

Cannabis Arteritis: Review of the Literature

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Abstract: Consumption of cannabis in young adults has continued to increase in recent years. Cannabis arteritis was first described in the 1960s, but the number of cases has continued to increase. We reviewed current knowledge of the different types of cannabis arteritis in young adults and found 70 cases of cannabis arteritis in the literature. We discuss physiopathological arguments in favor of cannabis vascular toxicity per se, although we did not find sufficient evidence to identify cannabis arteritis as a specific diagnostic entity. Many factors suggest a link between cannabis consumption and arteritis in young adults, but it is difficult to say whether this type of arteritis is similar to thromboangiitis obliterans. We were unable to demonstrate a formal association between cannabis smoking and the development of thromboangiitis obliterans, because most case reports showed associated tobacco smoking (97%) and the number of years cannabis had been smoked by the participants was mostly unknown. Cannabis consumption would however seem to be an aggravating factor, together with tobacco, in arteritis, which occurs in young adults.

Key words: juvenile arteritis, cannabis, tobacco, drug abuse, Buerger disease, thromboangiitis obliterans, vascular ischemia

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Cannabis is the most common illegal substance consumed in France. In 2007, the French Monitoring Centre for Drugs and Drug Addiction estimated that 31% of adults aged between 16 and 64 years had tried it (including 9% who had tried it during the year and 1.4% who were regular users).¹ Cannabis is most often consumed by inhalation and is strongly linked to tobacco and alcohol consumption.^{2,3}

Δ 9-Transetrahydrocannabinol (Δ 9-THC) is considered to be the main active agent in cannabis and is responsible for its principal effects on the body. Acute psychiatric disorders

induced by cannabis are mostly acute psychotic disorders, delirium, anxiety, and depressive symptoms. When addiction is present, an amotivational syndrome is frequently observed (reduction in social activity, asthenia, depressive mood, and cognitive disorders).⁴

Somatic disorders are mostly cardiovascular and bronchopulmonary.^{5–7} The cardiovascular effects, which have been studied, have mostly focused on Δ 9-THC rather than on combustion products, on other cannabinoids, and on associated toxic substances.⁶ The mechanisms of action on the cardiovascular system are complex, and different actions exist.⁸ However, in general, the cannabinoid system opposes the autonomic nervous system producing paradoxical vasoconstriction^{8,9}; a drop in cardiac output and hypoxia and an increase in carboxyhaemoglobin¹⁰; an increased risk of infarction in coronary patients¹¹; an arrhythmogenic effect⁸; a cerebrovascular adverse event in young adults¹²; and orthostatic hypotension.⁸

The other constituents of cannabis smoke are also toxic to blood vessels. During combustion, 15% to 60% of Δ 9-THC is absorbed,¹³ and cannabis smoke also contains most of the same toxic substances as that present in tobacco smoke.¹⁴

Δ 9-THC seems to have a direct toxic effect on blood vessels. On the one hand, there seems to be an in vitro oxidizing effect causing endothelial lesions,¹⁵ as activation of the cannabinoid receptors inhibits nitric oxide synthase,¹⁶ leading to an impaired vascular relaxation. On the other hand, a vasoconstriction effect has been shown in rats after intravenous administration of Δ 9-THC.¹⁷

Arteriopathy of the lower limbs in adults younger than 50 years accounts for 1% to 7% of cases of arteriopathy of the lower limbs. Cannabis is one of the most frequent causes of arteriopathy in young adults. In their study, Sauvanier et al¹⁸ found that 70% of cases of arteritis were due to atheroma and 20% due to Buerger disease (thromboangiitis obliterans [TAO]). Cannabis consumption was more frequent in the patients with TAO (21% vs 8%), and their Leriche stage was more advanced when the medical treatment was started (atheroma 57% stage II vs TAO 86% stage IV) (Rutherford classification's grade II and III = Leriche & Fontaine classification's stage III and IV). They also diagnosed TAO more frequently in cannabis consumers (43%) than nonconsumers (19%),¹⁸ suggesting a link between cannabis use and TAO.

