论著·军事医学

高寒环境暴露后大鼠不同强度肠道冲击伤的比较研究

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[摘要] **目的** 对高寒环境暴露后大鼠的不同强度肠道冲击伤进行比较,并对其损伤特点进行初步探究。**方法** 选取体质量为200~250 g的2月龄健康雄性SD大鼠60只,按随机数字表法将大鼠分为6组(n=10):空白对照组、低温对照组、5.0 MPa冲击对照组、低温+4.0 MPa冲击组、低温+4.5 MPa冲击组和低温+5.0 MPa冲击组。低温+冲击组在-10 ℃低温环境预处理30 min后,立即采用BST-Ⅰ型生物激波管以不同驱动压冲击大鼠腹部致肠道损伤。检测致伤后3、8、24 h大鼠血清白介素-6(interleukin-6,IL-6)、肿瘤坏死因子-α(tumor necrosis factor-α,TNF-α)、肠脂肪酸结合蛋白(intestinal-fatty acid binding protein,I-FABP)、二胺氧化酶(diamine oxidase,DAO)水平,并记录大鼠24 h存活情况。实验24 h后,麻醉处死大鼠并解剖,观察肠道损伤特点并行病理学检测。通过比较各组差异,研究高寒环境暴露后大鼠不同强度肠道冲击伤特点。**结果** 较空白对照组,其余各组大鼠均出现不同程度肠道损伤,且低温+冲击伤组大鼠肠道更易出现水肿及损伤,低温+5.0 MPa冲击组存活率明显下降(P<0.05)。病理学及血清学研究发现,低温+冲击组大鼠肠黏膜在高寒环境和冲击伤双重作用下,表现出肠黏膜出血、水肿、黏膜固有层崩解等;肠黏膜损伤指标及肠道炎症因子也较空白对照组明显升高,且随冲击强度增加,组间差异亦有统计学意义(P<0.05)。**结论** 低温暴露叠加腹部冲击波损伤后,大鼠死亡率明显增加,肠道损伤表现为血清学指标及肠道炎症因子升高,不同程度的肠壁水肿、黏膜下出血等,且损伤严重程度与冲击强度相关,随冲击强度增加而加重。

[关键词] 低温暴露;肠道损伤;冲击伤

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Intestinal blast injuries after different intensity waveshock in rats exposure to extremely cold environment: a comparative study

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[**Abstract**] **Objective** To compare the intestinal injury induced by different intensities of waveshock in rats exposed to extremely cold environment and to preliminarily explore the characteristics of the injury. **Methods** Sixty healthy male SD rats (2 months old, weighing 200~250 g) were randomly divided into 6 groups(n=10): blank control group, low-temperature control group, 5.0 MPa shock control group, and low-

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temperature+4.0, 4.5 and 5.0 MPa shock groups. The rats in the experimental groups were pre-treated in a −10 ℃ low-temperature environment for 30 min and then subjected to intestinal injury by using BST-I biological shock tube with different driving pressures. At 3, 8, and 24 h after injury, the serum levels of IL-6, TNF-α, intestinal fatty acid binding protein (I-FABP), and diamine oxidase (DAO) were detected, and the survival of rats within 24 h was recorded. At 24 h after injury, the rats were anesthetized and dissected, the characteristics of intestinal injury were observed, and pathological examination was performed. The differences of intestinal injury were compared among the 6 groups to explore the characteristics of intestinal injuries after different intensities of shockwave in rats after exposure to extremely cold environment. **Results** Compared with the blank control group, the other 5 groups exhibited different severities of intestinal injury, and the rats in the low-temperature+ different shock groups were more prone to intestinal edema and trauma. The mortality rate was significantly increased in the low-temperature+5.0 MPa shock group ($P<0.05$). Pathological and serological studies found that dual effects of very cold environment and blast injury resulted in intestinal mucosal hemorrhage, edema, and disintegration of lamina propria in the experimental rats. The indicators of intestinal mucosal injury and intestinal inflammatory factors were also significantly increased when compared with the blank control group, and significant differences were among the groups with increment of shock intensity ($P<0.05$). **Conclusion** Exposure to very cold environment combined with abdominal blast injury increases mortality rate in rats, manifested by elevated serological indicators and intestinal inflammatory factors, as well as varying severities of intestinal wall edema and submucosal bleeding. Furthermore, the severity of the injury is positively related to the impact intensity, with worsened as the impact intensity increasing.

