Percutaneous Conchotome Muscle Biopsy. A Useful Diagnostic and Assessment Tool

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ABSTRACT. Objective. To evaluate the diagnostic yield, performance simplicity, and safety of the percutaneous conchotome muscle biopsy technique for clinical and research purposes in an outpatient rheumatology clinic.

Methods. Biopsies taken by rheumatologists in an outpatient clinic during 1996 and 1997 were evaluated for histopathological and clinical diagnoses.

Results. A total of 149 biopsies were performed on 122 patients. Physicians learned the method easily. Samples were of adequate size and quality to allow for diagnostics. In total 106 biopsies were taken due to different diagnostic suspicions: 24 polymyositis (PM) or dermatomyositis (DM); 43 PM, DM, or vasculitis in addition to another rheumatic condition; 19 systemic vasculitis; and 20 myalgias. Criteria for definite or probable PM/DM were fulfilled in 21 patients, 18 with positive biopsies. Thirteen patients received vasculitis as clinical diagnosis, 3 with positive biopsies. No patient with myalgia had a biopsy with inflammatory changes. Fifteen of 43 rebiopsies performed to assess disease activity had signs of active inflammation. In 48% there were changes in immunosuppressive therapy after biopsy results. Four complications occurred; one was a serious subfascial hematoma.

Conclusion. The percutaneous conchotome muscle biopsy technique gives a good size sample that allows for diagnostic evaluation and has a high yield in patients with myositis. It is a simple procedure, easy to learn and to perform, with a low complication rate and minimum discomfort for the patient. The method can preferably be used as a diagnostic tool and to perform repeated biopsies to assess the effect of a given therapy for both clinical and research purposes. (J Rheumatol 2001;28:1591–9)

Key Indexing Terms: MUSCLE BIOPSY MUSCULAR DISEASES POLYMYOSITIS DERMATOMYOSITIS
quality to allow required diagnostic procedures such as histopathology and biochemistry analyses. It has also been questioned whether the sample size with this method is large enough to provide a high diagnostic yield, especially in myositis diagnostics, since patchy distribution of the inflammatory infiltrates and skip lesions has been reported14-16. The open muscle biopsy technique, which results in larger biopsy samples, has often been advocated to increase the diagnostic sensitivity for this patient group17. Still, in most studies, roughly 25–30% of patients with myositis have negative biopsies8,10,18-20. Moreover, the safety of the conchotome biopsy technique as a routine diagnostic tool has not been evaluated in a rheumatology setting.

We evaluated the utility and safety of the percutaneous conchotome muscle biopsy technique performed in a rheumatology clinic. The majority of the investigated patients were suspected of having an inflammatory muscle disease. Due to the easily available method, a large number of biopsies have been taken from patients with myalgia without other signs of muscle disease, thus presenting a vague suspicion of myopathy. We also evaluated the diagnostic outcome of these biopsies. We assessed the utility and safety of the technique as a tool for repeated biopsies. The patients who were subject to these rebiopsies had previously been diagnosed with IIM. These biopsies were performed due to the persisting muscle symptoms of weakness, fatigue, and/or pain.

**MATERIALS AND METHODS**

We completed a retrospective evaluation of all muscle biopsies performed during 1996 and 1997 at the Department of Rheumatology, Karolinska Hospital, Stockholm. The percutaneous conchotome muscle biopsy technique was used in all cases. Medical records and pathology reports were reviewed. The following information was registered for all 149 biopsies: muscle selected for biopsy site, performer of biopsy, clinically suspected diagnosis before biopsy and clinical diagnosis after biopsy, the histopathological evaluation and diagnosis, change of treatment, adequate muscle tissue size, and complications. Creatine phosphokinase (CPK) levels (normal values: men, 38–174 units/l; women, 26–140 units/l), electromyography (EMG) results, cortisone dose, and clinical symptoms at time of biopsy were also registered. In the histopathology report, the presence of the following pathological variables was noted: inflammatory cellular infiltrate, type 2 muscle fiber atrophy, degenerating or regenerating muscle fibers, centrally located nuclei, ring fibers, signs of neuropathy, fatty tissue deposits in muscle fibers, ragged red fibers, endothelial involvement (perivascular inflammation and/or inflammation in blood vessel walls), and positive immunohistochemical analyses.

