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Protective effect of chicken egg yolk antibody in colostrum-deprived neonatal puppies

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초유결핍 신생자견에서 난황 항체의 방어효과

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초 록 : 총 15두의 초유를 섭취하지 않은 신생자견을 대상으로 난황항체를 경구투여한 후 개 파보바이러스를 경구 접종하여 실험감염을 유발시켜 난황항체의 수동 면역에 의한 예방효과를 알아보고자 한다.

항체역가는 면역화된 산란계로부터 분리한 난황항체를 투여한 자견이 비면역 난황항체를 투여한 자견에 비해 높았다. 개 파보바이러스 접종 직전의 항체역가는 대조군의 경우 1:40에서 1:80, 실험군의 경우는 1:320에서 1:1280이었다. 모든 대조군의 자견들은 바이러스 접종후 4일에 임상증상을 나타내었고 총 7두중 6두가 폐사된 반면 실험군 자견은 2두만이 증상을 나타내었고 폐사 자견은 없었다($p<0.01$). 개 파보바이러스를 경구 접종한 후 전체 자견의 혈구응집억제반응역가는 접종후 6일까지 감소하는 경향을 보였다. 접종후 5일의 분변내 혈구 응집반응역가는 실험군 자견의 경우 < 2에서 64였으며 대조자견은 216에서 2048로 높았다.

Key words : *canine parvovirus,* hemagglutination inhibition test, egg yolk antibodies, passive immunization, HPMCP.

Introduction

Canine parvovirus infection has been associated with one of the most common acute and highly contagious gastroenteritis characterize by lethargy and inappetence followed by an acute onset of diarrhea, vomiting, and fever[1,3]. Studies on pound dogs and family dogs have shown that parvoviral diarrhea was more severe and prevalent than all other causes of viral diarrhea. The virus has an affinity for

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the rapidly dividing cells of the intestine, bone marrow, and lymph tissues, and thus causes intestinal crypts necrosis, severe diarrhea, leukopenia and lymphoid depletion[1,4]. Bloody diarrhea, fever, leukopenia and death are much more likely to be associated with parvoviral diarrhea than coronaviral or parasitic diarrhea[5]. dogs of any age can be infected, but the incidence of clinical disease is highest in puppies between 6 and 20 weeks of age[3].

Diagnosis is relatively easy and often made by clinical observations. Few diseases in dogs cause the constellation of acute enteritis, fever, and leukopenia. confirmation of active parvoviral disease is most easily made by demonstrating canine parvovirus in the stool, a relatively simple task in acute cases because of the amount of virus present. There are a number of different methods available -- viral isolation, fecal hemagglutination or ELISA, electron microscopy[6,7]. These tests have been available only at diagnostic laboratories but the advent of a solid-phase ELISA test for parvovirus made it possible to test in practice. Treatment of CPV enteritis is non-specific and supportive care including fluid therapy is the most important for patients. The general guidelines for any acute nonspecific gastroenteritis should be followed.

The principal challenge for practitioners in CPV infection is to provide protection for young puppies. Although widespread vaccination against CPV-2 has markedly reduced the incidence of the disease, parvoviral enteritis is still common in puppies as they are nearing the end of their maternal antibody protection at 6 to 20 weeks of age[8]. The most common causes of a vaccine failure are inadequate quarantine or hygiene practice and vaccine interference by maternal antibodies[9].

In susceptible populations, most adult animals seroconvert without manifesting clinical signs indicating that mild or inapparent infection is common, and thus enteritis may spread rapidly through the younger animals. A substantial problem is that virulent virus is able to infect and cause severe disease in animals with levels of maternal antibody that prevent active immunization[10]. There is a 2- to 5- week window of vulnerability when animal can be infected with virul ent virus but cannot be successfully immunized, so called the critical period of susceptibility. Puppies do not seroconvert to canine parvovirus until their maternal antibody titers drop below a protective level between 6 to 15 weeks of age, indicating that maternal antibody itself sufficient to prevent active infection[11~13]. Puppies vaccinated when their maternal antibody titers are still high have no evidence of an active immune response. Puppies from bitches with low titers may be susceptible as early as 4 to 6 weeks after birth, whereas puppies from bitches with high titers may be refractory to infection for 12 to 16 weeks[3]. Virtually, the amount of maternal antibody required to prevent infection is greater than the amount of that interferes with vaccination[12].

