Review Article

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The Incidence of Pars Interarticularis Defects in Athletes

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Abstract

Study Design: Literature review.

Objective: To conduct a literature review of studies reporting the incidence of pars interarticularis defects in athletes of specific sports, in order to allow more targeted prevention and treatment strategies to be implemented for the groups at highest risk.

Methods: Electronic searches were performed using PubMed, Ovid Medline, EMBASE, Google Scholar, Cochrane Database of Systematic Reviews, and Cochrane Database of Controlled Trials from their dates of inception to September 2017, with the following keywords: "spondylolysis," "sports," "low back pain," and "pars defects."

Results: A total of 509 total articles were retrieved, of which 114 were used in the final review. The incidence of pars interarticularis defects was found to be highest in diving (35.38%), cricket (31.97%), baseball/softball (26.91%), rugby (22.22%), weightlifting (19.49%), sailing (17.18%), table tennis (15.63%), and wrestling (14.74%). Only 5 studies reported the management instituted for their participants, and these were all case reports. Of 74 players with spondylolysis in these studies, 70 (94.59%) underwent conservative treatment and 4 (5.41%) underwent surgical treatment. 61 (82.43%) returned to their previous level of play, 6 (8.11%) retired, and the disposition of the final 7 was not reported.

Conclusion: The current medical literature provides good evidence that the incidence of pars interarticularis defects is higher in the athletic population, with the highest incidence in diving. There remains no gold standard protocol for the management of pars interarticularis defects. Further research is required to compare conservative therapy to surgical therapy and to compare the various surgical techniques to each other.

Keywords

spondylolysis, incidence, athletes, sports, review, low back pain, prevalence

Introduction

Pars interarticularis defects relate to spondylolysis and/or spondylolisthesis of the spinal vertebra. The pars interarticularis is the segment of bone bounded by the lamina, pedicle, inferior articular process, and superior articular process of each vertebra (Figures 1 and 2). Pars interarticularis defects begin as stress reactions (pre-lytic stage), then progress to acute fractures (spondylolysis) and eventually chronic fractures.[1] Complete fractures of the pars interarticularis may lead to anterolisthesis of the affected vertebra relative to the vertebra immediately inferior to it[2] (spondylolisthesis), at which point neurological symptomatology may occur.

The patient with lumbar spondylolysis typically complains of progressive back pain in the lumbar region, exacerbated by extension or twisting of the spine.[3] Radicular pain and urinary disturbances are uncommon unless nerve root compression has occurred as a result of spondylolisthesis. A social history indicates previous or current athletic activity in 93% of

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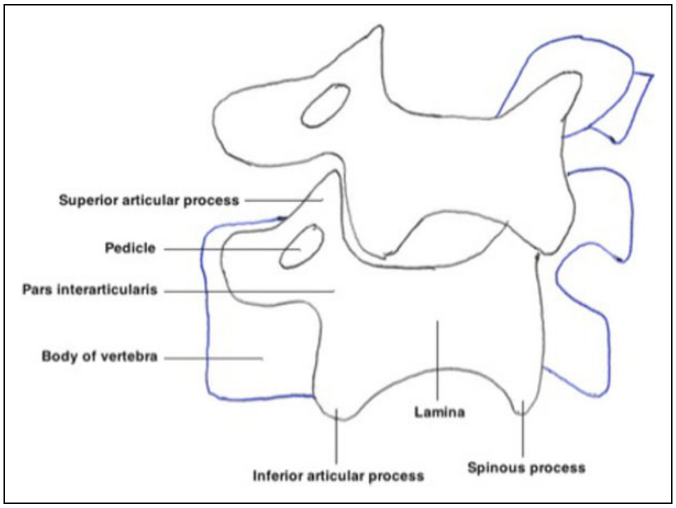

Figure 1. Diagrammatic representation of 2 lumbar vertebra. The pars interarticularis is the region between the lamina, pedicle, superior articular process, and inferior articular process of each vertebra. Each vertebra resembles a "Scotty dog," with defect across the pars interarticularis producing the "Scotty dog collar" sign.

Figure 2. X-ray of the lumbar spine (lateral view) showing a spondylolytic defect.

spondylolysis patients.[4] There may be a family history of spondylolysis, spondylolisthesis, or spina bifida occulta.[5] On examination, there may be loss of lumbar lordosis, as well as restricted lumbar flexion and extension. Paraspinal muscle spasm and tenderness often occurs.[6] The pathognomonic sign is the one-legged hyperextension test (stork test), but this test is not sensitive[7] or specific[8] enough to be relied on for assessment. If spondylolisthesis is present, hamstring tightness leads to shortened stride with hip and knee flexion.[9] There may also be lumbar hyperlordosis leading to the appearance of excessive abdominal convexity.[10,11] Neurological signs are rare, occurring only if nerve root compression has developed.[6,12]

Spondylolysis is most commonly bilateral[13] (Figure 3), possibly because unilateral spondylolysis increases stress on the contralateral pedicle.[14] The majority of spondylolysis occurs in L5, followed by L4, then combined L4-L5 involvement (Table 1).[15-21] Furthermore, the severity of spondylolysis varies with location. Sixty-three percent of L5 spondylolysis exhibits terminal-stage defects despite young skeletal age, while most L3/L4 spondylolysis exhibits early-stage defects.[4]

