

ANTERIOR KNEE PAIN SYNDROME

Abstract

Many diseases and types of injuries of the knee joint take a lot of time for diagnosis and the elimination of all nosologies that may cause pain. The complexity of the anatomical device of the knee joint makes it possible to isolate from the general concept of pain in the knee joint of this pathogenetically grounded syndrome. The review gives an idea of a significant number of types of knee joint pathology, which, due to the structural features and similarity of the clinical picture, can be combined into the anterior knee joint pain syndrome.

Keywords: knee joint, syndrome, pain, pathology.

INTRODUCTION

Pain is interpreted as subjective sensory and emotional experiences associated with actual or potential tissue damage. Nociceptive somatic pain associated with irritation of peripheral nerve endings, damage to cell membranes and release of pain and inflammation mediators occurs in bones, joints, muscles, skin and connective tissue. The knee joint with a complex structural architectonics, with the presence of structures with rich vascularization and innervation, and sometimes subjected to excessive loads, compared with other joints of the musculoskeletal system, is the most common reason for seeking a specialist [1]. The presence of many types of injuries and diseases of the knee joint, until recently, took away the efforts to exclude all nosologies that may cause pain, thereby increasing the duration of disability of the patient. The complexity of the device and the presence of both intra-articular and extra-articular components of the knee joint allows us to introduce the anterior pain syndrome of the knee joint. Isolation from the general concept of pain in the knee joint of this syndrome is pathogenetically justified because will allow the specialist to reduce the time spent on search, differential diagnosis and treatment of the patient [2].

The knee joint with complex biomechanics is often subjected to loads that exceed the functionality of the latter. Consequently, the main group of patients seeking help are athletes. Knee anterior pain syndrome in athletes is a difficult problem in terms of diagnosis and treatment. The nature of injuries of the knee joint, leading to the development of anterior pain, can be divided into acute and chronic injuries caused by prolonged excessive exercise. The main cause of acute injuries is sports, especially contact sports, such as football, basketball, while non-contact sports, such as athletics, often lead to chronic injuries [3]. The concept of anterior pain in the knee joint is far from the old understanding of the structural etiological causes in the development of pathological processes, including both

39 bone and soft tissue structures, a significant violation of homeostasis, as the main
40 cause in the genesis of patellofemoral pain [4].

41 There is a division of the causes of this syndrome into those in which focal
42 lesions can be diagnosed clinically and radiologically, and the causes of “unclear”
43 genesis, for example, the syndrome of lateral hyper pressure of the patella [5]. It
44 was also proposed to narrow down the causes of potential diagnoses depending on
45 the time and persistence of pain: constant pain, pain associated with physical
46 activity, acute and remitting pain [6]. Christian (2006) with co-workers added
47 anatomically isolated points, dividing into pain the causes of which are the patellar
48 tendon ligament [PTL], the patella, intra-articular pathology and bursitis [7]. We,
49 in turn, cannot fail to note such etiological causes of this syndrome as
50 manifestations of systemic diseases of the connective tissue, bone tissue tumours,
51 pathology of the synovial membrane, chondromatosis, exostotic disease.

52 This article is intended to highlight the anatomical separation of the causes
53 of anterior knee pain syndrome, although all structures of the knee joint are closely
54 interrelated and should be considered as a single mechanism.

55 We would like to note the importance of the patellofemoral joint (PFJ) in the
56 genesis of anterior pain, as an important functional structure of the knee joint with
57 complex biomechanics [8]. In the case of superphysiological loads that exceed the
58 allowable, the homeostasis of the PFJ, which leads to increased injury and pain.
59 The ability to transfer loads depends on many factors: the correctness of the
60 trajectory of movement, neuromuscular tone and control, absolute loads for a long
61 time, etc. [9]. Peripheral soft tissues, in particular, the peripapillary synovial folds,
62 the fatty body of Hoff also contribute to the appearance of patellofemoral pain.
63 Any PFJ structure can be a potential cause of knee anterior pain syndrome. Nerve
64 endings are concentrated in the PTL, in the tissues of the retinaculum, in the
65 “goose foot” (**pes anserine**), and especially in the synovial folds and the fat body.
66 Articular surfaces, menisci and ligaments are less sensitive. The articular cartilage
67 has no innervation, the subchondral bone can be the cause of pain in case of severe
68 overload and damage to the articular cartilage [10].