In some breeding kennels where the level of environmental contamination with CPV is so high virtually every puppy born within the kennel contacts with CPV before it can be successfully immunized. In kennels with this problem, the best way is the total isolation of the puppies from kennel at 4 to 6 weeks of age. It is not to allowed for them to return until they have completely their full immunization program, at approximately 6 months[10]. Also it is important that these isolated puppies should not have direct or indirect contact with persons or equipments from the contaminated kennel until their immunization program in complete, because CPV is so stable in outside environment and can persist on fomites for weeks. But it is difficult to implement that way in breeding kennel. Thus, prevention of exposure is almost impossible[3]. Almost all apparent vaccination failures in puppies probably result from exposure to infection during critical period of susceptibility.

The most appropriate vaccine at present is a modified-live preparation of parvovirus of canine origin. The vaccination schedule adopted in most practice is administration of the vaccine every 2-3 weeks from 6-9 weeks of age until the puppy reaches 16-20 weeks of age[8]. But the immunity gap can not be filled by more frequent vaccination because intervals of less than 14 days result in vaccine interference[9].

Therefore, although a variety of vaccines against canine parvovirus are developing both alone and in combination with other canine pathogens, maternal antibody interference with vaccination accounts for the vast majority of vaccine break. As the CPV is firmly entrenched in both the wild and domestic canine population, elimination of the virus is impossible.

Most young animals diarrheal diseases due to a various virus and bacteria represent considerable therapeutic, preventive and economic problems. In puppies canine parvovirus infection is the most fatal disease. Most common measures to control diarrhea are application of antibiotics and symptomatic substitution of water and balanced-electrolytes solution. Thus it is necessary to seek the other ways to control the young animals diarrhea include the active immunization. In case of enterotoxigenic colibacillosis in calves and piglets, maternal immunization in order to establish a local immunological protection in the gut via colostrum were reported[14,15].

Oral administration of antibodies derived from serum and colostrum and even with monoclonal antibodies has been very successful and the therapeutic studies of passive local immunity in the small intestine were recently performed[16~21]. However, as it is prohibitively expensive to obtain the large amounts of specific antibodies required from serum, milk and colostrum, a considerable technical and financial effort is necessary. There is increasing interest in the use of chicken egg yolk for antibody production as the antibody source. This approach is relevant not only for production of antibodies to be used in immunochemical assays but also for antibodies to be used therapeutically in passive immunization programs[21, 22]. With regard for animal welfare as well as cost, the chicken egg is a cheaper and attractive alternative to these antibody sources[23,24,20].

The present study is concerned with the prophylactic use of crude chicken egg yolk antibodies as an alternative agent for the direct passive immunization to the canine parvovirus infection in colostrum-deprived neonatal puppies.

Materials and Methods

Experimental Animals : Fifteen colostrum-deprived mixed breed puppies from the three litters were used. Dams of puppies(n=3) were used, between 2 and 5 years old and weighing between 6 and 12 kg for studies. Dams were individually housed in container house, were fed commercial dog food twice a day, and were provided water ad libitum. From 5 days before whelping, body temperature was monitored twice a day and blood was collected for HI titer, and two to 4 days before whelping, 24-hour observation was performed. Whelping was observed and puppies were immediately separated from their dam. All puppies(n=15) were restricted from suckling and permanently separated from their dams, deprived of colostrum, and fed a bitch-milk substitute. Every puppies were healthy, vigorous, and had normal suckling responses. Body weight at birth was measured. They were fed with milk bottle.

Blood samples : Less than one ml of blood was collected on days 0, 1, 3, 5, 7, 9, 10, 11 and 12 after birth by jugular veinpuncture for each whelp. Sera for antibody titer determination against canine parvovirus were separated after overnight incubation at 4℃ and stored frozen at -20℃ until the analysis.