For a clinical diagnosis of myositis the criteria proposed by Bohan and Peter were used11,22 and patients fulfilling the criteria for definite or probable PM or DM were classified as having myositis. For clinical diagnosis of vasculitis we used the 1990 classification criteria of the American College of Rheumatology23. The following definitions of certain muscle biopsy findings were used: (1) myositis was defined as in Bohan and Peter’s definition of the histopathological findings in myositis; (2) vasculitis was defined as inflammation in blood vessel walls and endothelial involvement; (3) “unspecific findings” were used for muscle biopsies that displayed some pathological variables, but not enough for a specific histopathological diagnosis; and (4) normal biopsies were without evident pathological variables.

**Muscle biopsy procedure.** The biopsy procedure was performed in the outpatient clinic or, in some cases, in the intensive care unit by a rheumatologist assisted by a nurse. Under local anesthesia, a 5 to 10 millimeter incision of the skin and muscle fascia was carried out with a scalpel. Two to 4 samples of muscle tissue were removed using Weil-Blakesly’s conchotome (article no. 72-0222, Stille Surgical AB, Stockholm, Sweden) (Figure 1). The closed jaw of the conchotome is inserted into the muscle. To remove the sample, the jaw is opened, closed, and then rotated 180°, thus cutting off tissue. The conchotome’s sharp-edged jaw (3.6 mm width, 8 mm length) is opened and closed similarly to scissors. A steristrip was applied to close the incision, and the patient was mobile immediately after the procedure. The samples were transported on a moist tissue kept cool in a jar on ice to the Neuropathology Laboratory at Huddinge Hospital. This transport takes about 30 minutes. After orientation of the material using a stereo microscope, it was snap frozen in isopentane and dry ice and kept in a freezer (–20°C) until further preparation. Six to 8 µm thick sections were cut in a cryostat. The routine analyses included staining for hematoxylin-eosin, Gomori trichrome, periodic acid-Schiff (PAS), oil red, NADH (the reduced form of nicotinamide-adenine dinucleotide), myos phosphorylase, acid phosphatase, and adenosine triphosphatase (ATPase) at 3 different pH levels (Figure 2). Immunohistochemical stainings for major histocompatibility complex class I (MHC I) antigens and CD3+, CD4+, CD8+ positive cells were performed by demand only on exceptional patients. All biopsies were analyzed by the same neuropathologist (IN).

![Figure 1. A. The Weil-Blakesly conchotome. B. Removal of muscle biopsy from m. vastus lateralis using the conchotome.](https://www.jrheum.org/content/28/7/1592/F1.png)
Figure 2. Histopathological specimens of muscle tissue with different stainings. A. Muscle section with hematoxylin-eosin stain from a patient with inclusion body myositis. There is variation in fiber size, and rimmed vacuoles are present in many fibers (magnification ×262.5). B. Muscle section with cytochrome C oxidase stain from a patient with unspecific myopathic changes. A few cytochrome C oxidase negative fibers are visible (×131). C. Muscle section with acid phosphatase stain from a patient with polymyositis. Acid phosphatase positive cells are attacking a nondegenerating muscle fiber (×525). D. Muscle section with Gomori-trichrome stain from a patient with inclusion body myositis showing a ragged red fiber (×525). E. Muscle section with hematoxylin-eosin stain from a patient with vasculitis shows inflammation of a vessel in the perimysium (×258).
RESULTS

A total number of 149 biopsies were performed on 122 patients, 73 during 1996 and 76 during 1997. Sixty-three patients were subject to muscle biopsies in 1996 and 67 in 1997. Among these, 102 patients were subject to one biopsy each, whereas the remaining 20 had 2 to 5 biopsies performed. Ten of these patients participated in a study that assessed the effect of corticosteroids on inflammation in muscle tissue. Eighty-five (70%) of the patients were women and 37 (30%) were men. The median age of the patients was 56 years (range 16–85).