[**Key words**] cold exposure; intestinal injury; blast injury

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冲击损伤是指恐怖袭击、燃气及化学危险品爆炸、交通事故碰撞等事件发生时产生的冲击波造成的冲击载荷损伤,会对人体组织器官造成极大危害,甚至危及生命。冲击波在其高速运动中会形成好似双层球形的2个区域:外层为压缩区,内层为稀疏区。压缩区内的空气因被压缩而超过正常大气压,形成超压。冲击波在其高速运行中所产生的冲击力称为动压。在稀疏区内,空气因压缩时所产生的真空作用而高度稀疏,该区内的空气低于正常大气压,称为冲击波的负压。冲击波主要通过超压和动压的作用使人体致伤,负压也能引起明显的损伤[1-2]。

爆炸引发的冲击波对人体的直接损伤称为原发性冲击损伤(primary blast injury, PBI),PBI以空腔器官为主,最易导致肺损伤,主要表现为肺泡-毛细血管损伤和随之而来的肺出血、肺水肿[3]。同时,小肠也是易受损的空腔脏器。由于十二指肠后方是脊柱,当腹部受到暴力撞击时,外力与脊柱之间形成挤压,冲击伤暴力挤压下可直接造成十二指肠损伤。此外,空肠上段、回肠末段是肠管活动部与固定部的移行地带,冲击伤导致的突然的加减速运动易导致肠系膜及其血管撕裂进而导致该段肠管水肿、出血或缺血坏死[4]。

肠道是消化和免疫调节的重要器官,充当生物体与其外部环境之间的直接接口,在营养吸收和免疫调节中起重要作用。冷应激会破坏肠道结构,如改变绒毛长度及肌肉层厚度、降低肠道通透性等,导致动物肠黏膜上皮损伤,肠黏膜屏障结构受损,从而引起肠道功能紊乱[5]。我国青藏高原、蒙新高原、黑龙江北部以及云贵高原和黄土高原的部分区域均为高寒地区,尤其是新疆部分地区冬季气温可达−10~−30 ℃,甚至更低。高寒环境对当地居民生活和军事活动的展开,尤其是医疗救援提出了特殊要求。研究高寒环境暴露下的肠道冲击波损伤特点对于该类疾病的救治具有重要的指导意义,但目前国内外相关的研究较少。因此,本研究利用冷柜模拟高寒环境,用激波管模拟稳定可控的爆炸冲击波[6],通过二者结合,从一般情况、大体改变、病理学、血清学等方面,对成年大鼠高寒环境暴露后,不同强度肠道冲击波损伤进行比较,以期为低温肠道冲击伤的病理特点、康复机制、诊断方法、治疗手段等方面的研究奠定实验基础。

1 材料与方法

1.1 实验动物

选取60只体质量为200~250 g的2月龄健康雄性SD大鼠(陆军军医大学大坪医院实验动物中心提供),实验动物自由摄食饮水,适应性喂养4 d后,用于后续实验。动物饲养环境:温度为室温(28 ℃),相对湿度

45%~60%。本实验经陆军第948医院实验动物福利伦理审查委员会审核通过(20241101),实验过程符合国家和单位有关实验动物管理和使用规定。

1.2 实验方法

1.2.1 实验动物分组及模型制作 将60只SD大鼠按随机数字表法分为6组:空白对照组、低温对照组、5.0 MPa冲击对照组、低温+4.0 MPa冲击组、低温+4.5 MPa冲击组和低温+5.0 MPa冲击组,每组10只。致伤前一晚,所有实验大鼠禁食,但自由饮水。低温预处理时,大鼠于实验开始后先行置于-10 ℃环境(采用2021款志高BD/BC-58A118D一级冷柜模拟低温环境)内30 min,每隔10分钟检查1次大鼠情况,持续30 min,观察指标包括精神状态、排泄情况、存活情况等。冷暴露后立即用3%戊巴比妥钠(50 mg/kg)麻醉,并单独固定到特制的铁丝鼠笼(肺防护,排除因肺部损伤导致死亡)中,固定于距离BST-Ⅰ型生物激波管(陆军特色医学中心自主研制)末尾端挡板10 cm处,闭口加挡板;实验组高压段驱动压力值分别设定为4.0 MPa、4.5 MPa、5.0 MPa,采取腹部朝向冲击波波源体位。致冲击伤后,迅速取出鼠笼,室温(28 ℃)饲养,对致伤后大鼠的生理情况进行观察,并于冲击后3、8、24 h内眦静脉取血,以4 ℃、3 500 r/min离心15 min后,检测血清学指标。记录24 h大鼠存活情况,死亡大鼠立即行解剖,存活大鼠24 h后采用3%戊巴比妥钠(50 mg/kg)麻醉后脊椎脱臼法处死,并立即解剖,观察腹部脏器损伤情况,并进行病理分析。低温对照组在-10 ℃环境暴露30 min后室温饲养24 h,不行冲击波损伤;5.0 MPa冲击对照组直接在5.0 MPa高压下冲击,不行高寒环境暴露;空白对照组室温饲养24 h;其余处理同低温+冲击各组。