The incidence of spondylolysis has been shown to be 6% in the general adult population.[17] The incidence is significantly higher in the athletic population, with studies showing as many as 52% of athletes with low back pain suffering spondylolysis and 60% with low back pain suffering a pars interarticularis defect of any grade.[7] Other studies show that spondylolysis and spondylolisthesis constitute 47% of low back pain in adolescent athletes.[22] Athletic participation is indeed a well-known risk factor for the development of pars interarticularis defects, but studies on the incidence of pars interarticularis defects often have 1 of 3 limitations: (*a*) small sample sizes; (*b*) the athletes being studied are from the same sporting institutions, the sporting styles of which may independently contribute to pars interarticularis defects; or (*c*) the studies measure incidence in a small number of sports. As such, this article aims to systematically review the current literature on the incidence of pars interarticularis defects in athletes, with detailed knowledge about particularly high-risk sports allowing the development of more targeted and effective prevention and treatment strategies in the future.

Methods

Data Sources

The authors performed a comprehensive search of the published medical literature, using the following electronic databases from their dates of inception to September 2017: PubMed, Ovid Medline, EMBASE, Google Scholar, Cochrane Database of Systematic Reviews, and Cochrane Database of Controlled Trials. Searches were performed with the following terms as MeSH headings and keywords, with Boolean operations, including "AND" and "OR": "spondylolysis," "sports," "low back pain," and "pars defects." Synonyms were used to identify the remainder of relevant studies. Citations and abstracts were retrieved. A hand search of the bibliographies was also performed to identify relevant articles missed by the electronic search. Two independent researchers performed the literature search.

Study Selection Criteria

Articles were included in the current review if their primary subject matter was the classification, epidemiology, risk factors, diagnosis, or management of pars interarticularis defects, or if they discussed pars interarticularis defects in athletic populations. Articles were excluded if the athletic population was not a significant focus of the article, or if they discussed the total incidence of spinal disorders without providing statistics specifically on pars interarticularis defects. Articles were also

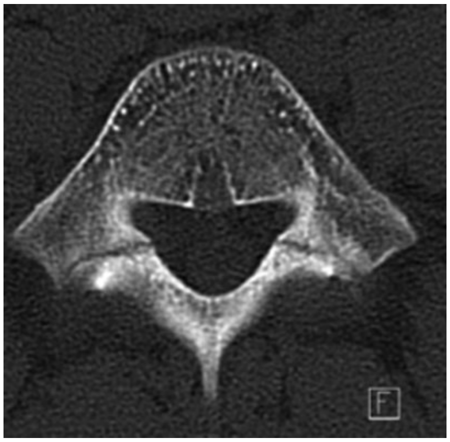

Figure 3. X-ray of the lumbar spine (axial view) showing bilateral spondylolytic defects. This radiograph is from one of the author's patients.

Table 1. Proportion of Spondylolysis Occurring at Specific Lumbar Levels.[a]

Lumbar Level of Spondylolysis	Incidence (%)
L1	0-5
L2	0-5
L3	0-5
L4	5-23
L5	71-95

[a] The majority of lumbar spondylolysis occurs at L5 (71%-95%) followed by L4 (5%-23%). Spondylolysis at L1-L3 is rare (0%-5% each).

excluded if they were not in English, unless the statistics from such articles were recounted in other English articles, in which case they were included in the current review. The articles were reviewed for inclusion or exclusion independently by 2 of the authors, and disputes were resolved by group consensus.

Results

A total of 502 articles were retrieved from database searches, and 115 were retrieved from the reference lists of articles (n = 617). There were 108 duplicate articles, 37 articles not in English, 77 irrelevant articles, 68 articles outside the scope of the current review, 1 commentary, and 1 opinion article (n = 325). Of the 325 remaining articles, 114 were required for the current review with quantitative data collected from 42 articles (Figure 4).

Epidemiology

The incidence of spondylolysis in neonates is zero,[17,24] 4.4% in 6-year-olds, and 6% in adults.[17] The incidence in athletic populations is considerably higher (Table 2). Approximately 75% of spondylolysis will develop into spondylolisthesis.[17]

Hockey. The incidence of pars defects in hockey players has been reported as 2.83% to 44%,[25-28] with spondylolisthesis occurring in 15.9% of this population.[29] The majority of players affected are forwards, with a greater proportion of spondylolysis ipsilateral to their handedness.[25] Defense players more commonly present with spondylolysis contralateral to their handedness.[25] This difference may be accounted for by the different spinal motions required of players in different positions. Forward players are required to shoot the puck on the side of their handedness, leading to ipsilateral spondylolysis. Defense players are required to quickly twist and turn in both directions while defending against players of the opposing team, leading to contralateral spondylolysis.

Tennis. Pars defects are more common in elite tennis players than the nonathletic population,[30] with spondylolysis occurring in 1.1% to 40%[26,27,31-37] of this population due to the excessive forces placed on the lumbar spine during the service game.[38] The topspin serve puts players at a greater risk of lumbar injury than the flat or slice serves,[38-40] since it involves a racquet head more posterior and more medial to the shoulder compared with the flat and slice serves.[39]

Diving. Pars defects in athletic divers have a reported incidence of 0% to 40.35%,[26,27] with the incidence of 0% likely due to the small sample size studied (n = 8).[27] Divers reach speeds of 51 km/h before entering the water, then decelerate to 33 km/h on impact with the water, exerting a strong physical force on their lumbar spines.[3] Divers with low back pain have a larger trunk extension angle than those without low back pain. Trunk extension angle corresponds to the shoulder flexion angle, so having a flexible shoulder can decrease the trunk extension angle in divers, thereby decreasing the risk of low back pain.[3] These results may apply to other sports involving compound movements such as gymnastics and throwing sports.