69 Since the perception of pain is a function of the central nervous system, in
70 addition to direct nociception from PFJ structures, pain in the knee joint can be
71 provoked by the pathology of the hip joint. The hip joint extensors play an
72 important role in the movements of the lower limb and contribute to the absorption
73 of up to 25% of the load when walking. When the muscles of the hip joint do not
74 absorb their share of the load, this should be compensated by other structures, in
75 particular, the knee joint [44]. For example, children with hip joint pathology, such
76 as femoral head epiphysiolysis, and adults with varying degrees of osteoarthritis
77 may complain of pain in the knee joint, although there is no pathology in the latter.
78 The lack of changes in the knee joint should suggest a thorough examination of the

79 hip joint [11]. Pathological movement of the lower extremities is observed in
80 rotational deformities in young patients: increased femoral anteversion, the internal
81 orientation of the patella, external rotation of the tibia, pronation of the foot,
82 causing pain in the front of the knee joint.

83 Anterior pain syndrome of the knee joint may be caused by trauma to the
84 intra-articular structures [45]. Damage to the anterior cruciate ligament (ACL)
85 leads to rotational instability and overload of the medial part of the knee joint. The
86 most common damage to the articular surface of the medial condyle of the femur
87 in ACL injuries. Damage to the posterior cruciate ligament (PCL) leads to the
88 posterior displacement of the tibia and overloading of the anteromedial part of the
89 knee joint. Syndrome of anterior pain of the knee joint with a decrease in the range
90 of movements occurs after the reconstruction of the ACL and is a consequence of
91 the development of the following complications: arthrofibrosis, cyclops syndrome
92 and syndrome of infrapatellar contracture [12].

93 Patellar trauma is a direct cause of anterior pain syndrome and may be due to
94 a fracture or a patellar contusion. In children, tear-off fractures of the lower pole of
95 the patella are rare, but there may be tearing of the articular cartilage or
96 periosteum. In children and adolescents, Osgud-Schlätter or Larsen-Johansen
97 syndrome may be a likely cause of anterior pain. Pain in the distal pole of the
98 patella or above the tibial tuberosity, are characteristic clinical signs.
99 Radiographically, ossification or fragmentation due to a partial separation of bone-
100 cartilage fragments will be determined, magnetic resonance imaging (MRI) will
101 determine the presence of ossification, thickening of the PTL and swelling of the
102 near-tempering tissues [13, 14]. Osgood-Schlatter disease can be a predisposing
103 factor for the development of stress fracture tibial tuberosity [15]. Early diagnosis
104 of stress fractures of the patella, occurring predominantly at the junction of the
105 middle and distal third of the patella, is important for ensuring adequate
106 conservative treatment before the separation of fragments occurs [16]. Violations
107 of the ossification of the patella in 1-2% of cases are the cause of the development
108 of the lobed patella. In rare cases, after physical exertion and acute injury, the
109 lobed patella can cause anterior pain of the knee joint. Differential diagnosis
110 should be carried out with Larsen-Johansen syndrome, a detachable fracture of the
111 distal pole of the patella and a stress fracture when finding a fragment from the
112 lateral side [16, 17, 18].

113 The consequences of acute dislocation of the patella, whether as a result of
114 an acute injury or rotational tension of the extensor apparatus, can also be the cause
115 of anterior pain syndrome. The impact on the outer surface of the knee joint can
116 result in injury to the medial structures of the knee joint, which is often found in
117 young athletes [19]. MRI is the method of choice for diagnosing the effects of
118 patellar dislocation such as hemarthrosis; rupture of the medial retinaculum and the

119 capsule of the knee joint, with the formation of hematoma, edema, partial rupture
120 of fibers; contusion of the lateral condyle of the femur and medial facet of the knee
121 joint, trabecular microcracks; damage to the articular cartilage of the lateral
122 condyle of the femur and the medial facet of the patella, and the associated finding
123 of osteochondral free bodies [20].

124 The most common causes of cartilage damage are injuries, physical overload
125 and loss of stability of the joint. It can occur both in isolation and in combination
126 with damage to other intra-articular structures, PTL, Goff's fatty body, instability
127 of the patella. Bohndorf indicated arthroscopic and MRI signs of cartilage damage,
128 divided injuries into concomitant, with and without cartilage lesions [21]. MRI as a
129 diagnostic method perfectly differentiates the structure of cartilage and other soft
130 tissues, thinning or surface defects are clearly visible in FSEPD-mode. Perspective
131 methods for physiological visualization of cartilage damage, such as T2 images,
132 diffuse weighted images, and dGEMRIC (delayed gadolinium-enhanced imaging)
133 mode, are promising [22]. Damage can be divided into subchondral injuries,
134 osteochondral fractures and exclusively cartilage damage. The term "dissecting
135 osteochondrosis or osteochondritis" is described as bone-cartilaginous pathology
136 found in young athletes. The most frequent localization is in the femoral sulcus, on
137 the inner surface of the medial condyle of the femur. The defect can be detected on
138 the patella. MRI allows you to diagnose, determine the stability of the fragment
139 [23, 24].