1.2.2 24 h存活情况 观察大鼠造模前后一般情况,如精神状态、皮肤颜色改变、排泄情况等。记录各组大鼠死亡数量及死亡时间,计算24 h存活率,绘制生存曲线。

1.2.3 大鼠肠道组织大体观察 各组大鼠实验24 h后,3%戊巴比妥钠(50 mg/kg)溶液麻醉大鼠,脊椎脱臼法处死后,剖腹整体取出肠道,观察并记录各组大鼠肠道损伤情况。

1.2.4 大鼠肠道组织病理学检测及损伤评估 各组大鼠肠道组织固定48 h后,脱水、包埋,制作石蜡切片,行HE染色。将切片置于光镜下,观察小肠组织病理改变情况。参照Chiu法[7]评估小肠黏膜上皮损伤程度(0~5级),并赋予相应的分值(0~5分),对小肠损伤情况进行评分:0分为正常黏膜绒毛结构;1分为肠黏膜绒毛顶端上皮下间隙增宽,伴毛细血管充血,2分为绒毛上皮下间隙进一步扩大,上皮抬高与固有膜剥离,3分为绒毛两边上皮成块脱落,4分为上皮完全脱落,仅存固有膜结构,毛细血管充血明显,5分为黏膜固有膜崩解,出现出血和溃疡。

1.2.5 血清学检测 二胺氧化酶(diamine oxidase,DAO)主要存在于肠黏膜上层绒毛细胞胞质内,肠黏膜细胞受损后释放入血,被认为是肠黏膜损伤早期的诊断指标,具有较好的临床价值[8]。肠脂肪酸结合蛋白(intestinal-fatty acid binding protein, I-FABP)是一种仅存在于胃肠道黏膜的细胞质蛋白,正常情况下血液中含量较低,肠黏膜缺血时I-FABP比DAO更早释放入血,故而在早期肠黏膜屏障受损时,其血浆含量较高[9],且损伤越重,血浆I-FABP水平越高[10]。肿瘤坏死因子-α(tumor necrosis factor-α,TNF-α)是机体在受到各种外界因素作用后,产生最快、到达高峰时间最短的炎症介质。TNF-α可诱导内皮细胞和巨噬细胞释放血清白介素-6(interleukin-6,IL-6),而IL-6随后又刺激其他细胞因子的生物合成,并反过来增强组织细胞对TNF-α的敏感性[11]。各组大鼠于冲击后3、8和24 h内眦静脉取血,以4 ℃、3 500 r/min离心15 min后,检测肠黏膜屏障损伤指标I-FABP、DAO及肠道损伤后炎症因子IL-6、TNF-α,通过检测这4种血清学指标的改变,对比各组大鼠肠道损伤情况。

1.3 统计学分析

采用SPSS 29.0软件进行统计学分析。血清I-FABP、DAO、IL-6、TNF-α等计量资料以$\bar{x}\pm s$表示,多组间独立样本比较采用单因素方差分析,进一步两两比较采用LSD-t法;死亡率等计数资料用频数和百分比(%)表示,进一步两两比较采用拓展的t检验法。$P<0.05$为差异具有统计学意义。

2 结果

2.1 各组大鼠低温暴露时间及冲击波强度物理参数比较

分别记录各组大鼠-10 ℃环境暴露时间及使用不同高压段驱动压力值冲击时的超压峰值、正向持续时间、正向冲量等冲击波强度物理参数,比较各组大鼠致伤因素的差异(表1)。

2.2 各组大鼠24 h存活情况及脏器损伤情况

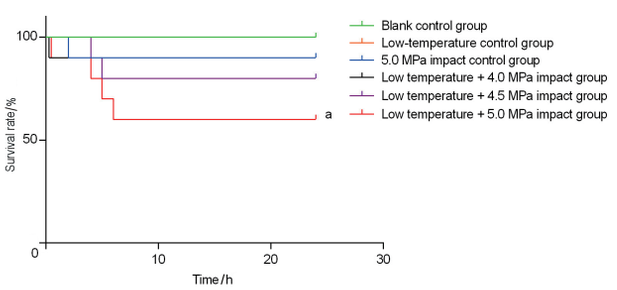
各组大鼠体质量组间比较差异无统计学意义($t=1.766$,$P=0.171$)。空白对照组大鼠实验期间活动自如,未见异常。低温对照组及低温+冲击组大鼠在-10 ℃环境暴露30 min后,大多表现为皮肤苍白、安静,抱团,活动明显减少,且排尿增加,但均能存活。各组大鼠在冲击波致伤后均出现呼吸急促、心率加快。