Thus, although the main etiology of arteritis in young adults is atherosclerosis, cannabis consumers account for 10% of those patients, who smoke less tobacco, who have more distal

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disease, and whose upper limbs are more often affected. Venous thrombosis of a superficial vein is occasionally seen.¹⁹ This clinical picture is quite similar to TAO, suggesting that cannabis arteritis may be a specific form of TAO.

The TAO is a nonatheromatous form of distal arteritis, which is a segmental multifocal inflammatory disease of the small- and medium-sized arteries and of the superficial veins. The reported incidence of TAO is 8 to 12.6/100,000 population in North America.²⁰ The TAO classically affects young male adults (younger than 40 years), and there is a very strong link with tobacco consumption.²¹ Although TAO existence was questioned in the 1960s, many publications have since confirmed TAO as a distinct clinical entity.^{22,23} No biochemical markers help in making a positive diagnosis of TAO.²¹ Many pathogenic hypotheses exist, although the etiology is still unknown, but tobacco, an autoimmune origin (hypersensitivity to tobacco antigens, increased reactivity of white blood cells) and a genetic predisposition²⁴ have been incriminated.

In view of the widespread consumption of cannabis in Europe,²⁵ its cardiovascular complications,²⁶ and the increasing number of cases of cannabis-induced arteritis being reported, we decided to review the cases of cannabis-induced arteritis described in the literature, with the aim of looking for specific criteria that may show a possible link with TAO.

METHODS

We reviewed the literature using several databases: Medline, Embase, Google Scholar, and the Catalogue of Theses from the French Interuniversity Library of Medicine.

We found 70 cases of cannabis arteritis in the literature (Table 1). We classified them according to age, sex, quantity and duration of cannabis consumption, consumption of tobacco and other drugs, how the disease was discovered, its clinical presentation, imaging, treatment, and outcomes.

Because the case reports mostly fitted Olin's criteria for TAO or Buerger disease,²⁷ we decided to compare our collected data (Table 1) with the results of two large series of patients suffering from TAO: the first with 112 patients²⁸ and the other with 110 patients²⁹ (Table 2).

RESULTS

The mean age of the patients was estimated as 28.5 years (SD 8.25, min 18, max 48), which is comparable to patients with TAO whose mean age was 35 years.²⁹ As in TAO, there was a large male predominance (92.8%). The mean cannabis consumption was estimated at 3.8 joints per person per day, with duration between 1 and 20 years. Because cannabis is always consumed mixed with tobacco, we were unable to isolate the effects of cannabis by itself. However, we noted that whenever it was looked for, coaddiction with tobacco was (apart from two cases) always reported. The quantity of tobacco was not always specified, and the methods used to quantify it varied considerably from one study to the other (pack-years, cigarettes per day, or length of tobacco consumption).

Other coaddictions were uncommon. Only nine patients consumed other drugs, either alone or in association (amphetamines, heroin, lysergic acid diethylamide (LSD), or high-dose Buprenorphine), some of which also have cardiovascular toxicity (amphetamines).³⁰

From a clinical point of view, although risk factors were not systematically specified, we found that 11 patients had a previous cardiovascular history. Raynaud's syndrome was noted in seven of the 70 cases (10%), which is much lower than rates found in TAO (44% in the study by Olin et al²⁸), but this data were available in only 18 cases, estimating the rate at 38.8% (7/18 cases). Venous thrombosis was found in two of the 70 cases (2.85%). It was superficial, single, in a lower limb, and without any hemostatic abnormalities, the latter being frequent in TAO (38%).²⁸ Finally, arterial lesions affected the lower limbs twice as often as the upper limbs, the lesions were distal in all of the cases, and an associated proximal lesion was present in 18.8% of the cases (figures that are quite close to those from the study by Olin et al).

When imaging was performed, (arteriography and/or Doppler ultrasound), comparable lesions (thin arteries, many obliterations, segmental occlusive lesions, and the presence of collateral circulation) were seen, which were suggestive (as in TAO) of inflammatory nonatheromatous arteritis.

