First report of SVC infection in koi carp in Switzerland

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Abstract
In May 2001, the first case of SVC infection in koi carp (Cyprinus carpio koi) in Switzerland was diagnosed. The fish suffered from lethargy, skin ulcers, oedema, enteritis, pericarditis, nephrosis and a secondary bacterial infection. Inoculation of organ material on EPC cell lines resulted in a cytopathic effect (CPE). The virus was identified as SVCV by indirect fluorescence antibody test (IFAT). This SVC infection was an isolated case in Switzerland: No other SVC infection in koi carp has been diagnosed since.

Spring viraemia of carp (SVC) is an acute, systemic, contagious disease. The viral aetiology (rhabdovirus) of the disease was first described by Fijan et al. (1971). Affected fish were diagnosed in many European countries and Russia, but recently also in China (Hoole et al., 2001). The first case of SVC disease in Switzerland was detected in 1980 in common carp (Cyprinus carpio). Spring viraemia of carp may lead to great losses mainly in common carp. However, other cyprinid species like crucian carp, goldfish, bream, tench, barbel, roach, grass carp, silver carp, as well as koi carp are also susceptible (Hoole et al., 2001).

This report described the first case of SVC infection in koi carp in Switzerland which occurred in 2001.

Some fish out of 50 imported koi, held in a quarantine tank at 12-15°C water temperature, stopped feeding shortly after purchase, became lethargic and showed skin ulcers. However no mortality occurred. One fish (21 cm body length) was sent to the National Fish Disease Laboratory for disease diagnosis in May 2001.

This fish underwent a complete necropsy with parasitological, bacterial, histological and virological investigations. For the virological investigations, samples from heart, brain, spleen and kidney were taken, minced in antibiotic solution (10^6 IU penicillin-G, 10 ml amphotericin B, 1g streptomycin sulphate per litre ddH2O) and stored at 4°C overnight. After dilution in phosphate-buffered saline, PBS (1:10), the homogenates were inoculated (1:10 and 1:100 diluted) the following day onto EPC (epithelioma papulosa cyprini) and BF (bluegill fry) cell lines. The inoculated cell cultures were incubated at 15°C and checked daily for cytopathic effects (CPE). After 7 days, subcultures were established from negative cultures, and cultivated for a further 7 days.

For bacteriology, isolation was performed on sheep blood (Bio Mérieux, Genève, Switzerland) and brilliant green agar. Grown gram positive bacteria were identified using the API-20NE system (Bio Mérieux, Genève, Switzerland).

Organ samples were fixed in Bouin’s solution for 24 h and processed for histological investigation according to routine procedures. Samples were stained with haematoxilin-eosin.

Indirect fluorescence antibody test (IFAT) was performed according to the protocol described in European Community Commission Decision 92/532/ECC (anonymous, 1992). Briefly, the cells of the wells showing CPE were scraped off and pooled together after discharging most of the supernatant. One drop of the cell solution was pipetted into each well of a defatted slide holder glass (Standard 25mm x 75mm immunofluorescent printed slides, with 12x5mm-wells, frosted one end, Semadeni AG, Switzerland). Drying at room temperature (2-4h) was followed by fixation with 100% Isopropanol (40-60 min; at 2-8°C) and subsequent re-drying. 10ml of mouse monoclonal antibody against SVCV (BioX, Brussels, Belgium) were pipetted onto the wells with the cell solution and incubated for 2h in a humid chamber at room temperature. 10ml of fluorescein isothiocyanate conjugated anti-mouse IgG (BioX, Brussels, Belgium) was then pipetted on the wells and incubated for 1h at room temperature in a humid chamber, followed by immediate examination under an incident UV light microscope.

The koi carp exhibited distinctly protruding eyes, a slightly swollen abdomen and raised scales. A severe skin ulcer (3 cm in diameter) was located on the left side behind the dorsal fin. The belly was slightly red. On the caudal fin a red spot was present (diameter 0.4 cm). The muscle tissue was slightly oedematous. A small quantity of transparent fluid in the peritoneal cavity was observed. The atrium of the heart was covered with multiple white foci. The appearance of the swim bladder was normal.

Bacteria could be isolated from both the skin ulcer and spleen. These were identified as Aeromonas hydrophila and Citrobacter youngae.

Microscopically, a moderate pericarditis and enteritis was observed. The pericardium was covered with a layer of fat and was infiltrated with mononuclear leukocytes (mainly plasma cells). The myocardium was atrophic. Inflammatory cells were present in the distinctly oedematous submucosa. The mucosa of the intestine was characterised by necrosis and desquamation of the epithelium. The spleen showed hyperaemia. Both the distal tubules and the haematopoietic tissue of the kidney showed distinct necrosis. A high number of tubular epithelial cells showed hyalinic degeneration.

Four days after inoculation of fish cell line cultures for virus detection, a distinct CPE was evident in EPC cell line culture for both dilutions. The virus was identified as SVCV by indirect fluorescence antibody test (IFAT). This result was confirmed by the European Reference Laboratory in Aarhus, Denmark.

The koi carp investigated showed all the clinical signs of a SVC infection (Fijan, 1999, Hoole et al., 2001), with the exception of petechial haemorrhages in the inner organs and the musculature, and a thickening of the swim bladder. With a positive result for SVC in IFAT and the confirmation of the result, this is the first case of SVC disease in koi carp in Switzerland. In Switzerland SVC is of minor importance with regard to carp farming which is almost insignificant in the country. This is revealed by the low number of SVC cases recorded in Switzerland. Since 1980, three cases of SVC in cultured common carp were diagnosed at the National Fish Disease Laboratory.
Surprisingly, there are hardly any published reports of SVCV infections in koi carp. As far as we know, the first isolation of SVCV from koi carp was reported by Hill (1981). He isolated SVCV from an experimentally inoculated koi, thus proving the susceptibility of koi carp to SVCV. The biggest SVC outbreak in the UK happened in 1988, where mass mortalities occurred in carp (Way, 1991). The variety of carp from which SVCV was isolated was not mentioned in this report, however, some of those isolates were from koi carp (pers. com. Dr. K. Way, Weymouth UK). In Germany, SVCV infections in two koi carp were published by Neukirch & Kunz (2001), and three other cases were diagnosed in 1994, 1995 and 1997 (pers. com. Dr. D. Mock, Kirchunden G). Although clinical cases and isolations of SVCV from koi carp are apparently not rare, it seems that those cases are rarely published, assuming everyone was aware that koi carp are susceptible to SVCV. Due to this lack of data, it is impossible to list all the countries where SVCV in koi carp has been isolated to date. However, with regard to the steadily increasing business with koi carp, and as a consequence, the extent exchange of koi carp between countries and cohabitation of fish during koi shows, there is an increasing risk of SVCV disease transmission. This is of particular importance with regard to the sometimes extraordinarily high value of single fish. Therefore, in our opinion a surveillance of diseases in koi carp will continue to increase in importance.

According to the Swiss Federal Animal Health Ordinance, SVC is a notifiable fish disease since 1995 (SR 916.401, Art.5) and must be kept under surveillance. However no measures have to be taken against an SVCV infected population by law, except the announcement of the findings to the veterinary authorities. In agreement with the owner of the infected koi, the other koi carp of the imported lot will be kept in the quarantine pond separated from healthy fish.

References