Volleyball. A total of 3.77% to 20.69% of beach volleyball players suffer from spondylolysis.[12,26,27,31,37] This increased prevalence is due to the powerful overhead hitting motion that volleyball players employ during serve or smash movements, causing malalignment of the shoulders relative to the hips. When this is combined with repetitive lumbar hyperextension to increase the force exerted on the ball, spondylolysis occurs.[12]

Cricket. The incidence of spondylolysis in cricket players is 10.98% to 55%.[34,41-46] L5 is the most commonly affected spinal level, with L4 and L3 less commonly affected.[45,47] Defects tend to arise contralateral to the bowling arm, perhaps explaining why such defects are more common on the left than the right.[42,47] Spondylolisthesis occurs commonly in bilateral pars defects of these athletes, but is rare in unilateral defects.[48] This occurs primarily due to the bowling movement, which requires lumbar flexion, hyperextension, and lateral rotation. Also, reaction forces from the ground on the front foot and back foot, transmitted through the lumbar spine during delivery, are significantly higher than body weight.[45,48] A mixed front-and-side bowling style provides a higher risk of spondylolysis than

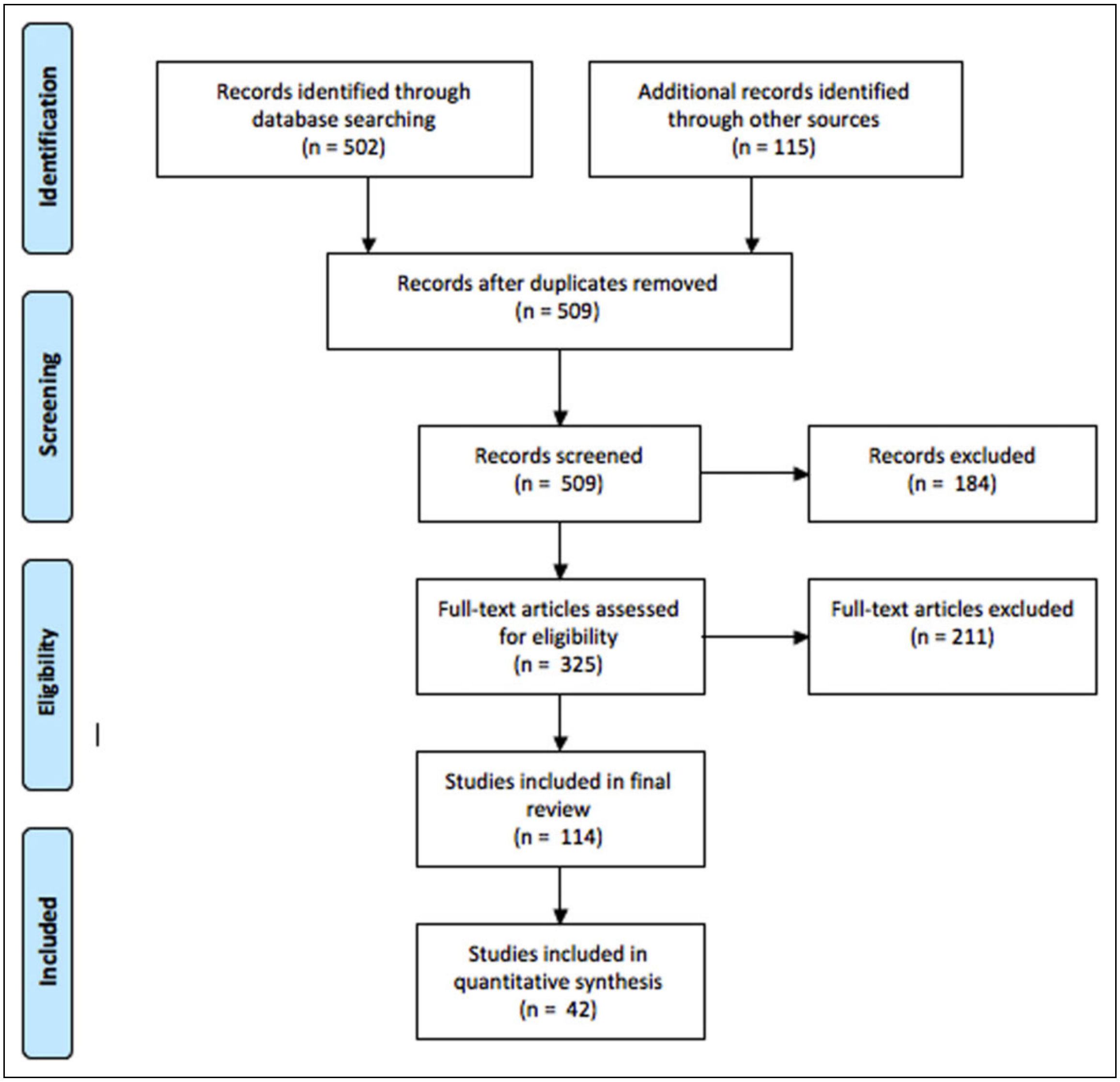

Figure 4. PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flowchart.[23] A total of 504 original articles were retrieved. A total of 114 articles were used in the final review with quantitative data retrieved from 42 of these articles.