Histology from amputation specimens or cutaneous biopsies was rarely performed. Only Sterne and Ducastaing³¹ found fatty deposits without any inflammatory infiltrate, suggestive of atherosclerosis, whereas Disdier et al³² described a patient with endarteritis and an inflammatory infiltrate composed of neutrophils and macrophages, which had invaded the media and had fragmented the internal elastic lamina, with *in situ* thrombosis. Other authors believe that these two types of lesion are identical at different stages of the disease process.³³

Biochemical tests, performed to look for thrombotic cardiovascular risk factors, only found six cases of moderate hyperhomocysteinaemia, one case of S-protein deficiency, two cases of hypercholesterolaemia, three cases of hypertriglyceridaemia, 10 cases with an inflammatory syndrome, and no cases of diabetes.

Treatments given varied greatly from one team to another and was mostly symptomatic. The only consensus seems to be that cannabis should be stopped. In fact, stopping cannabis proved an essential element in the favorable outcome in 18 patients treated by cannabis withdrawal, in determining the clinical course of 13 other patients, which depended on whether they stopped or restarted cannabis consumption, and in the clinical worsening in three cases, who continued their consumption.

It is, however, noteworthy that one case improved despite not stopping cannabis and that two cases improved after cannabis withdrawal despite continuing tobacco smoking.

The amputation rate was lower than that in the study by Olin et al (14.5% vs 27%), but because the data were incomplete, it is difficult to compare them.

In the 70 case reports, cannabis arteritis mostly affected young men (18 to 40 years), whose cannabis consumption varied from abuse to addiction, with almost systematic to-

