Cat-scratch disease and bacillary angiomatosis *

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Summary: Cat-scratch disease (CSD) was first described by Debré in 1950, yet the causative bacterial agent of CSD remained obscure until 1992, when Bartonella (formerly Rochalimaea) henselae was implicated in CSD by serological and microbiological studies. B. henselae had initially been linked to bacillary angiomatosis (BA), a vascular proliferative disease most commonly associated with long-standing human immunodeficiency virus (HIV) infection or other significant immunosuppression. B. henselae has also been associated with bacillary peliosis, relapsing bacteraemia and endocarditis in humans.

Cats are healthy carriers of B. henselae, and can be bacteraemic for months or years. It has recently been demonstrated that B. henselae can be transmitted from cat to cat by the cat flea, but not by direct contact between animals. The author discusses the present state of knowledge on the aetiology, clinical features and epidemiological characteristics of cat-scratch disease and bacillary angiomatosis.

KEYWORDS: Bacillary angiomatosis – Bartonella – Cat – Cat-scratch disease – Human immunodeficiency virus.

INTRODUCTION

Cat-scratch disease (CSD) in humans is typically a benign, subacute, regional lymphadenopathy resulting from dermal inoculation of the causative agent (2). Recent evidence has demonstrated that Bartonella (formerly Rochalimaea) henselae (11), a bacterium that has been isolated from patients with bacillary angiomatosis (BA), is associated with CSD (22, 32, 42, 43, 46, 48, 49, 55). Bacillary angiomatosis is a vascular proliferative disease, which is seen mainly in people who have been infected with human immunodeficiency virus (HIV) (45). Several other clinical manifestations of BA have been reported, such as bacteraemia (48), peliosis hepatis (49, 55), endocarditis (27), neuroretinitis and aseptic meningitis (56). Although cat-scratch

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disease was first described in France by Debré et al. in the 1950s (20), the causative agent remained obscure until 1992, when *B. henselae* was implicated through a serological study (44).

**AETIOLOGY**

The cause of CSD has long been in question. Initially it was considered to be a possible virus, then a gram-negative bacterium. However, it is only in recent years that a specific organism has been identified. In 1983, a small bacillus was identified by Warthin-Starry silver deposition stain on the lymph-node biopsies of thirty-nine patients with CSD (54). In 1988, a pleomorphic, gram-negative bacterium was isolated from the lymph node of a CSD patient at the Armed Forces Institute of Pathology (AFIP) in the United States of America (USA) (23). *Afipia felis* was then considered to be the most probable agent causing CSD. However, isolating the new bacterium was difficult and only a few strains were isolated in a very few laboratories. The serology was not highly specific and was difficult to standardize.

The identification of *B. henselae* as the agent of CSD was an indirect result of the acquired immunodeficiency syndrome (AIDS) epidemic. A new disease known as bacillary angiomatosis (BA), a type of vascular proliferative lesion in immunocompromised hosts (reviewed in 33), was described in HIV-infected patients between 1983 and 1988 (31, 37, 50). It was attributed to a new gram-negative bacillus, which was subsequently named *Rochalimaea henselae* (42, 46, 55). A new, indirect, immunofluorescence antibody test (IFA) was developed in 1992 at the Centers for Disease Control and Prevention to detect antibodies to this organism (44). Using this test, it was noted that 88% of a cohort of CSD patient sera had antibodies to *B. henselae*, compared to only 3% of control patient sera (44). *B. henselae* was first isolated directly from the cutaneous lesions of HIV-infected patients with BA in 1991 (32), and thus this organism has been directly cultured from the lesions of both BA and CSD. BA is also caused by *B. quintana*, the agent that causes trench fever (32). *B. quintana*, however, has never been associated with a case of CSD.

It was only in the early 1990s that evidence clearly indicated that *B. henselae* was more likely to be the agent of CSD than *A. felis*. Bartonella are morphologically very similar to *A. felis* when examined by Warthin-Starry staining, which may explain the previous confusion. Serological studies and isolation of the organism from the lymph nodes of probable CSD cases demonstrated the major role played by *B. henselae* in the aetiology of CSD (7, 19, 21, 22, 40, 44, 51, 57). Furthermore, amplifying an antigen gene of *B. henselae* by polymerase chain reaction (PCR) on CSD skin-test material confirmed the presence of *B. henselae* but not *A. felis* DNA (4); and in July 1992, *B. henselae* bacteraemia was reported in a cat with a healthy owner (43).