The biopsy sites were as follows: m. vastus lateralis for 84 (56%) biopsies, m. tibialis anterior for 33 (22%) biopsies, and m. deltoideus for 6 biopsies (4%). One patient had one biopsy taken from m. vastus lateralis and one from m. tibialis anterior at the same biopsy visit. Records of biopsy site were not available for 25 (17%) of the biopsies. The weight and cross sectional areas were determined on a limited number of biopsies. The weight varied between 23 and 123 mg and the cross sectional area varied between 2.5 and 16 mm². In total 149 routine histopathological and 6 immunohistochemical analyses were performed. Sixteen rheumatologists performed 126 of the biopsies. Twenty-two biopsies were performed by 13 different rheumatology fellows. Information concerning biopsy performer was missing for one biopsy.

Histopathologic and diagnostic evaluation. It was possible to carry out histopathological evaluations in 145 of the 149 biopsies. Four (3%) biopsies did not contain enough muscle tissue to allow assessment. These biopsies consisted of endstage muscle tissue consisting of fat, connective tissue, or subcutaneous fat. The muscle biopsies were well preserved, and there were no signs of damage due to biopsy sampling. Thirty-nine (26%) biopsies had definite histopathological changes, mostly showing signs of muscle inflammation compatible with active myositis or signs of vasculitis. Forty-six (31%) biopsies were normal. Unspecific findings were detected in the remaining 60 (40%) biopsies.

The majority of biopsies (n = 106) were carried out for primary diagnostic purposes. In some cases, there was more than one diagnostic suspicion. We divided the biopsies into 4 groups: (1) biopsies performed on patients with no previous rheumatological disease and with suspicion of myositis (n = 24), (2) biopsies performed on patients with other rheumatological diseases and with suspicion of associated myositis (overlap syndrome enquiry) or vasculitis (n = 43), (3) biopsies performed on patients with a primary suspicion of systemic vasculitis (n = 19), (4) biopsies performed on patients with myalgia as the main symptom and without proximal muscle weakness (n = 20). Thus there was a low clinical suspicion of myositis in these cases and a biopsy was performed to exclude IIM or a metabolic myopathy.

A second indication for biopsy was to evaluate the effect of treatment or to investigate an eventual relapse of the IIM. Forty-three such rebiopsies were performed.

Biopsies for Diagnostic Purpose

1. In 24 biopsies, the major indication for muscle biopsy was suspicion of myositis. The biopsy results are presented in Figure 3 and Table 1. The criteria for definite or probable

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**Figure 3.** Clinical diagnosis and muscle biopsy findings in patients with primary diagnostic suspicion of myositis. A positive myositis biopsy was defined as inflammation and degenerated and/or regenerated muscle fibers. *Denotes biopsies from patients fulfilling definite or probable DM/PM according to Bohan and Peter criteria. PM: polymyositis, UCTD: unspecified connective tissue disease, SSc: systemic sclerosis, DM: dermatomyositis, PMR: polymyalgia rheumatica, SLE: systemic lupus erythematosus, SS: Sjögren’s syndrome.
PM or DM were fulfilled in 12 of the 24 patients with a clinical suspicion of myositis, and a positive muscle biopsy was found in 10 of these 12 cases, meaning a positive biopsy rate of 83% in this group of patients.

2. For 43 biopsies, the patients already had a rheumatological diagnosis and an associated myositis or vasculitis was suspected. The biopsy results are presented in Figure 4 and Table 2. The criteria for definite or probable PM were fulfilled in 9 of the 43 patients who already had another rheumatological diagnosis, and a positive biopsy was noted in 8 of these 9 cases, meaning a positive biopsy rate of 89% in this group of patients.

3. Nineteen biopsies were performed due to a clinical suspicion of systemic vasculitis (Figure 5). This was confirmed on histopathological grounds in 2 biopsies. Nine of the 19 patients received a clinical vasculitis diagnosis, one of the 2 with positive biopsies and 8 with negative biopsies. Thus, in the vasculitis suspicion group, a positive muscle biopsy showing vasculitis was found in one of 9 patients (11%). One biopsy was positive but the clinical context was not specific enough for a defined systemic vasculitis diagnosis.