TABLE 1. Description of the Population

Authors	Age	Gender	Cannabis Quantity	Duration (yr)	Concomitant Tobacco Smoking	Other Toxic	Other Cardiovascular Risk Factor	Other Procoagulation Systemic Diseases	Clinical Discovery	Imagery
Sterne and Ducastaing ³¹	25–35	Male (29)	15 pipes/d	Unknown	Unknown	Unknown	No	No	Claudication, toe and heel necrosis	Stenosis, collateral circulation
Swiader et al ⁴²	18	Male (1)	1 cig/d	1	Yes (<1 PY)	Unknown	Unknown	Unknown	Foot ischemia	Unknown
Swiader et al ⁴²	20	Male (1)	5–10 cig/wk	3	Yes (6 PY)	Unknown	Unknown	Unknown	Necrosis of 2 fingers	Occlusion
Constans et al ⁴³	30	Male (1)	4–5 cig/wk	15	Yes (12 PY)	Ecstasy	Unknown	Unknown	Ischemia of 2 fingers	Occlusion
Constans et al ⁴³	29	Male (1)	Unknown	Unknown	Yes (10 PY)	Ecstasy	Unknown	Unknown	Ischemia	Occlusion and stenosis
Constans et al ⁴³	40	Male (1)	Unknown	20	Unknown	No	Unknown	Unknown	Distal necrosis	Occlusion and stenosis
Constans et al ⁴³	38	Male (1)	Unknown	18	Yes (20 PY)	Heroin, Buprenorphine	Unknown	Unknown	Distal necrosis	Stenosis
Schneider et al ⁴⁴	38	Male (1)	1 cig/d	Unknown	Yes (stop smoking since 10 yr)	No	No	No	Distal necrosis	Occlusion, collateral circulation
Le Berre et al ⁴⁵	20	Male (1)	Unknown	Unknown	Yes (5 PY)	Unknown	Unknown	Unknown	Lower limb ischemia	Occlusion and stenosis
Michon-Pasturel et al ⁴⁶	21	Male (1)	Unknown	Unknown	Yes (<4 PY)	No	Unknown	Unknown	Lower limb ischemia	Unknown
Michon-Pasturel et al ⁴⁶	22	Male (1)	Unknown	Unknown	Yes (<4 PY)	No	Unknown	Unknown	Lower limb ischemia	Unknown
Michon-Pasturel et al ⁴⁶	26	Male (1)	Unknown	Unknown	Yes (<4 PY)	No	Unknown	Unknown	Lower limb ischemia	Unknown
Schneider et al ⁴⁷	30	Female (1)	1 cig/d	Unknown	Yes (16 yr)	No	Hypertriglyceridemia	No	Claudication	Stenosis
Disdier et al ³²	18–40	Male (10)	1–5 cig/d	1–20	Yes (<20 cig/d) (1 to 20 yr)	Amphetamin, Heroin, Buprenorphine, LSD	Diabetes (1)	Homocysteine level slightly elevated (3) Raynaud (2)	Necrosis, ischemia	Stenosis, collateral circulation
Cazalets et al ⁴⁸	22	Male (1)	1 cig/wk	5	Yes (5 PY)	No	No	No	Claudication, toe necrosis	Occlusion
Cazalets et al ⁴⁸	20	Male (1)	4 cig/d	4	Yes (4 PY)	No	No	Raynaud, Heterozygote mutation of factor II	Claudication, toe necrosis	Occlusion
Cazalets et al ⁴⁸	35	Male (1)	1 cig/d	10	Yes (10 PY)	No	No	Raynaud	Claudication, toe necrosis	Stenosis
Cazalets et al ⁴⁸	30	Male (1)	5 cig/d	12	Yes (15 PY)	No	No	Raynaud	Fingers' pulp necrosis	Stenosis
Sauvanier et al ¹⁸	28 (25–31)	Male (6); Female (1)	Unknown	Unknown	Yes (18 PY)	No	hypercholesterolemia (1) hypertriglyceridemia (2) diabetes (1)	No	Ischemia	Unknown
Gröger et al ⁴⁹	24	Female (1)	1–2 cig/d	6	Yes (5 cig/d)	No	No	Raynaud	Digital necrosis	Stenosis, collateral circulation
Karila et al ⁵⁰	18	Male (1)	6–10 cig/d	Unknown	Yes (moderate)	No	No	No	Claudication	Occlusion, collateral circulation
Ducasse et al ⁵¹	19	Male (1)	10 cig/d	4	Yes (5 cig/d)	No	No	No	Toe necrosis	Occlusion
Piquet et al ⁵²	32	Female (1)	10 cig/d	2	Yes (12 PY)	No	No	No	Fingers pain, pulpar phlycten	Occlusion
Combemale et al ³³	38	Male (1)	3–5 cig/d	20	Yes (20 cig/d)	No	No	No	Toe necrosis, painful lump on lower limb	Stenosis
Peyrot et al ³⁴	36	Male (1)	10 cig/d	20	No	No	No	No	Claudication	Stenosis
Ghfour et al ⁵³	26	Male (1)	6 cig/wk	3	Yes (6 PY)	No	No	Raynaud	Claudication, pain, plantar ulcerations, toes necrosis and amputations	Occlusion and stenosis
Noel et al ¹⁹	48	Female (1)	5 cig/d	20	No	No	No	No	Toe necrosis	Occlusion

PY, pack-years; cig, cigarettes.

TABLE 2. Comparison of Cannabis Arteritis (Case Reports) and TAO (Population Study)^{28,29}

	Cannabis Arteritis (70 Case Reports)	Olin et al ²⁸ (112 Patients)	Ohta et al ²⁹ (110 Patients)
Mean age (yr)	28.5	42	35 (17–49) age of onset
Male sex, no. (%)	65 (92.8%)	86 (77%)	106 (96%)
Cannabis dose, mean	3.8 cig/person/d	0	0
Cannabis mean duration	1–20 yr	0	0
Tobacco, no (%)	68 (97%)	112 (100%); 48% stopped smoking	110 (100%)
Amputation	14.5%	27%	43%

bacco coaddiction (in addition to the tobacco-cannabis mixture used for making joints). No other cardiovascular risk factors were noted, and other coaddictions were not thought to be linked to the arteritis. The lesions were severe, and their histology atheromatous and their imaging were compatible with descriptions of TAO. The prognosis was directly linked to cannabis consumption (sometimes leading to amputation, if cannabis was not stopped).