140 Injuries to the knee joint extensor apparatus (KJEA) are the main cause of
141 anterior knee pain in professional athletes. In recent years, the role of PTL
142 tendinopathy in pathology has increased, due to the increase in the duration and
143 intensity of training and competition [25]. Periods of relative inactivity and active
144 physical activity in professional athletes, during irregular sports competitions, as
145 well as "athletes of the day off" allow us to add them to the risk group. Injecting
146 steroids, taking systemic corticosteroids, fluoroquinolones increases the risk of
147 tendon rupture [26]. PTL tendon is 25-30% thinner than the quadriceps tendon and
148 therefore increases the risk of injury during sports [27]. KJEA performs 2
149 important functions, the function of strengthening with concentric contraction
150 (jumps, hitting the ball) and the deceleration function during eccentric contraction
151 (landing after the jump, descending the stairs). The braking mechanism is able to
152 overload the PTL above possible strengths. KJEA also plays an important role in
153 regulating the external and internal rotation of the tibia [28]. Due to the unique
154 anatomical properties and structure of the tendon, the forces generated during its
155 movement are usually insufficient for its rupture, the only regular excessive
156 physical exertion of KJEA can cause damage and, as a result, anterior pain of the
157 knee joint. Degenerative changes, as compared to inflammatory changes, are more
158 often found at tendon ruptures, which indicate the presence of a pre-rupture phase
159 or a predisposition to rupture. In addition to external causes of PTL tendinopathy,

160 which include repetitive mechanical loads, internal factors include instability of the
161 patella, high standing of the patella, impingement of the lower patella pole,
162 increased muscle tone [29]. After recent scientific studies in which no
163 inflammatory cells were found, the question of the diagnosis of tendinitis
164 sportsmen with pain and weakness in the area of PTL should be questioned [30,
165 31]. Loss of normal structure microscopically tears of collagen fibres, necrotic
166 modified fibres, as well as mucoid degeneration with different fibrosis and
167 neovascularization are detected macroscopically. For the first time, PTL
168 tendinopathy was described in jumpers, and the disease was called the jumper's
169 knee [32]. We must take into account that morphological changes do not always
170 correlate with the clinical picture, and specific signs can be found in asymptomatic
171 athletes. Patients with asymptomatic signs found by radiological methods (MRI)
172 should be under the supervision of a specialist because of increases the risk of
173 disease. In addition, characteristic signs of tendinopathy of PTL are local or diffuse
174 hypoechogenicity, tendon thickening, and uneven contours, swelling of the
175 parastomal tissues and structures, and increased vascularization on colour Doppler.
176 Hyperechogenic areas that are pockets of dystrophic ossification can also be
177 detected [33, 34]. Increased strength, biomechanical features, and enhanced
178 vascularization of the quadriceps femoris tendon (QFT) are the causes of the rarer
179 cases of PTL tendinopathy, compared with tendinopathy of PTL. Adolescent
180 fractures of the proximal patella pole are more common in adolescents compared
181 with tendinopathy. In elderly patients, degenerative changes, such as calcifications
182 and spurs of the upper patella pole, can be observed and cause anterior pain of the
183 knee joint.

184 Violation of the normal positioning of the patella relative to the block of the
185 femur can also be the cause of the syndrome of anterior pain of the knee joint, and
186 in severe cases, the cause of instability of the patella. Decentration of the patella,
187 disruption of its normal movement results in excessive stresses and shear forces
188 exceeding physiologically acceptable thresholds, and as a result, tendons,
189 ligaments, cartilage and bone injuries develop. The fact that an abnormal structure
190 can occur in people who do not complain, that differences can occur at different
191 angles of flexion in the knee joint, is the reason for the difficult diagnosis of the
192 pathology of PFJ [35, 36, 37]. The lateral inclination of the patella, as the most
193 common cause, as well as the high or low standing of the patella, the anomaly of
194 the position of the tibial tuberosity (TT), are variants of the PFJ pathology. Q-
195 angle is an angle showing the magnitude of the varus deformity of the knee joint.
196 The normal angle is 15 degrees. The TT-TG index determined in CT images can
197 replace the definition of a Q-angle in clinical diagnostics. An indicator of 1.8-2.0
198 cm is specific for a violation in the PFJ, namely for the decentered position of the
199 patella [38, 39]. The furrow angle, congruence angle, lateral patellofemoral angle,
200 and lateral displacement of the patella are used more frequently [40, 41].