Oral administration of antibody : After oral administration of 1 ml of carbonate-bicarbonate buffer(pH 9.6), 600mg of crude chicken egg yolk antibody or placebo was administered orally through the 5 Fr feeding tube once a day for 3 days after birth.

Challenge : Inoculation of virus was done as follows. Puppies were challenged canine parvovirus(NVSL) with two times 10.5 log 10 TCID50/ml containing 2000HAU per orally on 4 days after birth.

Clinical observations : All the puppies were observed at least twice a day. Feces were collected and examined carefully for the consistency during the clinical observation. The total body weight, heart rate, respiration rate and rectal temperature were noted.

Hemagglutination tests : The HA test of puppies feces was performed by a method described by Carmichael[25].

Hemagglutination inhibition test : The HI test of puppies serum was performed by a method described by Carmichael *et al*[25].

Results

Clinical signs : The clinical signs of placebo and treatment group are shown in Table 1 and 2. In placebo group of each litter all puppies showed clinical signs such as depression, anorexia, diarrhea and vomiting and 5 of 7 pup-

Table 1. Clinical signs in experimental puppies

Group (Pup No.)	Clinical signs					
	Depression	Anorexia	Dirarrhea	Hemorrahagic	Vomiting	Death
Placebo (1,2,5,6,10,11,12)	7/7	7/7	7/7	5/7	4/7	6/7
CEYA (3,4,7,8,9,13,14,15)	0/8	1/8	1/8	0/8	0/8	0/8

Table 2. Clinical signs of newborn puppies after challenge with canine parvovirus

Litter	No.of puppies	Day after inoculation												
		0	1	2	3	4	5	6	7	8	9	10	11	12
1	1C	-	-	-	-	A+,D+,V	A++,D++	A++, D+++	A++, D+++	Die				
	2C	-	-	-	-	A+	A++,E	A+++, D+++	Die					
	3T	-	-	-	-	D++	D+	-	-	-	-	-	-	-
	4T	-	-	-	-	-	-	-	-	-	-	-	-	-
2	5C	-	-	-	A+,D	A+,D++	Die							
	6C	-	-	-	-	A++,D++	A++,V, D+++	Die						
	7T	-	-	-	-	-	-	-	-	-	-	-	-	-
	8T	-	-	-	-	-	-	-	-	-	-	-	-	-
	9T	-	-	-	-	-	-	-	-	-	-	-	-	-
3	10C	-	-	-	-	A+,E	A++,E,D+	A+++, D++	A+,V, D+++	D++	-	-	-	-
	11C	-	-	-	-	E	A+,D++	A++,D++	A++,V, D+++	A+++, D+++	Die			
	12C	-	-	-	-	D++	E,D++	A+,D+++	A++, D+++	Die				
	13T	-	-	A+	A+	-	-	-	-	-	-	-	-	-
	14T	-	-	-	-	-	-	-	-	-	-	-	-	-
	15T	-	-	-	-	-	-	-	-	-	-	-	-	-

A : depression and anorexia
D : Diarrhea
V : Vomiting
E : Emesis
C : placebo
T : administration of immunized chicken egg yolk

+ Mucoid
++ Watery
+++ Hemorrhagic
+ to +++ on severity

pies showed bloody diarrhea. Six of 7 puppies were died due to canine parvovirus infection. clinical signs were commenced on the 3 days or 4 days after virus inoculation. With slight dullness, weak suckling capability and inappetence No. 5 pup showed watery diarrhea was observed on day 3 after inoculation and died on day 5 after inoculation. By day 5 after inoculation, all puppies were dull and inappetency, in three(Nos. 1, 11 and 12) there were watery diarrhea and No. 6 pup showed hemorrhagic diarrhea without vomiting. In one of these puppies(No. 6), clinical signs were rapid worsening with soft mucoid feces progressing to foul hemorrhagic feces accompanied by dehydration, and then died on day 6 after inoculation.