A case control study to determine the risk factors associated with developing BA revealed that the only statistically significant risk factor was traumatic contact with a cat (that is, being scratched or bitten) (52). After this study, Koehler et al. identified BA patients who still had cats and performed blood cultures on these animals (34). They found that all seven cats, owned by the four patients, were bacteraemic with *B. henselae*. The researchers further demonstrated that 41% of the pet cats and impounded cats that were tested in the San Francisco Bay area (California) were also bacteraemic.
Humans

According to Jackson et al., there were an estimated 22,000 human cases of CSD in the USA in 1992, some 2,000 of whom were hospitalized (28). The estimated total health cost of CSD is more than US$12 million per year. In Connecticut, which is the only state where cat-scratch disease is a notifiable disease (since January 1992), 246 people met the case definition during the period 1992-1993. A prospective population-based surveillance system discovered that there was an average annual incidence of 3.7 cases of CSD per 100,000 persons (26).

Cat-scratch disease occurs in immunocompetent patients of all ages. Fifty-five to 80% of those patients are under the age of 20. In one study, Jackson et al. reported a higher proportion of cases among children and teenagers than adults, with 45% to 50% of the patients being younger than 15 years old (28). Cat-scratch disease is considered to be the most common cause of chronic, benign adenopathy in children and young adults, and more cases occur in males than in females (28). The incidence of the disease varies by season, with most cases being seen in autumn and winter. Seventy-five percent (184/246) of the cases in Connecticut had developed adenopathy during the period from October to February (26). In the same study, the age-specific attack rate was highest among people of up to ten years of age (9.3/100,000) and decreased as the ages of the patients increased (26). The median age of patients with CSD was fourteen years; the range of ages was from one to sixty-four years. Eleven percent of these patients were hospitalized, but there were no deaths.

In a previous study, also conducted in Connecticut, Zangwill et al. reported that patients with cat-scratch disease are more likely than healthy, cat-owning control subjects to have at least one kitten twelve months of age or younger, to have been scratched or bitten by a kitten and to have at least one kitten with fleas (57). Of forty-five patients, thirty-eight (84%) had antibodies to *B. henselae*, compared to four of the 112 controls (3%). Interestingly, 81% (39/48) of the cats of these CSD patients also had antibodies to *B. henselae*, compared to 38% (11/29) of the control cats.

Several studies have been able directly to associate *B. henselae* bacteraemia in cats, especially in young kittens, with clinical human CSD cases resulting from scratches inflicted by these cats (21, 36). A few human cases of CSD associated with *B. henselae* seropositivity were investigated in the Veterinary Public Health Laboratory of the School of Veterinary Medicine at the University of California, Davis. In one instance, *B. henselae* was isolated from all four cats owned by a veterinarian who had CSD. The youngest cat had the highest level of bacteraemia and was most likely to have caused the human case, as it was the cat most frequently involved in play and scratching (B.B. Chomel, unpublished findings). The veterinarian had an enlarged inguinal lymph node and an antibody titre of 1:256. All four cats were also seropositive.

BA caused by *B. henselae* has been associated mainly with exposure to cats (34, 52), whereas such a risk factor has not been associated with BA caused by *B. quintana*. A recent study also showed a strong link between the onset of neuropsychological decline or dementia in HIV-infected people and serum immunoglobulin M (IgM) antibodies to *B. henselae* (47). Cat ownership was associated with neuropsychological decline and dementia (the odds ratio [OR] = 2.4; 95% confidence interval
The presence of IgM antibodies was also strongly linked to cat ownership (OR = 6.4; 95% CI = 1.3-30.8), particularly if the cat was acquired less than one year before the measurement of antibody values.