4. Twenty patients had myalgia as the main symptom and no muscle weakness. Even though the suspicion of myositis was low, biopsies were performed to exclude IIM or a metabolic myopathy as diagnosis. CPK levels were slightly raised, < 360 units/l, in 5 patients and high, 2742 units/l, for one patient. The remaining 14 patients had normal CPK values. Eleven biopsies were normal, 6 showed type 2 fiber atrophy, one had signs of neuropathy, one showed fat atrophy, and one biopsy did not contain adequate muscle tissue, i.e., no biopsy showed histopathological changes of myositis. The clinical diagnoses acquired after the biopsies were unspecific myalgia or fibromyalgia in 17 patients, systemic sclerosis (SSc) in one, psoriatic arthropathy in one, and muscle trauma in one patient (the patient with the high CPK level).

Rebiopsies
Forty-three biopsies were performed to evaluate the effect of treatment or to investigate a possible relapse of myositis in patients already diagnosed with IIM. Fifteen had inflammatory infiltrates indicating relapse. Twenty-five showed no inflammatory infiltrates, and 3 biopsies consisted of inadequate muscle tissue.

CPK Levels, EMG, Cortisone Dose, and Biopsy Findings
CPK values were available for 130 of the biopsies. In 63 cases CPK levels were elevated (mean value 618 units/l), and in 43% of these patients there were signs of active muscle inflammation in the biopsy. In 67 cases the CPK levels were normal at the time of biopsy, yet 3 of these biopsies had signs of active muscle inflammation.

For the first 2 groups that underwent biopsy for primary diagnostic purposes (n = 67), i.e., myositis or vasculitis was suspected, cortisone dose and EMG results were also considered. Forty-four of 67 patients were not taking cortisone at the time of the biopsy and 13 of these had positive biopsies. Fourteen of the 67 patients had ≤ 10 mg cortisone daily, of which 5 had positive and 9 had negative biopsies. Nine of 67 patients had > 10 mg cortisone daily and all had negative biopsies.

Eighteen of the 67 patients had EMG results compatible with active myositis. Thirteen of these had positive and 5 had negative biopsies; 28/67 EMG analyses were normal.

Table 1. Muscle biopsies for diagnostic purposes due to primary suspicion of myositis, n = 24. The relationship between fulfilled diagnostic criteria, histopathological diagnosis, and clinical diagnosis for myositis.

<table>
<thead>
<tr>
<th>Diagnosis*</th>
<th>No. of Patients</th>
<th>Clinical Diagnosis</th>
<th>Cortisone Dose at Biopsy, mg/day (No. of patients)</th>
<th>Positive or Negative Myositis Biopsies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite PM</td>
<td>5</td>
<td>5 PM</td>
<td>0</td>
<td>+ 5</td>
</tr>
<tr>
<td>Definite DM</td>
<td>1</td>
<td>DM</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>Probable PM</td>
<td>6</td>
<td>3 PM</td>
<td>0</td>
<td>+ 3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 no diagnosis (PM later)</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 SLE (PM later)</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 UCTD</td>
<td>2.5 (every 3rd day)</td>
<td>+</td>
</tr>
<tr>
<td>Possible PM</td>
<td>3</td>
<td>1 SS</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 hereditary neuropathy</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 no diagnosis</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>No PM or DM</td>
<td>9</td>
<td>7 another inflammatory connective tissue disease</td>
<td>5 (1)</td>
<td>– 9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 no diagnosis</td>
<td>0 (8)</td>
<td></td>
</tr>
</tbody>
</table>

*Bohan and Peter criteria21,22.
SLE: systemic lupus erythematosus, UCTD: unspecified connective tissue disease, SS: Sjögren’s syndrome.
but 4 of these had positive and 24 had negative biopsies; 21/67 patients did not undergo EMG.

**Effect of Biopsy Result on Treatment**

In evaluating whether to change treatment or not, many variables were used in the clinical situation. Besides muscle biopsy pathology results and clinical data, anti-Jo-1 antibodies, CPK levels, EMG, and magnetic resonance imaging results were also taken into account. Treatment was changed after 69 (48%) out of 145 adequate biopsies. The treatment included cortisone and immunosuppressive drugs. Seventeen of these biopsies were normal and the remaining 52 presented with at least one pathological feature.