On day 6 after inoculation, the remaining 5 puppies of placebo group showed severe clinical signs without vomiting, such as profuse watery diarrhea or dark, foul smelling, hemorrhagic feces. Four of these Nos. 1, 2, 11 and 12 were died on 7, 8 and 9 days after inoculation respectively. Only

Table 3. Fecal consistency score in experimental puppies

Group	No. of puppies with diarrhea/total No. of pups on day				No. of dead/total puppies(%)
	0	4	6	8	
Placebo	0/7(0)	4/7(1.0)	5/5(3.0)	2/2(2.5)	6/7(85.7%)
CEYA	0.8(0)	1/8(0.25)[a]	0/8(0.0)[a]	0/8(0)[a]	0.8/(0.0%)[b]

a : $p<0.05$, b : $p<0.01$

Table 4. HI antibody titers of dams and puppies

Litter	Dam's titer	No. of puppies	HI titer of puppies	
			presuckle	3 days old
1	640	1	80	80
		2	40	40
		3	40	1280
		4	40	640
2	1280	5	40	40
		6	40	50
		7	40	640
		8	40	640
		9	80	1280
3	2560	10	80	80
		11	40	40
		12	80	80
		13	80	1280
		14	40	320
		15	80	640

one pup survived during observation period of 12 days.

In the chicken egg yolk immunoglobulin administration group of each litter all puppies did not show any clinical signs except 2 puppies. One pup of litter 1 showed diarrhea for 2 days through the day 4 and 5 days after inoculation and No. 13 pup of litter 3 showed partial anorexia on day 2 and day 3 after inoculation. However they survived during observation period of 12 days and showed mild clinical signs without hemorrhagic diarrhea and vomiting(Table 2). Difference in fecal consistency scores between the treated and placebo puppies was significant($p<0.05$) on day 4, 6 and 8 after inoculation. Difference in mortality rate between two groups was significant($p<0.01$)(Table 3).

Antibody titer in serum : The HI titer of each dam(Nos. 1, 2 and 3) were 1:640, 1:1280 and 1:2560, respectively. The HI titer of puppies after birth were ranging from 1:40 to 1:80. On three days after administration of chicken egg yolk antibodies, antibody titers of puppies were ranging from 1:320 to 1:1280. In placebo group the titer of puppies were same as the titer at birth(Table 4). Generally, antibody titers were higher in puppies administrated with chicken egg yolk antibodies from immunized hens than administrated with chicken egg yolk from nonimmunized hens. As shown in Table 5 and Fig 1, the 2 puppies(Nos. 3 and 4) of litter 1 had HI titer of 1:1280 and 1:640, but puppies of placebo (Nos. 1 and 2) had a HI antibody titer of 1:80 and 1:0 on 0 days after virus challenge. Mean antibody titer of puppies administrated with CEYA was decreased rapidly to 1:800 at 8 days after virus inoculation.

Table 5. Serological examination in puppies of litter 1 for the presence of HI antibody to CPV

Pup	Days after challenge						
	0	1	3	5	6	7	8
1c	80	80	80	40	40	20	-
2c	40	40	20	20	20	-	-
3	1280	1280	1280	640	640	640	320
4	640	640	320	320	320	160	160

c : control

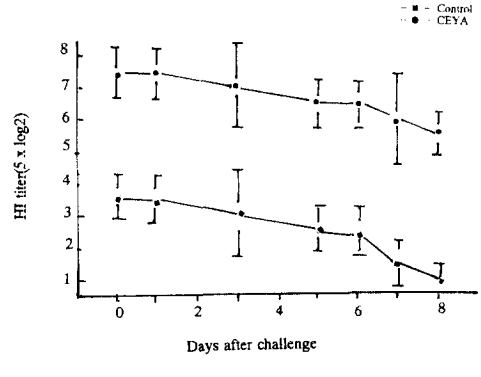

Fig 1. Mean serum HI antibody titer to CPV in puppies of litter 1 after experimental CPV infection.