front-only and side-only bowling styles, due to higher degrees of these lumbar movements.[42,46,49,50]

Rugby, American Football and Other Contact Sports. Spondylolysis occurs in 0% to 50% of American football players[26,27,51-56] and 5% to 30.77% of rugby players.[26,27,31,56] The lower limit of 0% for American football can be attributed to the small sample size studied (n = 13).[27] These contact sports are different from noncontact sports such as gymnastics, because collision exerts an extra axial loading force on the spine that is not present in noncontact sports. Also, locking of the lumbosacral spine (which physiologically protects the spine) cannot adequately occur in contact sports since there are multiple concurrent forces on the athlete as they compete for possession of the ball.[57] In rugby, lumbar spinal injuries occur most commonly in defensive players due to the axial loading forces during defensive tackles. The scrum and spear tackle also exert further axial and rotational forces which ultimately increase the incidence of pars defects in this athletic population.[58]

Classification

The currently accepted classification system for spondylolysis (Table 3) (type IIa spondylolisthesis) is based on the progression of pathological changes that occur in pars interarticularis defects. Radiological magnetic resonance imaging (MRI) changes are grouped in 5 grades (grades 0-4).[1] Grade 0 is a normal pars interarticularis, with no evidence of stress reaction. Grade 1 refers to T2 signal abnormalities of the pars interarticularis but not of the adjacent pedicle or articular process, representing a bone marrow stress reaction (edema) without cortical disruption. Grade 2 refers to T2 signal abnormalities and thinning, fragmentation or irregularity of the pars interarticularis on T1 or T2 image, representing incomplete pars

Table 2. Incidence of Pars Interarticularis Defects by Sport.

Sport	Sample Size	Athletes With at Least One Pars Interarticularis Defect	
		n	%
Archery			
Rossi et al[26]	26	0	0
Soler et al[27]	44	1	2.27
Total	70	1	1.43
Badminton			
Kobayashi et al[31]	3	2	66.66
Soler et al[27]	38	2	5.26
Total	41	4	9.76
Baseball			
Rossi et al[26]	21	1	4.76
Kobayashi et al[31]	60	37	61.67
Kobayashi et al[31,a]	3	0	0
Kono et al[36]	91	14	15.38
Akimoto et al[37]	143	18	12.59
Hasegawa et al[59]	95	22	23.16
Matsumoto et al[60]	40	11	27.5
Wakitani et al[61]	71	38	53.52
Total	524	141	26.91
Basketball			
Kono et al[36]	66	9	13.64
Rossi et al[26]	174	17	9.77
Kobayashi et al[31]	30	15	50
Soler et al[27]	288	19	6.6
Akimoto et al[37]	96	11	11.46
Total	654	71	10.86
Baton twirling			
Kobayashi et al[31]	1	0	0
Bobsledding			
Rossi et al[26]	36	5	13.88
Rossi et al[26,b]	25	2	8
Soler et al[27]	15	3	20
Total	76	10	13.16
Boxing			
Rossi et al[26]	27	3	11.11
Soler et al[27]	21	3	14.29
Total	48	6	12.5
Canoeing			
Rossi et al[26]	69	8	11.59
Soler et al[27]	162	10	6.17
Total	231	18	7.79
Cricket (fast bowling)			
Crewe et al[41]	46	15	32.61
Ranson et al[34]	28	12	42.86
Hardcastle et al[42]	22	12	54.54
Engstrom et al[43]	51	18	35.29
Foster et al[44]	82	9	10.98
Annear et al[45]	20	9	45
Elliott et al[46]	20	11	55
Total	269	86	31.97
Cycling			
Rossi et al[26]	95	13	13.68
Soler et al[27]	175	11	6.29
Total	270	24	8.89
Diving			
Rossi et al[26]	57	23	40.35
Soler et al[27]	8	0	0
Total	65	23	35.38
Equestrian			
Rossi et al[26]	83	5	6.02
Soler et al[27]	16	0	0
Total	99	5	5.05
Fencing			
Rossi et al[26]	143	19	13.28
Soler et al[27]	56	6	10.71
Total	199	25	12.56
Football (American)			
Jones et al[51]	104	5	4.81
Rossi et al[26]	400	65	16.25
McCarroll et al[52]	145	22	15.2
Soler et al[27]	13	0	0
Semon et al[53]	58	12	20.69
Iwamoto et al[54]	742	77	10.38
Ferguson et al[55]	12	6	50
Abe et al[56]	210	42	20
Total	1720	229	13.32
Golf			
Rossi et al[26]	38	2	5.26
Soler et al[27]	52	1	1.92
Total	90	3	3.33
Gymnastics			
Rossi et al[26]	673	112	16.64
Mohriak et al[62]	18	1	5.56
Kobayashi et al[31]	5	2	40
Toueg et al[11]	92	6	6.52
Sward et al[32]	52	8	15.38
Toueg et al[63]	93	6	6.45
Soler et al[27]	235	33	14.04
Bennett et al[64]	13	4	30.77
Kono et al[36]	49	4	8.16
Akimoto et al[37]	61	5	8.2
Jackson et al[65]	100	11	11
Total	1391	192	13.80
Handball			
Rossi et al[26]	42	3	7.14
Kobayashi et al[31]	1	0	0
Soler et al[27]	67	5	7.46
Total	110	8	7.27
Hockey (ice and field)			
Rossi et al[26]	170	13	7.64
Soler et al[27]	106	3	2.83
Donaldson et al[25]	25	11	44
Suzuki et al[28]	63	10	15.87
Total	364	37	10.16
Javelin			
Schmitt et al[66]	21	14	66.67
Martial arts			
Rossi et al[26]	64	10	15.62
Kobayashi et al[31]	3	1	33.33
Kobayashi et al[31]	2	1	50
Soler et al[27]	43	4	9.3
Kono et al[36]	38	4	10.53
Akimoto et al[37,c]	40	5	12.5
Akimoto et al[37,d]	49	5	10.2