**Complications**

Complications were reported following 4 of the 149 biopsies. Information was missing for one biopsy. A serious subfascial hematoma in the thigh occurred in a man with severe atherosclerosis and on treatment with low dose acetylsalicylic acid. The indication to perform muscle biopsy on this patient was due to suspicion of vasculitis, but this could not be confirmed. In another case, localized pain and persisting hypoesthesia occurred distal to the biopsy site in m. tibialis anterior. In 2 more cases, the patients experienced temporary pain at the biopsy site for less than 2 weeks.

**DISCUSSION**

In a rheumatology practice, the percutaneous conchotome muscle biopsy technique was easy to learn and was performed by most physicians with a low complication rate. The biopsy samples were of adequate size with a good quality to allow required diagnostic procedures. The diagnostic yield in patients with clinical evidence of myositis was high, but low in patients with clinical symptoms and signs of systemic vasculitis.

The percutaneous conchotome biopsy method is easy to perform, taking 15–30 minutes, with local anesthesia at the outpatient clinic. It was possible to analyze both histopathological and enzymatic stainings in all biopsies except 4, which consisted of fat or connective tissue due to endstage muscle or to a biopsy taken from subcutaneous tissue. Immunohistochemistry stainings were not routine analyses and were only performed when it was expected to add information to the diagnostic procedure. These stainings could easily be done retrospectively on stored, frozen tissue. Since one can take multiple samples at the same biopsy site/occasion, the tissue amount was also adequate for both histo-

### Table 2.

<table>
<thead>
<tr>
<th>Diagnosis*</th>
<th>No. of Patients</th>
<th>Clinical Diagnosis</th>
<th>Cortisone Dose at Biopsy, mg/day (No. of patients)</th>
<th>Positive or Negative Myositis Biopsies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite PM</td>
<td>2</td>
<td>MCTD</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SSc w/lung fibrosis and PM</td>
<td>2.5</td>
<td>+</td>
</tr>
<tr>
<td>Probable PM</td>
<td>5</td>
<td>SSc w/myositis</td>
<td>5 (1) and 0 (1) + 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SLE w/myositis</td>
<td>5</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RA w/myositis</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SS w/PM</td>
<td>2.5</td>
<td>+</td>
</tr>
<tr>
<td>Definite or probable PM (no EMG performed)</td>
<td>2</td>
<td>MCTD w/myositis</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td>APL</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>Possible DM</td>
<td>1</td>
<td>RA, SLE w/DM</td>
<td>10</td>
<td>–</td>
</tr>
<tr>
<td>Possible PM</td>
<td>2</td>
<td>SS w/neuropathy</td>
<td>15</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td></td>
<td>arthritis</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>No PM or DM</td>
<td>31</td>
<td>vasculitis</td>
<td>25 (1); 7.5 (1) –31 = 10 (6)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>27 rheumatic diagnoses: 6 RA, 3 UCTD, 4 SS w/ or w/o neuropathy, 4 SLE, 2 DLE, 2 MCTD, 1 Raynaud’s syndrome, 1 PMR, 1 myalgia, 1 psoriatic arthritis, 1 reactive arthritis, 1 APL</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>17.5 (1); 10 (1) –31 = 10 (5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>17.5 (1); 60 (1) –31 &gt; 10 (5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0 (16)</td>
<td></td>
</tr>
</tbody>
</table>

Figure 4. Clinical diagnosis and muscle biopsy findings in patients with rheumatic disorders and with diagnostic suspicion of associated myositis or vasculitis. A positive myositis biopsy was defined as inflammation and degenerated and/or regenerated muscle fibers. A positive vasculitis biopsy was defined as inflammation in blood vessel walls and endothelial involvement. *Denotes biopsies from patients fulfilling definite or probable DM/PM according to Bohan and Peter criteria. RA: rheumatoid arthritis, MCTD: mixed connective tissue disease, DLE: discoid lupus erythematosus, APL: antiphospholipid syndrome, PAN: polyarteritis nodosa, WG: Wegener’s granulomatosis.