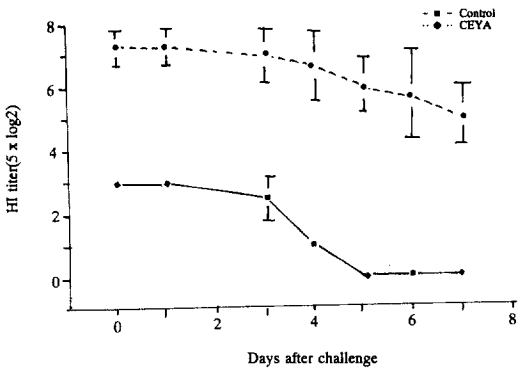

Fig 2. Mean serum HI antibody titers to CPV in puppies of litter 2 after experimental CPV infection.

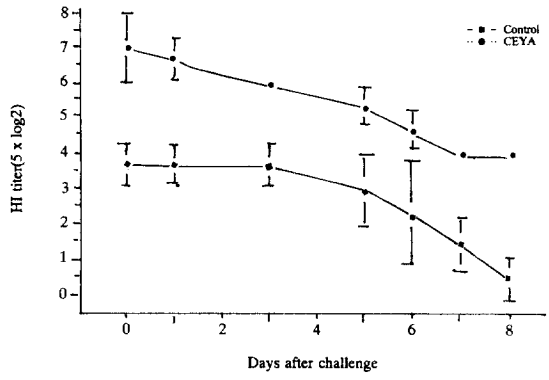

Fig 3. Mean serum HI antibody titers in puppies of litter 3 after experimental CPV infection.

Table 6. Serological examination in puppies of litter 2 for the presence of HI antibody to CPV

Pup	Days after challenge						
	0	1	3	5	6	7	8
5c	40	40	40	-	-	-	-
6c	40	40	20	<10	-	-	-
7	640	640	640	320	320	320	160
8	640	640	320	320	160	80	80
9	1280	1280	1280	1280	640	640	-

c : control

Table 7. Serological examination in puppies of litter 3 for the presence of HI antibody to CPV

Pup	Days after challenge						
	0	1	3	5	6	7	8
10c	80	80	80	80	80	-	-
11c	4	40	40	20	<10	<10	<10
12c	80	80	80	40	20	20	-
13	1280	640	320	320	160	80	80
14	320	320	320	160	80	80	-
15	640	640	320	160	160	80	-

c : control

Table 8. Fecal examination for the presence of canine parvovirus hemagglutinin in puppies of litter 1

Pup	Days after challenge						
	0	1	3	5	6	7	8
1c	<2	<2	<2	1024	1024	1024	D
2c	<2	<2	<2	2048	2048	D	
3	<2	<2	<2	16	16	16	<2
4	<2	<2	64	<2	<2	<2	<2

c : control, D : death

Table 9. Fecal examination for the presence of canine parvovirus hemagglutinin in puppies of litter 2

Pup	Days after challenge						
	0	1	3	5	6	7	8
5c	<2	<2	<2	D			
6c	<2	<2	64	2048	D		
7	<2	<2	<2	256	<2	<2	<2
8	<2	<2	<2	64	64	64	<2
9	<2	<2	<2	<2	<2	<2	<2

c : control, D : death

As shown in Table 6 and Fig 2, puppies(Nos. 7, 8 and 9) administrated with CEYA in litter 2 had HI titers of 1:640, 1:320 and 1:1280, respectively on 3 days after virus challenge and showed an decrease at 6 days. Mean antibody titer of puppies administrated with CEYA was decreased as same in pup of litter 1.

The puppies administrated with CEYA of litter 3, as shown in Table 7 and Fig 3, had a high mean HI titer which was 1:746 on 3 days after virus inoculation. On 3 days after challenge all of the puppies administrated with CEYA had a antibody of 1:320 HI titer and the titers were decreased to 80 on 7 days after virus inoculation. The an-

Table 10. Fecal examination for the presence of canine parvovirus hemagglutinin in puppies of litter 3

Pup	Days after challenge						
	0	1	3	5	6	7	8
10c	<2	<2	64	128	2048	2048	2048
11c	<2	<2	<2	216	256	1024	1024
12c	<2	<2	<2	256	512	1024	D
13	<2	<2	<2	16	64	<2	<2
14	<2	<2	256	<2	<2	<2	<2
15	<2	<2	16	64	<2	<2	<2

c : control, D : death

tibody titer of puppies in placebo was ranging from 1:40 to 1:160.

