum* antibodies. Fibrinogen and von Willebrand factor were determined in one dog. All results were within normal limits.

All dogs received immunosuppressive treatment with corticosteroids according to the protocol described by Tipold et al. (2010). For those animals that had finished the treatment protocol at the time of writing (30/33), median treatment duration was 7 months (range 2 – 16), and the length of treatment was not significantly different between groups ($P = 0.354$). Presence of relapses could only be assessed in 23 cases (4/7 cases in group 1 and 19/26 in group 2), because three dogs in group 2

**Table 2**
Comparison of main clinical and diagnostic findings between dogs in groups 1 and 2.

| | SRMA with associated vascular complications | Non-complicated SRMA cases |
|---|---|---|
| Number of dogs (*n*) | 7 | 26 |
| Median age (median, months) | 11 | 8,5 |
| Body weight (median, kg) | 26 | 20 |
| Dogs presenting with fever (%, *n*) | 100 (7/7) | 100 (26/26) |
| Dogs presenting with neutrophilic leukocytosis (%, *n*) | 100 (6/6) | 88,8 (22/25) |
| Dogs presenting with neurological deficits (%, *n*) | 100 (7/7) | 26,9 (7/26)* |
| Dogs presenting with CSF pleocytosis (%, *n*) | 100 (5/5) | 100 (26/26) |
| Dogs presenting with CSF xanthochromia (%, *n*) | 71,4 (5/7) | 4,3 (2/23)* |
| Length of treatment (median, months) | 7 | 8 |
| Relapses (%, *n*) | 50 (2/4) | 36,8 (7/19) |
| CRP value (median, mg/l) | 87,3 | 108,5 |

CSF, Cerebrospinal fluid; CRP, C-Reactive protein;
* $P < 0,05$

and two dogs in group 1 were still receiving treatment at the time of writing and 4 dogs were lost to follow-up after treatment discontinuation. In group 1, 2/4 (50 %) dogs suffered one relapse, whereas in group 2, seven dogs (36,8 %) suffered at least one relapse, and 2 of these dogs relapsed twice. Even though the relapse rate was higher in dogs from group 1, the difference was not statistically significant ($P = 0.624$). The overall outcome of all dogs included in the study was favorable, although two dogs in group 1 became paraplegic without deep pain sensation after the occurrence of a spinal cord subarachnoid hemorrhage (Fig. 1). After treatment, fever and spinal pain disappeared in both animals, but only one dog recovered deep pain sensation and ambulation.

## Discussion

SRMA is a common disease that has received much attention in the past 20 years. However, few reports can be found in the literature describing the occurrence of vascular complications in the CNS caused by the systemic arterial inflammation (Hughes et al., 2015;

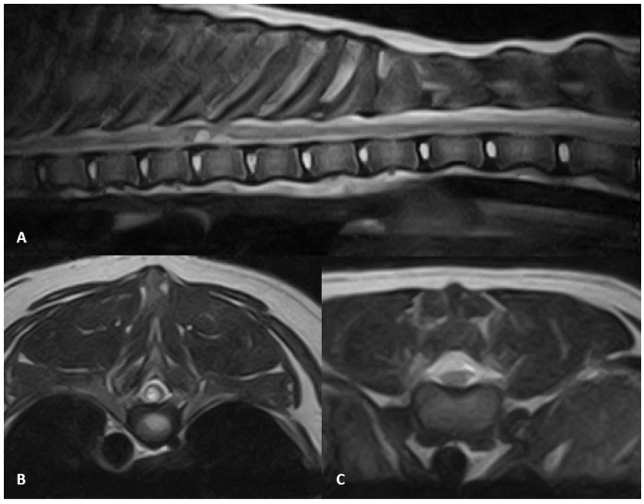


**Fig. 1.** T2W sagittal plane of the thoracic spinal cord (A) and transverse views at the level of T8 (B) and T12 (C). Hyperintense intramedullary lesion consistent with hematomyelia (A, B). Diffuse enlargement of the subarachnoid space causing dorsal spinal cord compression over T10-L1, consistent with subdural hemorrhage (A, C).

Wang-Leandro et al., 2017; Zilli et al., 2021; West et al., 2023). The overall prevalence of vascular complications in our study was 21,2 %, which is a remarkable proportion of acute SRMA cases. Such high prevalence could be attributed to the advances in veterinary medicine and the establishment of cross-sectional imaging techniques as standard procedure for the diagnosis of neurological diseases. The authors hypothesize that such complications have always existed but remained underdiagnosed, especially considering that definitive diagnoses of both SRMA and neurovascular events rely on histopathology, thus their association is highly difficult to demonstrate. Another reason that could account for the high prevalence of vascular complications in this study could be a population bias given that this was a single-center study. Future larger-scale multi-centric studies should be performed to address this matter. Nevertheless, to the authors' knowledge, this is the first study that aimed to describe thoroughly these complications and to identify clinical factors associated with their occurrence.

Traditionally, the presence of neurological deficits has been associated with the chronic protracted form of SRMA, and dogs with the acute SRMA form just show cervical hyperesthesia and pyrexia (Tipold and Schatzberg, 2010). Moreover, in some SRMA pathophysiological studies the presence of neurological deficits is considered an exclusion criterion (Lau et al., 2019). The assumption that acute SRMA patients cannot exhibit neurological deficits may lead to a clinical bias, and SRMA might not be considered as a differential diagnosis in animals with associated vascular complications because these can cause a variety of clinical signs depending on lesion localization and mask the traditional signs of SRMA. Vascular events can present in different ways: spinal cord ischemic or hemorrhagic infarcts, microbleeds or extensive subarachnoid or intramedullary hemorrhages.