Cats

The domestic cat is a major reservoir for the human pathogen, *B. henselae*. Bacteraemia in cats was reported for the first time, in the cat of a healthy owner, by Regnery *et al.* (43). Antibodies had been detected in the cat by serology (IFA) a few weeks earlier. Koehler *et al.* reported that 41% (25/61) of a group of pet and impounded cats from the San Francisco Bay area were bacteraemic (34). Similarly, Chomel *et al.* were able to demonstrate that 39.5% of a convenience sample of 205 cats from northern California were bacteraemic, and that 81% of them were antibody-positive (15). Cats below the age of twelve months and impounded cats were more likely to be bacteraemic, and flea infestation was also a significant risk factor. There was no direct correlation between the level of bacteraemia and the antibody titre; however, cats with serological titres of over 512 were more likely to be bacteraemic than cats with a titre of up to and including 512.

Various serosurveys of cat populations have been conducted in the USA. A serological and epidemiological study of banked cat sera, collected between 1980 and 1985 in Baltimore (Maryland), indicated a *Rochalimaea* antibody prevalence of 14.7% (87/592) (13). In another survey, Childs *et al.* reported a prevalence of 28.2% (370/1,314) from cats in various parts of the USA (14). A sero-epidemiological survey, using 628 samples from throughout North America, identified an overall prevalence of 28% (175/628), ranging from a low of 3.7% to 6.7% in the Midwest and Great Plains regions, to 60% in the South-east (29). A high seroprevalence appeared to be correlated with warm, humid climates. These warm, humid areas with the highest seroprevalence were also reported to have the highest number of potential arthropod vectors, including fleas. In a serosurvey of 518 sick cats from North Carolina, 109 cats, or 21%, were positive to *B. henselae* (9). In Hawaii, of thirty-one kittens involved in human cases of CSD, twenty-one (68%) had positive blood culture and elevated antibody titres to *B. henselae* (21). Only one out of twenty-three adult cats (4%) had a positive culture, but eighteen cats (78%) had elevated antibody titres. In a cluster of CSD encephalopathy cases in south Florida, it was found that 22% of the 124 cats tested were bacteraemic and 62% (77/124) had *B. henselae* antibodies (41).

Information on the prevalence of *B. henselae* in cats from other parts of the world is still limited. In Japan, Ueno *et al.* reported a 15.1% (30/199) prevalence among domestic cats (53). In Australia, Flexman *et al.* reported the first isolation of *B. henselae* from the blood and fleas of a cat belonging to a patient with CSD (24). Three weeks after the patient had removed fleas from his cat, he developed fever, lethargy and anorexia, which lasted for three days, followed by the appearance of axillary lymphadenopathy. There was no history of a bite or a scratch and no primary lesion on the skin. Therefore, this case could also be one of the first confirmed cases of CSD in humans to be transmitted by fleas.

In Europe, Allerberger *et al.* reported a 33% prevalence of antibodies to *B. henselae* in Austrian cats (32/96) (3). In France, Chomel *et al.* isolated *B. henselae* from 11% (7/64) of a convenience sample of cats tested at the teaching hospital at Alfort Veterinary School, between 2 October and 12 October, 1995 (16). Twenty-three of the
sixty-four cats (36%), which had been hospitalized mainly for convenience surgery, had *B. henselae* antibodies.

The way in which CSD is transmitted from cat to human is presumed to be predominantly by cat scratches, but the method of transmission from cat to cat is unknown. Studies were conducted in the Veterinary Public Health Laboratory of the School of Veterinary Medicine at the University of California, Davis, to determine the most likely route of infection in cats which had been experimentally infected. The intradermal route is the most effective (6/7 kittens), when compared to the intravenous route (2/16 cats) or the intraperitoneal route (0/5 cats) (1). When experimentally infected, kittens develop a high bacteraemia within two to three weeks and usually clear the infection within two to three months. In some cases, bacteraemia can last for several months, and relapses of bacteraemia at levels much lower than those of the initial infection can be observed. A naturally infected cat was followed for twenty-four months; and the long-term bacteraemia and stable antibody titres of this animal were documented. The bacteraemia was shown to be cyclical, with the level of bacteraemia fluctuating by as much as 100-fold, and intermittent negative cultures. This suggests that a proportion of infected cats may carry the infection for years. Long-lasting bacteraemia in cats was suggested by Koehler *et al.* (34) and was well demonstrated recently by Kordick *et al.* (36).