(continued)

Table 2. (continued)

Sport	Sample Size	Athletes With at Least One Pars Interarticularis Defect	
		n	%
Kuroki et al[67]	21	7	33.33
Total	260	37	14.23
Motorcycling			
Rossi et al[26]	8	0	0
Mountaineering			
Soler et al[27]	63	1	1.59
Paddleball			
Soler et al[27]	20	2	10
Pole vaulting			
Rebella et al[68]	135	4	2.96
Rowing			
Maurer et al[69]	22	6	27.27
Rossi et al[26]	246	19	7.72
Soler et al[27]	77	13	16.88
Total	345	38	11.01
Rugby			
Rossi et al[26]	65	7	10.76
Kobayashi et al[31]	5	1	20
Soler et al[27]	40	2	5
Abe et al[56]	169	52	30.77
Total	279	62	22.22
Sailing			
Rossi et al[26]	128	22	17.18
Shooting			
Rossi et al[26]	76	8	10.52
Soler et al[27]	81	4	4.94
Total	157	12	7.64
Skating (ice)			
Rossi et al[26]	42	3	7.14
Skating (roller)			
Soler et al[27]	7	0	0
Skiing			
Rossi et al[26]	154	25	16.23
Rossi et al[26,e]	18	2	11.11
Soler et al[27]	77	6	7.79
Total	249	33	13.25
Soccer			
Kobayashi et al[31]	47	25	53.19
Sward et al[32]	31	2	6.45
Soler et al[27]	55	1	1.82
Murase et al[70]	160	14	8.75
Kono et al[36]	264	23	8.71
Akimoto et al[37]	320	28	8.75
Kyo et al[71]	37	18	48.65
Matsumoto et al[60]	60	19	31.67
Total	974	130	13.35
Sumo			
Nakagawa[72]	37	5	13.51
Swimming			
Rossi et al[26,f]	307	34	11.07
Kobayashi et al[31]	1	1	100
Engstrom et al.[43]	20	4	20
Soler et al[27]	176	18	10.23
Soler et al[27,g]	11	1	9.09
Kono et al[36]	55	10	18.18

(continued)

Table 2. (continued)

Sport	Sample Size	Athletes With at Least One Pars Interarticularis Defect	
		n	%
Akimoto et al[37]	117	12	10.26
Total	687	80	11.64
Table tennis			
Rossi et al[26]	1	0	0
Kobayashi et al[31]	4	3	75
Kono et al[36]	25	3	12
Akimoto et al[37]	34	4	11.76
Total	64	10	15.63
Tennis			
Rossi et al[26]	306	36	11.76
Kobayashi et al[31]	10	4	40
Sward et al[32]	30	3	10
Soler et al[27]	91	1	1.1
Maquirriain et al[33]	139	3	2.16
Alyas et al[73]	33	9	27.27
Rajeswaran et al[35]	98	29	29.59
Kono et al[36]	53	8	15.09
Akimoto et al[37]	67	9	13.43
Total	827	102	12.33
Track and field			
Rossi et al[26]	353	61	17.28
Rossi et al[26,h]	54	11	20.37
Kobayashi et al[31]	13	4	30.77
Soler et al[27]	685	61	8.91
Kono et al[36]	144	19	13.19
Akimoto et al[37]	206	23	11.17
Soler et al[27,i]	9	0	0
Soler et al[27,j]	90	7	7.78
Total	1554	186	11.97
Volleyball			
Rossi et al[26]	150	16	10.66
Kobayashi et al[31]	12	1	8.33
Soler et al[27]	70	7	10
Külling et al[12]	29	6	20.69
Akimoto et al[37]	53	2	3.77
Total	314	32	10.19
Weight lifting			
Rossi et al[26]	112	25	22.32
Kotani et al[74]	26	8	30.77
Soler et al[27]	85	11	12.94
Granhed et al[75]	13	2	15.38
Total	236	46	19.49
Wrestling			
Rossi et al[26]	80	20	25
Soler et al[27]	143	16	11.19
Sward et al[32]	30	2	6.67
Granhed et al[75]	32	4	12.5
Total	285	42	14.74

[a] Softball.
[b] Luge, not bobsledding.
[c] Judo.
[d] Kendo.
[e] Water skiing.
[f] Synchronized swimming and water polo.
[g] Synchronized swimming.
[h] Pentathlon/triathlon.
[i] Pentathlon.
[j] Triathlon.