DISCUSSION

The objective of this study was to collect cases of arteriopathy linked to cannabis and to try to separate out the particular features offered in the initial description from 1960.³¹ So far, we have found 70 cases in the literature. This figure is probably an underestimate because of associated tobacco consumption, which is the main factor for confusion between the various cardiovascular risk factors. Peyrot et al³⁴ actually found only 55 cases using different data collection methods than our own methods.

An initial remark is that most of the authors are French speakers. Apart from the fact that consumption has considerably increased during the past 10 years in France, possible sources of bias may be from a greater genetic susceptibility in our population or from French-speaking vascular doctors being more aware of this phenomenon than in other countries. Finally, since the initial description, more than half of the cases were reported during the past 5 years. Apart from the fact that access to publication in medical journals has increased, we consider that this increase also reflects changes in addictive behavior with respect to cannabis, because 50% of 17-year-old adolescents have already tried it.²

A second point is that our results are from case reports collected from the literature and are not from a population-based study. In those cases, many factors are in favor of a link between cannabis consumption and arteritis in young adults, but it is important to note that most cannabis users also belong to that age group. It is difficult to say if this type of arteritis is similar to atheromatous disease, to TAO, or is a new pathologic entity. In addition, consumption was very difficult to estimate, in view of the different methods of consumption and the different methods of data collection used.

From these cases, we observed characteristics in favor of both atheroma and TAO—the main aetiologies of arteriopathy in young adults. In fact, all sizes of artery were affected in the cases seen, with segmental or diffuse lesions, although the histology was similar to that of atheroma, and in certain cases, blood tests showed an inflammatory syndrome

to be present. In addition, the almost systematic association of cannabis and tobacco consumption (a major risk factor for arteritis) makes it impossible to state categorically that cannabis was the only substance causing blood vessel damage. Sixty-eight subjects (97%) also smoked tobacco in our review. Because most cases of TAO reported in the literature included associated tobacco smoking, and because the length of time cannabis had been smoked is unknown, it is not possible for us to make a formal link between cannabis smoking and the development of TAO. However, we suspect that the vasoconstrictive effect of cannabis may act as an aggravating factor, when it is associated with tobacco, which would seem to have been confirmed by the cases in which the symptoms varied according to the patients' cannabis consumption.

Finally, other factors make it difficult to prove that cannabis is involved in causing arteriopathy:

- the composition of the inhaled smoke (carbon monoxide, free radicals), which may cause endothelial lesions by itself,
- consumption of other drugs, apart from cocaine, which is suspected as having a direct vascular toxic effect, although this still has to be confirmed,
- the existence of other biochemical risk factors for thrombosis.

Analysis of data from the literature poses a certain number of problems: the lack of data and the variability of diagnostic methods. Other means, such as a prospective case-control study, are required before wider conclusions can be drawn.

We can only conclude that there is link between cannabis consumption and certain cases of arteritis in young adults, but we are currently unable to identify cannabis-induced arteritis as a separate pathologic entity. The TAO is itself a clinical diagnosis by exclusion, without any clear laboratory or histologic parameters to prove it. Apart from cannabis, comparable clinical pictures of vascular pathology have been described for amphetamines, cocaine, and arsenic.^{30,35–37} It is generally accepted that cannabis, like other drugs such as amphetamines or cocaine, may have an aggravating or synergistic effect with tobacco.

According to Noel et al,¹⁹ cannabis arteritis and TAO have almost the same clinical and arteriographical presentation. Chronic arsenic exposure causes dysfunction of endogenous nitric oxide, which may lead to vascular occlusion.³⁸ Epidemiological studies have found that consumption of

home-made cigarettes is associated with an increased risk of TAO.^{39,40} High arsenic content can be found in cannabis and home-made cigarettes, exposing consumers to chronic intoxication and vascular complications.⁴¹

In conclusion, cannabis seems to be a promoting factor for the arteritis disease process, perhaps in synergy with tobacco, although apparently not the only triggering factor.

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