In order to clarify the mode of transmission of *B. henselae* from cat to cat, Abbott *et al.* looked at the possible, direct horizontal transmission from cat to cat and the possible vertical transmission from infected queens to their offspring (1). Neither horizontal nor vertical transmissions were observed. Susceptible cats housed in an arthropod-free environment in prolonged intimate contact with infected cats remained non-bacteraemic and seronegative. Only four out of eighteen kittens born to seropositive and bacteraemic queens acquired maternal antibodies after nursing, but these antibodies had disappeared by six weeks of age. None of the eighteen kittens was ever blood-culture positive and all of the kittens remained seronegative after weaning.

Koehler *et al.* (34) suggested that fleas may play a role in the transmission of the infection. Viable *B. henselae* organisms were cultured from cats with *B. henselae* bacteraemia, and the presence of *B. henselae* DNA was demonstrated by PCR in fleas that had been combed from infected cats (34). The author *et al.* studied forty-seven cats from one cattery over a period of twelve months to determine the longitudinal prevalence of feline *B. henselae* bacteraemia, the prevalence of *B. henselae* in the fleas infesting these cats (detected by PCR), and whether *B. henselae* bacilli could be transmitted experimentally from cat to cat via the cat flea (*Ctenocephalides felis*). Bacteraemia was detected in 89% of the cats and *B. henselae* DNA was detected in 29% to 80% of the fleas collected from the cats. Fleas removed from bacteraemic cats were allowed to feed on five specific-pathogen-free (SPF) kittens in a controlled environment. All the experimentally exposed kittens became bacteraemic within two to six weeks (17). These studies demonstrate that transmission of *B. henselae* occurs from cat to cat mainly via an arthropod vector (the cat flea), and that the presence of *B. henselae* in fleas does not correlate to the presence or level of bacteraemia in the infested cat. Cats represent the main reservoir for *B. henselae*, but could also be the reservoir of some other new *Bartonella* species, as has recently been reported (18, 36). However, the frequency of infection by these new species seems to be much lower than it is for *B. henselae*. These findings have important implications for the prevention of *B. henselae* infection in both humans and cats.
CLINICAL SIGNS

Humans

One to three weeks pass between the cat scratch or bite and the appearance of clinical signs of CSD in humans (2). In 50% of cases of CSD, a small skin lesion, often resembling an insect bite, appears at the inoculation site (usually the hand or forearm), and evolves from a papule to a vesicle and partially healed ulcers. These lesions heal within a few days to a few weeks. Lymphadenitis is generally unilateral and commonly appears in the epitrochlear, axillary or cervical lymph nodes (12). In the 1992-1993 Connecticut study (26), it was found that younger CSD patients (less than fifteen years old) were more likely to have cervical adenopathy (relative risk [RR] = 1.5; 95% CI = 1.2-1.9). Older patients (15 years old, or more) were more likely to have inguinal adenopathy (RR = 1.4; 95% CI = 1.1-1.8) and axillary adenopathy (RR = 1.4; 95% CI = 1.1-1.7). The lymphadenopathy develops approximately three weeks after exposure. Swelling of the lymph node is usually painful and persists for several weeks to several months. In 25% of the cases, suppuration occurs. The large majority of patients show signs of systemic infection: fever, chills, malaise, anorexia and/or headaches. In general, the disease is benign and heals spontaneously without sequelae.

Atypical symptoms of CSD occur in 5% to 10% of cases. The most common of these symptoms is Parinaud's oculoglandular syndrome (peri-auricular lymphadenopathy and palpebral conjunctivitis), but meningitis, encephalitis, osteolytic lesions and thrombocytopenic purpura may also occur. Encephalopathy is one of the most serious complications of CSD, usually occurring two to six weeks after the onset of lymphadenopathy. However, patients usually make a complete recovery with a few or no sequelae. A cluster of five cases of children with acute encephalopathy associated with CSD was recently reported in south Florida (41). New clinical presentations associated with B. henselae infection were reported last year in immunocompetent people, such as neuroretinitis, and bacteremia as a cause of chronic fatigue syndrome (56), as well as a case of aggressive B. henselae endocarditis in one cat owner (27).