Table 3. Classification of Pars Interarticularis Defects (Type IIa Spondylolisthesis) by Radiological and Pathological Features.[a]

Grade	Radiological Change	Pathological Change
0	Nil	Nil
1: Stress reaction	T2 signal abnormalities, with no pars defect on T1/T2 images	Bone marrow edema
2: Incomplete fracture	T2 signal abnormalities, with thinning, fragmentation, or irregularity of the pars interarticularis on T1 or T2 images	Bone marrow oedema with pars defect (incomplete fracture), but spondylolysis (cortical disruption) has not yet occurred
3: Complete fracture	Cortical disruption on T1/T2 images, with T2 signal abnormalities present	Cortical disruption (spondylolysis) has occurred and bone marrow edema is present, but reunion is still possible
4: Chronic complete fracture	Cortical disruption on T1/T2 images, with no T2 signal abnormalities present	Complete pars interarticularis fracture that has never reunited, with no associated bone marrow edema

[a] Spondylolysis is characterized by cortical disruption (grade 3 and 4 defects). Grade 1 and 2 defects are prespondylolytic.

interarticularis fractures that have not yet caused cortical disruption. Grade 3 refers to complete unilateral or bilateral cortical disruption (spondylolysis) with T2 signal abnormalities, representing acute complete fractures of the pars interarticularis. Grade 4 involves cortical disruption without abnormal T2 signals, representing old pars interarticularis fractures that have not united.[76]

The 5 grades of radiological changes can be used in various combinations to produce classifications of varying categories. Currently, most classifications use a 5-category system (grade 0, grade 1, grade 2, grade 3, grade 4), but the most reliable is likely to be a 3-category system (grade 0, grades 1-3, grade 4).[1] In any case, the classification used must achieve adequate accuracy while still being able to differentiate between the various stages of pars interarticularis damage.

The currently accepted classification of spondylolisthesis (Table 4) is based on etiology: type I (dysplastic), type II (isthmic), type III (degenerative), type IV (traumatic), type V (pathologic), and type VI (postsurgical).[2] Dysplastic spondylolisthesis occurs due to a congenital defect in the neural arch. Isthmic spondylolisthesis occurs due to a defect in the pars interarticularis, and has 3 subtypes: type IIa (spondylolytic, occurring due to stress fractures of the pars, such as by repetitive hyperextension and twisting), type IIb (repeated microtrauma occurs to the pars; as it heals, nonlinear forces cause the pars to elongate and thin, making it susceptible to future fracture), and type IIc (acute traumatic fracture of the pars). Degenerative spondylolisthesis occurs due to progressive

Table 4. Classification of Spondylolisthesis by Etiology.[a]

Type	Etiology	Pathogenesis
I	Dysplastic	Congenital defect in the neural arch
II	Isthmic	Pars interarticularis defect
IIa		Stress fracture of the pars (spondylolysis)
IIb		Repeated microtrauma and nonlinear forces cause elongation of the pars
IIc		Acute traumatic fracture of the pars
III	Degenerative	Degeneration of the facet joint complex (capsule, ligaments)
IV	Traumatic	Acute traumatic fracture of posterior column, but not the pars
V	Pathologic	Infection, neoplasm, endocrine disorder, or other pathology causes vertebral instability
VI	Postsurgical	Postsurgical lumbar instability

[a] Pars interarticularis defects in athletes correspond to type IIa spondylolisthesis.

degeneration of the facet joint complex (eg, capsule, ligaments). Traumatic spondylolisthesis is caused by acute spinal trauma, which leads to a posterior column fracture, but not pars fracture. Pathologic spondylolisthesis occurs due to a pathological process such as infection, malignancy, or endocrine disorder. Postsurgical spondylolisthesis occurs due to postsurgical lumbar instability. The focus of this review is pars interarticularis defects in athletes (type IIa spondylolisthesis).

Spondylolisthesis may also be classified into developmental and acquired causes.[77,78] Developmental spondylolisthesis corresponds to the dysplastic and isthmic etiologies. Acquired spondylolisthesis corresponds to the traumatic, pathologic and postsurgical etiologies.

Risk Factors

Known risk factors for pars interarticularis defects include childhood/adolescence, male sex, race, particular sports, and other lifestyle choices that lead to repetitive hyperextension and rotation of the lumbar spine.[24,79-81] Possible risk factors include family history and spina bifida occulta.[4,5]

Spondylolysis is more common in children since their bones are still in the growth stages, with weaker osteochondral junctions and thinner cortices. Children also participate more frequently in physical activity than adults, not allowing sufficient time for recovery from microtrauma.[79] Another reason that children develop pars defects more commonly than adults is that children can develop injuries due to unaccustomed loads. In other words, a child who is an expert at soccer may be injured playing a sport they are not accustomed to (eg, tennis), even if tennis presents similar loading forces on the body. Contrastingly, adults do not often develop injuries from unaccustomed loads.[82] Males develop spondylolysis more commonly than females with a ratio of 2:1,[24,80] but females are more likely to develop severe spondylolisthesis.[83] Incidence in white males is 6.4%, black males is 2.8%, white females is 2.3%, and black females is 1.1%.[24] Eskimos have the highest