表1 各组大鼠低温暴露时间及冲击波强度物理参数(n=10)

Table 1 Cold exposure time and physical parameters of shockwave intensity for each group of rats (n=10)

Group	Exposure time to a −10 ℃ low-temperature environment /min	Pressure value at the high-pressure end /MPa	Overpressure peak /kPa	Positive duration /s	Positive impulse / kPa. s	Impact position
Blank control group	0	–	–	–	–	–
Low-temperature control group	30	–	–	–	–	–
5.0 MPa impact control group	0	5.0	364.04	0.05480	6.160 829 32	Abdomen facing the source of the impact
Low-temperature + 4.0 MPa impact group	30	4.0	260.88	0.05248	4.310 122 25	Abdomen facing the source of the impact
Low-temperature + 4.5 MPa impact group	30	4.5	350.12	0.05112	5.779 543 35	Abdomen facing the source of the impact
Low-temperature + 5.0 MPa impact group	30	5.0	363.34	0.05674	6.159 338 47	Abdomen facing the source of the impact

5.0 MPa冲击对照组大鼠,冲击后2 h死亡1只,解剖见其脾脏破裂、肠系膜血肿。低温+冲击组大鼠冲击后24 h内随冲击强度的增加;死亡率有所增加,低温+4.0 MPa冲击组大鼠,冲击后20 min死亡1只,解剖见其肝脏破裂、肠壁轻度水肿,腹腔出血;低温+4.5 MPa冲击组大鼠,冲击后4 h死亡1只,解剖见其脾破裂、肠系膜血肿;5 h死亡1只,解剖见其局部肠壁水肿、浆膜下血肿等;低温+5.0MPa冲击组大鼠,冲击后30 min后死亡1只,解剖见其鼻腔出血、肺部分淤血、肠道血肿,4 h死亡1只,解剖见其脾破裂、肠道水肿,系膜血肿形成,胃黏膜出血,5 h死亡1只,解剖见肠道水肿明显,局部血肿,6 h死亡1只,解剖见小肠水肿、盲肠黏膜下出血、睾丸血肿。冲击后所有大鼠体表未见明显伤痕。低温+5.0 MPa冲击组大鼠存活率与空白组对照组比较差异具有统计学意义(P<0.05,图1)。



a: P<0.05, in comparison with the blank control group

图1 各组大鼠24 h生存曲线图(n=10)

Figure 1 Survival curves of rats in 6 groups at 24 h (n=10)

2.3 各组大鼠小肠组织大体观察结果

空白对照组及低温对照组大鼠24 h解剖结果显示,肠道未见明显损伤。冲击组大鼠均出现不同程度的肠道损伤。5.0 MPa冲击对照组肠道出现部分系膜及肠壁血肿,水肿较轻;低温+4.0 MPa冲击组大鼠肠道出现局部水肿,部分肠壁血肿;低温+4.5 MPa冲击组大鼠肠系膜血肿、出血,肠壁中度水肿、局部肠壁血肿;低温+5.0 MPa冲击组大鼠肠壁重度水肿,黏膜下出血(图2)。

2.4 各组大鼠肠道病理观察结果

光镜下,低温对照组大鼠肠道下层出现轻度水肿及炎细胞浸润,绒毛长度变短;低温+冲击组大鼠主要表现为不同程度的肠道黏膜及黏膜下层水肿,炎性细胞浸润,上皮细胞变性、坏死,绒毛变短,稀疏或消失。低温+4.0 MPa冲击组及低温+4.5 MPa冲击组可见肠绒毛下间隙扩大,上皮抬高与固有膜脱离等;低温+5.0 MPa冲击组可见毛细血管充血,黏膜固有层崩解,溃疡形成等,肠黏膜损伤较5.0 MPa冲击对照组明显加重(图3、表2)。

2.5 低温暴露叠加腹部冲击波损伤后肠黏膜损伤指标血清DAO及I-FABP升高

实验发现,冲击后3 h开始,除空白对照组外,各组大鼠血清DAO及I-FABP均较空白对照组升高,提示肠道黏膜损伤,且冲击强度越大,升高越明显;低温+冲击组较低温对照组、低温+5.0 MPa冲击组较5.0 MPa冲击对照组升高更为明显(P<0.05)。但检测24 h DAO及I-FABP显示,低温对照组与空白对照组差异无统计学意义,考虑为肠黏膜自我修复所致(图4)。