The symptomatology is rather different for immunocompromised people with BA. BA, also called epitheliod angiomatosis, is a vascular proliferative disease of the skin, characterized by multiple, blood-filled, cystic tumours. This disease is also characterized by violet-coloured or colourless papular and nodular skin lesions, that may clinically suggest Kaposi’s sarcoma, but histologically resemble epitheliod haemangiomas (45). When visceral parenchymal organs are involved, the condition is referred to as bacillary peliosis hepatis, splenic peliosis or systemic BA. Patients with disseminated BA may develop fever, weight loss, malaise and enlargement of the affected organs. Endocarditis has also been reported (27).

Cats

No clinical signs of CSD have been reported in cats under natural conditions. Suspicions of lymphadenopathy, caused by a CSD-like organism identified by silver-stained section, have been reported (30).

Breitschwerdt and Kordick (9) reported a self-limiting, febrile illness, lasting forty-eight to seventy-two hours, and transient neurological dysfunction in two cats experimentally infected with B. henselae by blood transfusion (9). No such clinical
manifestations were observed in any of the experiments conducted by the author and colleagues, but these studies did not use blood transfusions to infect cats with *B. henselae*.

Cat-scratch disease infection is very common in cats, especially in young kittens. Bacteraemia usually lasts from a few weeks to a few months. The organisms have been reported to be intra-erythrocytic (35), and pili may be a pathogenic determinant for *Bartonella* species (8). Cats can yield more than one million colony-forming units per ml of blood (17).

**Dogs**

Surveys have been conducted among dogs in California (B.B. Chomel, unpublished findings), and in Hawaii (21). None of the dogs was found to be bacteraemic with *B. henselae*, and only a very small percentage of the dogs tested in Hawaii (6.4% or 2/31) were found to be seroconverted (21). When the dogs were experimentally inoculated with *B. henselae* intradermally, they did not become bacteraemic, although they seroconverted after a few weeks (B.B. Chomel, unpublished findings). Breitschwerdt et al. have reported the isolation of a new subspecies of *Bartonella* in the case of a dog with endocarditis (10).

**DIAGNOSIS**

For years, the diagnosis of cat-scratch disease was based on clinical criteria, exposure to a cat, failure to isolate other bacteria, and/or histological examination of biopsies of lymph nodes. A skin test was also developed. However, the antigen prepared from pasteurized exudate from the lymph nodes of patients with CSD was not standardized, and concerns were raised about the safety of such a product. In the past few years, serological tests, such as the IFA (19, 43) and enzyme-linked immunosorbent assay (ELISA) (5, 7, 51), and techniques to isolate the organism from human and cat specimens have been developed. Because *B. henselae* is an intra-erythrocytic bacterium (35), cell lysis, using a lysis centrifugation technique, greatly aids in isolating bacteria from the blood. For blood culture from cats, 1.5 ml of blood is drawn into lysis-centrifugation tubes (34). The tubes are centrifuged and the pellet spread onto heart infusion agar plates containing 5% fresh rabbit blood. These plates are maintained at 35°C in a high humidity chamber with 5% CO₂ for three or four weeks. Colonies will usually develop in a few days from cat blood, although some strains may require a few weeks. The identification of isolates as *B. henselae* is confirmed by DNA amplification (6), using PCR-restriction fragment length polymorphism (PCR-RFLP) analysis. Two different restriction endonucleases, *TaqI* and *Hhal*, should be used to digest the single product amplified using primers previously described (42).

**TREATMENT**

**Humans**

Antimicrobial treatment is generally indicated for patients with BA, bacillary peliosis or relapsing bacteraemia. Treatment with erythromycin, rifampin, or
doxycycline for at least two to three months is recommended for immunocompromised people (45), but relapses can occur.

For CSD, antimicrobial treatment is not generally indicated, as most typical cases do not respond to antimicrobial administration (38). Intravenous administration of gentamicin and doxycycline, and oral administration of erythromycin, have been successfully used in the treatment of disseminated CSD (25, 39). According to Wong et al., therapy with doxycycline and rifampin appears to be helpful in the treatment of patients with neuroretinitis (56).