Table 5. Studies in Which Treatment Modality and Return-to-Play Time Were Reported.[a]

	No. of Athletes With Pars Defects	Conservative Therapy	Surgical Therapy	Returned to Previous Level of Play	Retired
Donaldson et al[25]	11	11	0	10	1
Hardcastle et al[42]	12	9	3	Not reported	5
Engstrom et al[43]	18	18	0	18	0
McCarroll et al[52]	22	21	1[b]	22	0
Jackson et al[65]	11	11	0	11	0
Total	74	70	4	61	6

[a] Five studies reported treatment modality for participants found to have spondylolytic defects.
[b] Intervertebral disc surgery, not pars interarticularis surgery.

incidence,[81] supporting a genetic predisposition to spondylolysis.[5] Spondylolysis occurs more commonly in athletes[81] due to repetitive hyperextension and twisting movements of the lumbar spine, as previously mentioned.

Family history and spina bifida occulta (SBO) are possible risk factors for pars defects. Sixty-three percent of L5 spondylolysis exhibits terminal-stage defects despite young skeletal age, while most L3/L4 spondylolysis exhibits early-stage defects.[4] This suggests that certain risk factors (possibly genetic) predispose to L5 spondylolysis, but not to spondylolysis at other lumbar levels. Another possibility could be the fact that L5 experiences more stress than other lumbar levels. Of the patients studied by Sakai et al,[4] 93% of L5 spondylolysis patients suffered from SBO, while 0% of the L3/L4 spondylolysis patients suffered from SBO. This indicates a strong correlation between L5 spondylolysis and SBO, either due to a common genetic predisposition, with autosomal dominant inheritance[84,85] or because SBO directly predisposes to spondylolysis. This is supported by Yamada et al,[5] who studied 3 brothers—2 of whom were twins—who all developed lumbar spondylolysis and concomitant SBO. It is postulated that defective osteogenesis during the growth period leads to both SBO and spondylolysis.[4]

Athletes have been shown to have larger sacrohorizontal angles (angle between the lumbar vertebra and upper endplate of the sacrum) than nonathletes,[32] leading to increased lumbar lordosis. Increased lumbar lordosis increases the risk of pars interarticularis defects due to greater shear and compressive forces on the lumbar spine.[86,87] Lumbar lordosis may also be increased during adolescence, when rapid bone growth causes tightness of the iliopsoas and thoracolumbar fascia.[19,88] Additionally, specific sporting positions may be associated with a higher body mass index, such as defensive players in rugby, and higher body mass index is associated with increased lumbar lordosis.[89,90]

Investigations

Imaging modalities are the mainstay of diagnosis in pars defects since clinical assessment is unreliable,[8] but there is no universally agreed algorithm for the diagnostic workup.[91] A trial of conservative management may be attempted before undertaking any imaging investigations, if the lower back pain is of recent onset and there is insufficient clinical suspicion for spondylolysis.[92] Oblique and lateral X-rays of the lumbar spine may be performed as an initial investigation, with lucency of the pars interarticularis indicating a pars defect. The pathognomonic sign on oblique lumbar X-ray is the "Scotty dog collar" sign (the "Scotty dog" is the appearance of the normal spine, and the "collar" indicates the nondisplaced pars fracture).[93] Computed tomography (CT), MRI, and single photon emission computed tomography (SPECT) are all more sensitive than X-ray[8] and allow greater appreciation of the spinal anatomy, but their exact role in the diagnostic workup remains debated. Generally, early-stage disease is best detected by SPECT, but MRI has an increasingly positive role in this area.[93] CT is excellent for assessing more progressive disease, determining fracture size and extent, and providing a baseline on which to assess adequacy of healing.[93] In all cases, a high index of suspicion is required to detect less common types of pars defects, such as unilateral spondylolysis, spondylolysis of the upper lumbar vertebra (L1-L3), multilevel spondylolysis, and early stage lesions in which cortical disruption has not yet occurred.[94,95]

Management

There is no gold standard protocol for the management of pars interarticularis defects. Further studies are required to compare conservative therapy to surgical therapy, and to compare Buck's repair with Scott's wiring technique, Morscher technique and other novel surgical techniques involved in these defects. The literature summatively suggests a mean return-to-play time of 3.7 months for conservative therapy, and 7.9 months for operative therapy.[96]

Only 5 studies in this review reported the management instituted for their participants,[25,42,43,52,65] and these were all case series (Table 5). Of 74 players with spondylolysis in these studies, 70 (94.59%) underwent conservative treatment and 4 (5.41%) underwent surgical treatment. Sixty-one (82.43%) returned to their previous level of play, 6 (8.11%) retired,[25,42] and the disposition of the final 7 was not reported.[42]

Conservative Therapy. Management of pars interarticularis defects typically begins with rest, orthosis, and physical therapy[97]; transcutaneous electrical stimulation has also been

Table 6. Buck's Repair, Scott's Wiring Technique, and Morscher Technique Are Used for Surgical Fixation of Pars Defects.