Viral shedding after challenge : Fecal hemagglutinin results of feces in puppies of each litter were shown in Table 8, 9 and 10. Fecal antigen were first detected in puppies on 3 days after challenge. Challenge virus was first excreted in the feces of puppies 1, 2, 11 and 12 at 5 days after challenge, and excreted during the subsequent three days. High titers of fecal antigen were detected in puppies 2 and 6 account for 1:2048, and low titer was detected in pup 1, 11, 12 accounting for 1: 1024. In pup 5 fecal antigen was not detected.

In CEYA administration group of each litter viral antigen were first detected in puppies 4, 14 and 15 on 3 days, and in pups 3, 7, 8 and 13 on 5 days after challenge. Puppies 3 and 8 excreted the virus for 3 days accounting for 1:16, 1:64 HA titer, respectively. Fecal antigens were detected in pups 4, 7 and 14 just for one days accounting for 1:64, 1:256 and 1:256 HA titer, respectively. HA titers of pups 13 and 15 were 1:64 on 6 and 5 days after challenge, respectively. In only one pup 9 antigen was not detected.

Discussion

In present study, I evaluated efficacy of the antibody from egg yolks of laying hens immunized with the commercial inactivated canine parvovirus vaccine on experimentally induced CPV infection in neonatal puppies. My results support the use of CEYA as an alternative method for direct passive immunization of newborn puppies against infectious intestinal disorders instead of colostrum, serum and hybridoma-derived monoclonal antibodies. Significant protection was achieved in all puppies administered with CEYA against CPV infection. When antibodies was administered in puppies, these puppies did not have severe diarrhea, and no puppies died after oral challenge of CPV. On the contrary, all puppies of the control group manifested severe clinical signs and succumbed to the disease, resulting in 85.7% mortality.

It is known that bovine milk immunoglobulins resist proteolysis and retain some specific antibody activity after passage through GI tract of infants[26]. Other studies have demonstrated that in adults, bovine immunoglobulin resist proteolysis and inactivation by gastric acid when antacid is given after meal[27]. The pH in the stomach of infants up to 5 months of age frequently stays between 4-5 even 2-3 hours after the intake of milk[28]. Appreciable inactivation of IgY in the infantile stomach in unlikely[29]. I used carbonate-bicarbonate buffer(pH 9.6) prior to administration of the egg yolk antibodies to protect the immunoglobulin in the stomach so that it could reach the small intestine.

Neonatal puppies are considered to be immunologically immature at birth and the newborn pup would be susceptible to a wide variety of infection without a competent immune system[30]. In the dog, most of this protection is transferred in the postpartum period[31]. The transport of colostrum from the bitch to the pup in the first 48 hours of life is critical to the overall health of the young dog[32]. Only 5 to 10% of total immunoglobulin to CPV is acquired transplacentally in puppies[33]. Transfer of the maternally-derived antibody to CPV accounts for approximately 90% of total antibody[12]. Colostrum contains 1,500mg of immunoglobulin/ 100ml at the time of parturition and decreases to 200 to 300 mg/100ml by 2 day after whelping[34]. Immunoglobulins secreted in colostrum are readily absorbed in the blood from the upper part of the small intestine. Absorption of macromolecule by neonates from the intestinal lumen into the blood take place in 2 steps : internalization of macromolecules to the blood[35~37]. Results of this study that increasing of antibody titer in neonatal serum was observed around 3 days of age indicates indirectly that the egg yolk antibodies are absorbed and transferred to the circulation. Also, oral administration of CEYA by stomach tube 8 hours after birth may be more effective in raising serum antibodies in the

newborn puppies. Further study is necessary to compare the differences between the intravenous route and PO for the effective absorption of egg yolk antibodies.