**Cats**

According to Koehler, antibiotic treatment (25 to 50 mg of doxycycline twice daily; 100 mg of lincomycin twice a day for three weeks) could suppress bacteraemia in cats (34). The author’s own experience shows that various antibiotics (doxycycline, erythromycin and enrofloxacin) may reduce the level of bacteraemia in experimentally infected cats, but do not totally suppress bacteraemia. A few weeks after the end of treatment, this suppressed level of bacteraemia may rise again, often surpassing the initial level of bacteraemia in the cat (B.B. Chomel, unpublished findings).

**PREVENTION**

A large reservoir for *B. henselae* exists among the 57 million pet cats living in one-third of the homes in the USA (34). As the possibility of *B. henselae* infection becomes more widely recognized, there is likely to be negative publicity about the possible hazards of cat ownership, especially for immunocompromised people. However, cats can be very comforting to the chronically and terminally ill.

The findings of this study clearly indicate that seronegative cats are likely not to be bacteraemic, but that young kittens, especially impounded and flea-infested kittens, are more likely to be bacteraemic. Therefore, the author suggests that people who want to acquire a pet cat, especially those who are immunocompromised, should obtain a cat raised in a cattery. If possible, the cat should be an adult and should come from a flea-controlled environment. Cats could be serologically tested so that prospective owners could adopt only a seronegative animal. Unfortunately, there is no correlation between seropositivity and bacteraemia. Bacteraemia can also be transient, with relapses.

Declawing the cats has also been suggested, but this would have limited value, as fleas can transmit the infection from cat to cat. Therefore, flea control appears to be one of the major measures needed to prevent cats from becoming infected and *B. henselae* being spread from cat to cat.

The most effective means of preventing infection by *B. henselae* are common sense, hygiene and, possibly, changing the behaviour of cat-owners themselves. People should wash their hands after handling pets, and clean any cuts, bites or scratches promptly with soap and water.

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MALADIE DES GRIFFES DU CHAT ET ANGIOMATOSE BACILLAIRE. – B.B. Chomel.

Résumé : La maladie des griffes du chat fut décrite cliniquement en 1950 par Debré, mais son étiologie est restée obscure jusqu’en 1992, date à laquelle les méthodes sérologiques et la biologie moléculaire ont permis d’incriminer une nouvelle bactérie, Bartonella (connue auparavant comme Rochalimaea) henselae, comme l’agent de la maladie des griffes du chat. B. henselae avait déjà été reconnue comme l’agent de l’angiomatose bacillaire, une maladie vasculo-proliférative sévissant principalement chez les sujets infectés par le virus de l’immunodéficience humaine ou atteints d’autres troubles immunodéficitaires majeurs. B. henselae est également responsable de la purpura hépatique ainsi que de formes récidivantes de bactériémie et d’endocardite chez l’homme.

Les chats sont porteurs sains de l’agent infectieux et peuvent présenter une bactériémie pendant des mois voire des années. L’infection se transmet de chat à chat sans contact direct, essentiellement par l’intermédiaire des puces. Les connaissances actuelles concernant l’étiologie, les manifestations cliniques et l’épidémiologie de la maladie des griffes du chat et de l’angiomatose bacillaire sont présentées.


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FIEBRE DE RASGUÑO DEL GATO Y ANGIOMATOSIS BACILAR. – B.B. Chomel.

Resumen: La fiebre de rasguño del gato (cat-scratch disease) fue descrita por primera vez por Debré en 1950. El agente bacteriano que la causaba, sin embargo, permaneció en la sombra hasta 1992, cuando estudios serológicos y microbiológicos establecieron un nexo entre Bartonella (anteriormente Rochalimaea) henselae y esta enfermedad. En un principio, B. henselae había sido relacionado con la angiomatosis bacilar, una enfermedad vascular proliferante ligada en general a la infección prolongada por el virus de la inmunodeficiencia humana u otras importantes inmunosupresiones. B. henselae también ha sido relacionado con la púrpura hepática, bacteriemia recurrente y endocarditis en el hombre.

El gato es un portador sano de B. henselae, el cual puede permanecer presente en su sangre durante meses o años. Recientemente se ha demostrado que B. henselae puede transmitirse de gato a gato mediante la pulga del gato,
mientras que no se transmite por contacto directo entre los animales. El autor examina el estado actual de los conocimientos sobre la etiología, los rasgos clínicos y las características epidemiológicas de la fiebre de rasguño del gato y de la angiomatosis bacilar.


REFERENCES


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