Buck's repair	Screw fixation of the pars interarticularis to compress the defect
Scott's wiring technique	A wire is passed through the transverse processes of the vertebrae, then wrapped around the spinous processes. This stabilizes the defect and allows bony repair
Morscher technique	Bone graft fills the defect then screws are inserted into the superior articular processes. A hook hangs over the lamina and is secured by a lock

described.[98,99] The orthosis prevents hyperextension and twisting of the spine, while physical therapy strengthens and stretches the muscles of the trunk and lower limbs.[100] Specific physical therapies include foam rolling, isometric strengthening exercises and exercises aimed at stretching the iliopsoas, piriformis, rectus femoris, and spinal erector muscles.[86] Conservative management is effective for unilateral and bilateral lesions, allowing 90% of patients to return to baseline activity levels within 6 months.[101] Unilateral lesions are the most likely to heal with conservative treatment, followed by bilateral lesions then pseudobilateral lesions (asymmetrical tracer uptake), but this is a radiological distinction since nonunion appears to have no effect on the overall outcome and ability to resume sport.[101] At 11-year follow-up, only 22% of athletes are limited by pars defects treated conservatively,[102] with limitation defined as any alteration to recreational activity.

Orthoses (Braces). There are 2 types of orthoses: (*a*) thoracolumbosacral orthoses and (*b*) lumbosacral orthoses. For each of these, they may be flexible orthoses which simply decrease the activity of the adjacent paraspinal and abdominal muscle, or they may be rigid orthoses which restrict movement primarily in the sagittal plane, thus preventing hyperextension of the spine. Rigid thoracolumbosacral orthoses are thus more commonly used[20,96,103] since they theoretically allow greater spinal rest and bony healing, but data suggests equally effective results and similar return-to-play time with lumbosacral orthoses.[96]

Surgical Therapy. Surgical intervention is indicated if there is failure of conservative treatment after 6 months, persistent back pain after 9 to 12 months, or pars pseudoarthrosis (nonunion after 9-12 months).[97 104,105] Spondylolisthesis more than 50% in those who have not reached skeletal maturity, neurological deficit and radiculopathy are relative indications for surgical management.[104] There are several methods of surgical management. A Buck's repair involves screw fixation of the pars interarticularis.[106] Variations include the Scott wiring technique[107] and the Morscher technique[108] (Table 6). Minimally invasive techniques have also recently been described, with quicker postoperative recovery but greater technical difficulty.[97,109-112] Currently, there are no randomized controlled trials comparing different techniques of pars repair.

Comparison of surgical outcomes is difficult as surgeons use various intraoperative and postoperative regimens in treating pars defects. Most surgeons immobilize patients for 3 months postoperatively in a rigid lumbosacral orthosis, then allow graduated return to activity.[104,113] Most surgeons allow resumption of sport after 6 months postoperatively in noncontact sports, and 12 months postoperatively in contact sports, although some surgeons advise patients to never recommence contact sports.[104,113] Approximately 50% of surgeons advise patients never to recommence collision.[113] In all cases, patients must be fully rehabilitated before returning to athletic activity. This involves no pain with sport-specific activity, as well as full strength and range of motion. Naturally, some athletic activities can never be recommenced using these criteria since they require extreme ranges of motion that cannot be recommenced after surgery, such as dancers who require lumbar hyperextension for their vocation.[112,113] The main factors ultimately influencing return to athletic activity are symptomaticity and time from surgery; it is unclear whether radiographic appearance is one of the least important[104,112,113] or most important factors[112,114] influencing return to athletic activity.

Prevention

Prevention of pars defects by targeting modifiable risk factors may be an important adjunct against this disorder. For example, notifying cricketers of the increased risk using a mixed front-and-side action or tennis players of the increased risk using a topspin serve may lead to decreased incidence of pars defects. Coaches should also be trained in the provision of safe training routines, and high-risk maneuvers should be reserved for competitive play and not employed during training sessions.

Limitations

This study elucidated several areas of weakness in the current literature on pars interarticularis defects in athletes. Specifically, there were multiple differences in the study protocols of the 42 articles from which quantitative data was collected. The studies were always retrospective or prospective cohort studies; performing a randomized controlled trial for this clinical question would be unethical and practically impossible, since it would involve preventing certain individuals from performing physical activity for several years. Furthermore, there was considerable heterogeneity in the populations selected for testing with some studies only considering athletes of a particular sex, professional level or age. A number of studies only considered symptomatic patients whereas others studied all patients regardless of their symptomaticity. The imaging modalities used to detect pars interarticularis defects were also inconsistent, with some studies only using X-ray or CT imaging, which is inferior to MRI for detecting prelytic lesions. Finally, in athletes in whom more than one defect was detected the defect of highest grade was counted, such that the present study does not differentiate between patients with single and multiple defects. Future studies on this topic would benefit from prospectively using highly sensitive imaging (MRI) to detect all lesions in a symptomatic population of well-defined athletic experience so that better comparisons can be made between sports subtypes.

Conclusion

The current medical literature provides good evidence that the incidence of pars interarticularis defects is higher in the athletic population, with the highest incidence in diving, cricket, and baseball/softball. There remains no gold standard protocol for the management of pars interarticularis defects. Further studies are required to compare conservative therapy (rest, orthosis, physical therapy) to surgical therapy, and to compare Buck's repair with Scott's wiring technique, Morscher technique, and other novel surgical techniques involved in these defects.

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