201 The syndrome of the orio-tibial tract (“runner’s knee”) is also a cause of pain
202 in the knee joint. Occurs as a result of constant friction between the orothibial tract
203 (OTT) and the lateral epicondyle of the femur. This syndrome is more common in
204 long-distance runners, cyclists, as well as military personnel, i.e. in any activity
205 that requires repeated flexion-extension movements in the knee joint. Barrel
206 deformity, excessive pronation with internal rotation of the leg, spur of the lateral
207 condyle, as well as the different length of the lower extremities, all this can
208 increase the tension of OTT and create friction on the lateral epicondyle. Other
209 potential factors for the development of OTT syndrome: large weekly runs, or
210 cycle load with weakness of the extensor muscles of the knee joint, flexor muscles,
211 hip abductors. The weakness of the hip abductors leads to an increase in hip
212 adduction and an increase in tension over OTT [42, 43].

213 Goff's adipose tissue is intraarticular, but the extrasynovial structure, rich in
214 vascularization and innervated. Often, the pathology of this structure is found
215 together with other problems of the knee joint, such as PTL tendinopathy,
216 conditions after PCA reconstruction, at meniscus ruptures, instability of the knee.
217 Direct injury can also be the cause of this pathology. Different variants of Goff's
218 fatty body oedema can occur in various pathologies, maybe in the form of 2
219 variants: infra-infraredular impingement syndrome and impinging of the upper
220 lateral angle of Hoff's body. It is believed that hypertrophy and inflammation of
221 the fatty body of Hoff are secondary after compression between the femoral
222 condyles and the tibial plate during extension of the knee joint. Symptoms include
223 pain in the anterior region of the knee joint, below the top of the patella. The pain
224 is exacerbated by extension in the knee joint [18]. MRI scans show increased
225 intensity at T2W, as well as a small effusion. In the subacute and chronic stages,
226 due to hemosiderin and fibrin deposits, a low signal is detected in the T1W and
227 T2W modes. Deviation of the patellar tendon may be due to a mass effect. Fibrous
228 tissue can be gradually organized into a fibrocartilaginous fibroid, in rare cases,
229 Hoff schismatization may occur [26]. Upper-lateral and pre-femoral swelling of the
230 fatty body of Hoff is often associated with patellar chondromalacia, dysplasia of
231 the femoral block, improper position of the patella, pathology of PTL and patella.

232 Synovial folds in rare cases can cause anterior knee pain in adolescents,
233 although the relationship between the crease and anterior pain is controversial. The
234 syndrome of the medial fold is a combination of clinical symptoms with the
235 presence of a pathological fold. Usually found in young athletes, with such
236 repetitive movements as flexion-extension, for example, rowing, swimming,
237 cycling, basketball. The large fold that covers the medial condyle of the femur may
238 be damaged when squeezed between the femoral condyle and the patella. Due to
239 the regular repetition of this movement, damage to the cartilage may be caused [5,
240 22, 40]. The suprapatellar fold is located on the border between the suprapatellar
241 sac and the cavity of the knee joint. Recently, it has been suggested that it may be

242 the cause of the anterior pain of the knee joint, especially with a full separation of
243 the suprapatellar pocket from the joint cavity. The infrapipellary fold is the most
244 frequent fold in the knee joint. On MRI, it is detected as a low-intensity signal in
245 front and parallel to the anterior cruciate ligament ACL in sagittal images.
246 Traditionally, the infrapatellar fold was thought to be a random discovery and not
247 associated with clinical symptoms. However, some studies describe it as a rare
248 cause of anterior pain in the knee joint, which can be thought of in the absence of
249 other pathological causes; it can mimic the rupture of ACL [11].