In neonates, closure of the intestinal epithelium is defined as the cessation of intake of macromolecules from the intestine to the blood. Various factors are reported to influence the closure of intestinal epithelium of domestic species : dietary management, stress dietary management, time and drugs[38]. Intestinal closure occurs almost entirely by 24 hours of age in dogs[39]. Brambell[40] has shown that the maximal transmission of immunoglobulins takes place at 8 hours after birth and is nearly complete by 15 hours after birth. In present study because protein in the canine milk-replacer results in early intestinal closure treated puppies were administered CEYA within 6 hours after birth, 24hrs and 48hrs, and began to feed the puppies a milk-replacer at 3 days. The rationale was to avoid closure of the intestinal epithelium. Passive immunity may be imparted to an individual by mechanisms other than colostral transfer. Serum also contains protective antibodies and it has been recommended that puppies that do not get colostrum be given adult dog serum as a substitute[41,42,31]. Though administration of immune serum or globulin is effective before newborn and orphaned puppies move into an animal shelter, pet store, or veterinary clinic. My results suggest that egg yolk antibodies are a good alternative of immunoglobulin for passive immunity.

Since the course of CPV infection is rather rapid in young puppies, vaccination is necessary[43]. The disease induced in this study was acute with a sudden onset and a short course. The first signs of dullness, depression and anorexia occurred at day 3-4 after inoculation in placebo group. Severe enteric illness with diarrhea started at day 5 and death were recorded at day 7 and 8. Treated group were clinically normal by days 7 to 8 after inoculation. In severely affected puppies, the time from the onset of illness to death was as short as 48 hours and rapid decline with dehydration and prostration occurred within 8 hours of beginning start of enteric signs. All puppies in control group developed diarrhea which varied from soft feces coated in mucus to watery and hemorrhagic diarrhea.

Clinical signs of CPV infection were anorexia, depression, vomiting and hemorrhagic diarrhea[43]. In this study we could examined all clinical signs by CPV except vomiting in the puppies of placebo group, but could observe the emesis in placebo group. Macartney et al[43] found that vomiting was less consistent feature and there was no evidence of an active gastritis.

The most common reason for the failure of CPV vaccines to induce protection in puppies is the presence of high titer of maternally derived antibodies[44]. As the titer of these antibodies declines, there is a short period during which individual dogs may still be refractory to immunization while, they have become susceptible to infection with field virus[45]. The present study suggests that the use of oral administration of specific antibodies to canine parvovirus could induce a protective response in puppies with maternally derived antibodies and could minimize this critical period.

It has been established that there is a good correlation between the serum hamagglutination inhibition antibody titer and protection from wild canine parvovirus infection. A minimum titer of 64[46] or 80[45] was required for protection against a virulent canine parvovirus challenge. In this study HI antibody titers of all puppies were ranging from 1:40 to 1:80 at birth. After administration of chicken egg yolk antibodies all puppies of treatment groups showed raising of HI titer to 1:160 to 1:1280. These titers were enough to protect against experimental challenge. In control group HI antibody titers were not changed and maintained the same level of HI titer at birth.

Xenogenic immunoglobulin from chicken egg yolks protected puppies from gastroenteritis induced by CPV. The principle of passive immunization of the gut is well illustrated by the role of maternal milk in preventing GI tract infections[47]. The previous studies have suggested that xenogenic immunoglobulin is effective in enterotoxigenic *E. coli* diarrhea in infants with immune bovine colostrum[27]. Hierlmeier[48] has shown that oral administration of egg yolk antibodies reduce the morbidity and mortality rate CPV infection in dog.

Conclusions

In conclusions, chicken eggs are a rich source of heterologous, polyclonal antibody that can be used in passive immunization for the mammalian GI tract. As adverse effects were not observed in puppies, orally administrated egg yolk antibodies can be used for CPV infection in colostrum-deprived puppies and are be useful alternatives for passive immunization. Furthermore, my results have exciting implications for the prevention and possibly treatment of common infectious intestinal diseases in puppies by applying the principle of this study

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