Figure 5. Clinical diagnosis and muscle biopsy findings in patients with primary diagnostic suspicion of systemic vasculitis. A positive vasculitis biopsy was defined as inflammation in blood vessel walls and endothelial involvement. UCTD: unspecified connective tissue disease.
chemical and biochemical methods. Unlike an earlier study, we did not find that structural damage caused by mechanical factors in the periphery of the muscle biopsies was a problem for analysis.

Moreover, the diagnostic yield in myositis patients was high, with 83% positive biopsies in the group with myositis as primary suspicion and 89% positive biopsies in the group with myositis complicating another rheumatologic disease. As this was a retrospective evaluation of a method adopted in a clinical practice, a comparison with other biopsy methods was not possible. Nonetheless, a comparable high success rate for the percutaneous conchotome method in comparison with the open biopsy method was observed in another controlled trial. Our rate of positive biopsies among myositis patients is also comparable with reports in which the needle biopsy technique was used. In patients with systemic vasculitis, however, the diagnostic yield from the muscle biopsy technique in our study was low, 11%, similar to other studies. This could probably be explained by the relatively few large blood vessels caught in these biopsies. Open biopsies with larger tissue samples might increase the positive biopsy rate in this particular group of patients. In the third group of patients who were subject to muscle biopsy, the biopsies were normal or displayed unspecific changes. These biopsies were clinically still relevant to exclude myositis or other myopathies. Also, none of these patients have been diagnosed with myositis during our 2 year observation period since the end of the study.

There was a change of treatment after half of the biopsies that consisted of adequate muscle tissue. These biopsies were both normal and pathological. Thus, a biopsy that did not show current inflammatory activity could change the patient’s treatment, for example, lower his/her cortisone dose. Normal biopsies were just as important as pathological ones in this aspect. Rebiopsies were performed on liberal grounds in patients with PM or DM who developed increased muscle symptoms during immunosuppressive treatment. Remarkably, one-third of these biopsies had signs of persisting inflammation and more intense immunosuppressive treatment was given.

The described method was safe for the patient. However, there were complications, of which one was serious, a hematoma followed by deep venous thrombosis in a man with advanced atherosclerosis. This complication may be avoided by applying a pressure bandage to the biopsy area and we changed our routines thereafter. Caution should be taken with patients undergoing anticoagulant treatment and with international normalized ratio > 1.80. The conchotome technique offers a wider range of muscles available for biopsy compared to the needle biopsy technique. In our study, m. vastus lateralis, m. tibialis anterior, and m. deltoideus were all adequate biopsy sites. Notably, pain was reported after occasional biopsies from m. tibialis anterior, and particularly for this site, the physician performing the biopsy has to be familiar with anatomy to prevent nerve damage. In a controlled trial no complications were reported with the conchotome method. This technique was also reported to be less painful than the needle method. Similarly to earlier studies, our patients were mobile immediately after the procedure. Only a few studies report repeated biopsies. Repeated biopsies have been considered doubtful due to the trauma caused by open muscle biopsies. In a study on corticosteroids, decreased cellular infiltration and a downregulating effect on the expression of interleukin 1α and β as well as on adhesion molecules in muscle tissue were observed in muscle biopsies with the conchotome. These findings support the feasibility of this method to evaluate the effect of a given therapy on cells and molecules in muscle tissue.

Although the percutaneous conchotome biopsy technique is a readily applicable method for both diagnostic and research purposes, there is, nonetheless, a limited place for the open and needle biopsies. The open biopsy technique may be useful to locate vessels in vasculitis. The open technique could also be preferable for use on infants and children with atrophic muscles. The needle biopsy may be sufficient if only biochemical investigations for metabolic analyses are to be performed on the samples.

The percutaneous conchotome muscle biopsy technique is a sensitive method to simply and safely make diagnostic evaluations and assess the effect of a given therapy on muscle tissue in patients with idiopathic inflammatory myopathies. A good technique such as this is paramount to study muscle tissue on a molecular level during different stages in the inflammatory process. Such studies might give insight into etiology and pathogenesis and thus provide increased possibilities to develop more targeted treatment for the idiopathic inflammatory myopathies.

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