It is currently unclear whether chronic SRMA cases can also be associated with such complications and if that could be the reason behind the neurological deficits that have been historically described in this form of the disease. Even though studying this was beyond the scope of this study, the authors believe that exploring possible vascular complications in chronic SRMA in the future would be helpful for the general understanding of the disease.

Currently, there is a lack of a specific biomarker for the diagnosis of SRMA (Andersen-Ranberg et al., 2021), and definitive diagnosis can only be reached postmortem through histopathological studies. The typical clinical picture (young, large-breed dogs with signs of cervical hyperesthesia and fever) and the presence of neutrophilic pleocytosis (usually with a severely elevated TNCC) in the absence of microorganisms in the CSF analysis are the mainstay for a presumptive diagnosis of SRMA (Cizinauskas et al., 2000; Tipold and Schatzberg, 2010). However, diagnosis of SRMA in dogs with vascular complications is further hindered due to the presence of blood contamination in the CSF of many animals. Although it is not infrequent to detect red blood cell (RBC) contamination in the CSF analysis of dogs with SRMA due to the intrinsic blood vessel inflammation (Wohlsein and Tipold, 2023), the existence of extensive subarachnoid bleeding may mask the underlying pleocytosis due to large amounts of RBCs in CSF samples. During macroscopic observation of the CSF, a red or yellowish colour can be indicative of hemorrhage into the subarachnoid space. Centrifuging the samples and detecting CSF xanthochromia (persistence of this yellowish appearance) should help to exclude iatrogenic hemorrhage (Vernau et al., 2008; Levine and Cook, 2020). In this study, the authors found a significant association between the occurrence of vascular lesions and CSF xanthochromia.

Due to the retrospective nature of the study, CRP measurement was not consistently performed in all cases. Only eleven dogs in this study had serological CRP testing and it was elevated in all cases, which supported the diagnosis of SRMA. However, statistical analysis correlating CRP values and the occurrence of vascular complications was not performed due to insufficient data. The authors suggest that further studies include serum CRP values as this will increase the usefulness of this marker in future findings.

Several breeds have been previously described to be predisposed to SRMA, including Golden Retrievers (Biedermann et al., 2016; Lau et al., 2019; Hilpert et al., 2020; Wohlsein and Tipold, 2023). Our study included 8 Golden Retrievers and 6 of them suffered vascular complications, showing a significant association between Golden Retrievers and the occurrence of these complications. The reason behind this association is currently unknown and should warrant further investigation. Additionally, one of these dogs had been diagnosed with hemothorax a month prior to SRMA diagnosis, which was thought to be caused by systemic arteritis. Therefore, it is possible that different genetic backgrounds may lead to different degrees of severity of the disease, and other breeds could be predisposed to develop vascular complications of SRMA. In accordance with this, a recent study published by West et al. (2023) reported three Whippets with subarachnoid bleeding secondary to SRMA.

Relapses have been described to occur in 16–60 % of dogs with SRMA (Cizinauskas et al., 2000; Bathen-Noethen et al., 2008; Biedermann et al., 2016; Lau et al., 2019; Hilpert et al., 2020; Giraud et al., 2021; Wohlsein and Tipold, 2023). Relapse rates in our study were similar, 50 % in group 1 and 36,8 % in group 2. A higher tendency was found in dogs with vascular complications, although the difference was not statistically significant. Larger studies should be performed to ascertain if complicated SRMA cases are associated with higher relapse rates. In fact, neurological deficits caused by vascular complications can be irreversible as happened in the dog that became paraplegic and lost deep pain sensation. In some cases, the prognosis can be largely influenced by the severe consequences of the vascular complications.

This study has several limitations: first, the number of dogs in group 1 is small and because of this some of the statistical analyses were not feasible. A larger sample size would be needed to perform statistical analyses that could demonstrate the association of other clinical factors with the occurrence of vascular complications. Moreover, all vascular events were considered together, even though the pathophysiology of ischemic infarctions and hemorrhages is different. It would be interesting to evaluate the different vascular events independently and analyze a potential association with the aforementioned clinical factors, for both acute and chronic SRMA cases. Moreover, none of the animals in group 1 died or was euthanised so histopathological studies were not performed in any of them. Because of this, none of the vascular lesions could be histopathologically confirmed, which can be a major limitation of the study. However, the abrupt onset of neurological deterioration after a period of lethargy, inappetence and presence of diffuse pain, as well as advanced imaging findings and CSF xanthochromia were highly compatible with vascular events secondary to SRMA.

Finally, given the retrospective nature of the study, dogs in group 1 had different diagnostic tests performed in order to exclude potential causes of coagulopathy. However, none of the affected dogs had suffered any vascular event unrelated to the diagnosis of SRMA that could indicate a potential underlying coagulopathy.

Taking all into consideration, the present study may assist the clinician to recognize SRMA patients even when they do not present with the classical form of the disease and this could imply an earlier diagnosis. Further investigation of these relatively frequent vascular complications is needed, especially given that prognosis of the disease may be influenced by their occurrence. There are currently many knowledge gaps regarding the pathophysiology of these complications and the findings of the present study could be used for further investigations in the future.

## Conclusions

Hemorrhagic and ischemic lesions affecting the CNS (intraparenchymal lesions or subarachnoid bleeding causing spinal compression) occurred in 21,2 % of dogs with SRMA. These complications may mask the traditional clinical presentation of acute SRMA and complicate diagnosis. The results of this study show that Golden

Retrievers are predisposed to vascular complications of SRMA and that neurological deficits on presentation and CSF xanthochromia are clinical factors associated with these complications. Larger studies are needed to identify other factors that might predispose to the development of such complications.

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## Conflict of Interest

None of the authors has any financial or personal relationship that could inappropriately influence or bias the content of the paper.

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