250 In addition to the above-described diseases and conditions in violation of the
251 biomechanics of the knee joint, benign and malignant tumours can be one of the
252 causes of pain in the anterior section of the knee joint. Vaginal-nodular pigmented
253 synovitis (VNPS) and a giant tumour of tendinous vaginal cells, terms often used
254 interchangeably to describe predominantly benign conditions, with the
255 proliferation of synovial cells of the joints, tendons and synovial bags. A tumour of
256 giant cells of diffuse type is defined as the destructive proliferation of mononuclear
257 cells of the synovial type with an admixture of multinucleated giant, foamy,
258 inflammatory cells and siderophages [14]. This form affects joints more often, but
259 there can be a lesion of extra-articular soft tissues, characterized by infiltrative
260 growth. The localized type, which can be found in tendon sheaths, bursa, is
261 characterized by the same cellular composition as the diffuse type, but as a rule, it
262 is smaller, well limited and less destructive growth. The intra-articular form of
263 VNPS is a rare disease with a frequency of 1.8:1,000,000, usually occurs in the
264 fourth decade of life without sex. It is the knee joint that is most often affected, and
265 the lack of physical examination data, and such nonspecific symptoms as sudden
266 pain, the appearance of oedema and restriction of movements, makes it difficult to
267 diagnose [6, 31, 36-37].

268 Synovial chondromatosis is a rare benign condition characterized by the
269 presence of cartilaginous nodules in the synovial membrane of the joints, tendon
270 sheaths and synovial bags, which are often found without prior injury and
271 inflammation [27]. As the disease progresses, free bodies may be ossified [42]. The
272 condition is generally considered monoarticular and more than 50% of reported
273 cases are described with lesions of the knee joint [28]. The extra-articular form is
274 rare, but with an X-ray picture with the presence of large extra-articular calcinates,
275 it is necessary to carry out differential diagnostics with idiopathic tumour scinosis,
276 which occurs in people of Africa and the Caribbean in the second decade of life
277 [9]. Extra-marginal lesions can be classified as tenosynovial chondromatosis or
278 chondromatosis synovial bags depending on localization [41]. It is believed that
279 the exact aetiology of synovial chondromatosis is unknown. Milgram, in 1977,
280 divided the development of the disease into 3 separate phases. In phase I,
281 metaplasia of the synovial membrane occurs, synovitis develops and nodules are
282 formed, without calcification. In phase II, nodular synovitis and free bodies of

283 cartilaginous origin in the joint are observed. In phase III, free bodies remain, but
284 synovitis is permitted. Free bodies tend to merge and calcify [35]. There is no
285 histological evidence of metaplasia in the third stage, but there are concerns about
286 a possible conversion to synovial chondrosarcoma.

287 The defeat of such a richly innervated structure, such as the synovial
288 membrane, can also be a direct cause of the pain syndrome, in particular, anterior
289 pain syndrome in the knee joint. Chronic inflammation of the synovial membrane
290 (synovitis) is a fairly common pathology, with a large variability of etiological
291 causes and the complexity of the differential diagnosis. The following groups of
292 etiological causes of chronic synovitis of the knee can be distinguished: non-
293 inflammatory (for example, in osteoarthritis, gout, accumulation diseases),
294 inflammatory (in rheumatoid arthritis, reactive arthritis, acute rheumatic fever),
295 septic in nature (purulent bacterial and septic arthritis) and hemorrhagic in nature
296 (arthritis associated with trauma, tumor, coagulopathy) [1]. An important role in
297 the development of chronic synovitis is played by disturbances in local
298 disturbances of homeostasis, disturbances in the antioxidant system and activation
299 of lipid peroxidation, contributing to the development of membrane-destructive or
300 inflammatory-dystrophic changes, which determine the degree of pathological
301 changes in the knee joint. "Diagnostics of the possible causes of chronic synovitis
302 are the histopathological analysis of the synovial membrane and biochemical
303 research with commercial fluid. Given the evidence that reactive arthritis is
304 detected in 10% of patients with rheumatologic hospitals, and the proportion of
305 urogenic patients accounts for up to 50-75% [2], a necessary mandatory study on
306 the polymerase chain reaction of synovial fluid.

307 **CONCLUSION**

308 Our review shows that there are a large number of diseases and nosologies
309 of the knee joint, which, due to the structural features and similarity of the clinical
310 picture, can be combined into anterior pain syndrome of the knee joint. Anterior
311 pain syndrome of the knee joint has the prospect of further study because The
312 education of young specialists and the sharing of our observations with
313 experienced doctors will lead to a regular optimization of differential diagnosis and
314 multimodal treatment of pain in the knee joint.

315 **CONSENT**

316

317 It is not applicable.

318

319 **ETHICAL APPROVAL**

320

321 It is not applicable.

322

323 **COMPETING INTERESTS**

324

325 Authors have declared that no competing interests exist.

326

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