2.6 低温暴露叠加腹部冲击波损伤后大鼠血清炎症因子TNF-α及IL-6升高

实验发现,冲击后3 h开始,除空白对照组外,各组大鼠血清TNF-α及IL-6均较空白对照组升高,提示肠黏膜出现炎症反应,且冲击强度越大,升高越明显;低温+冲击组较低温对照组、低温+5.0 MPa冲击组较5.0 MPa冲击对照组升高更为明显(P<0.05,图5)。

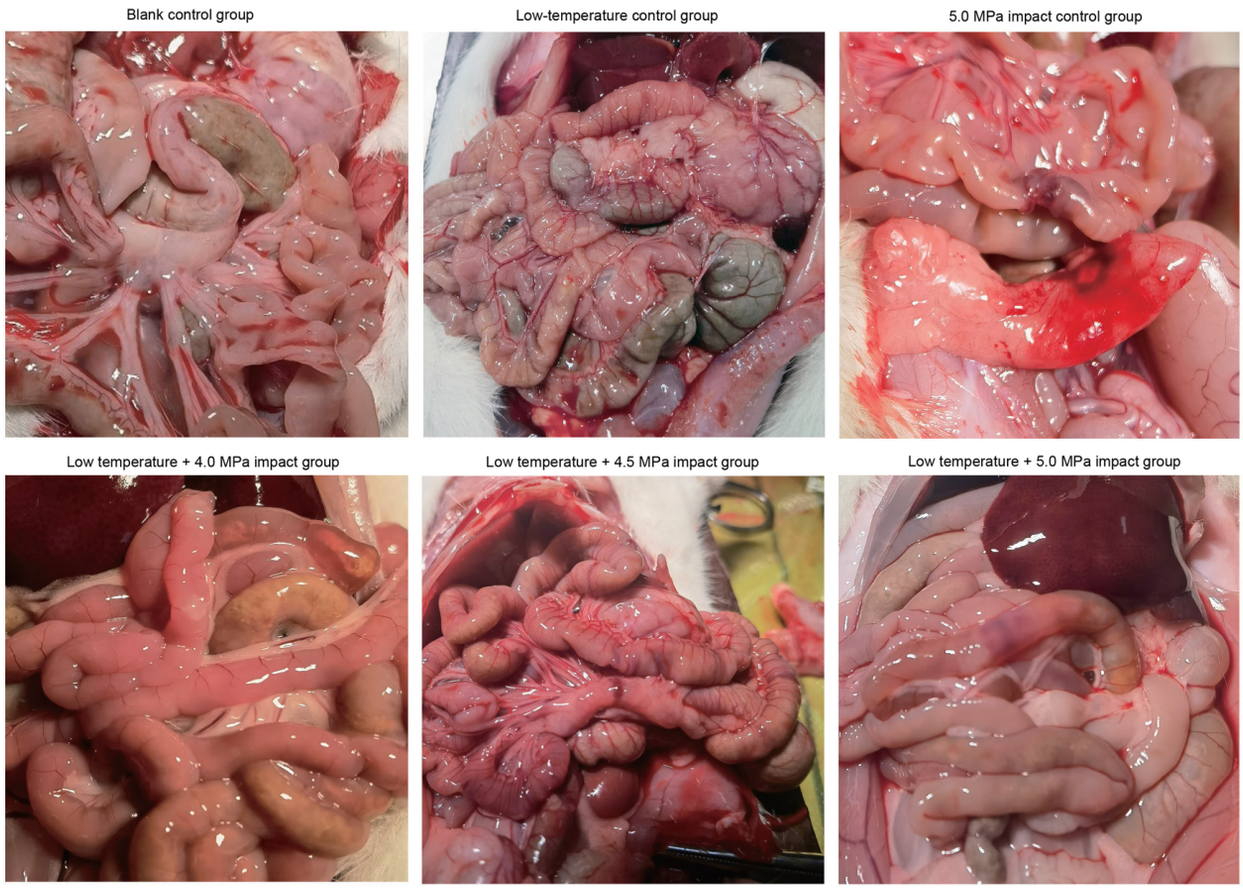


图2 大体解剖后各组大鼠肠黏膜损伤情况解剖图

Figure 2 Gross anatomical images showing various intestinal mucosal injuries in different groups of rats

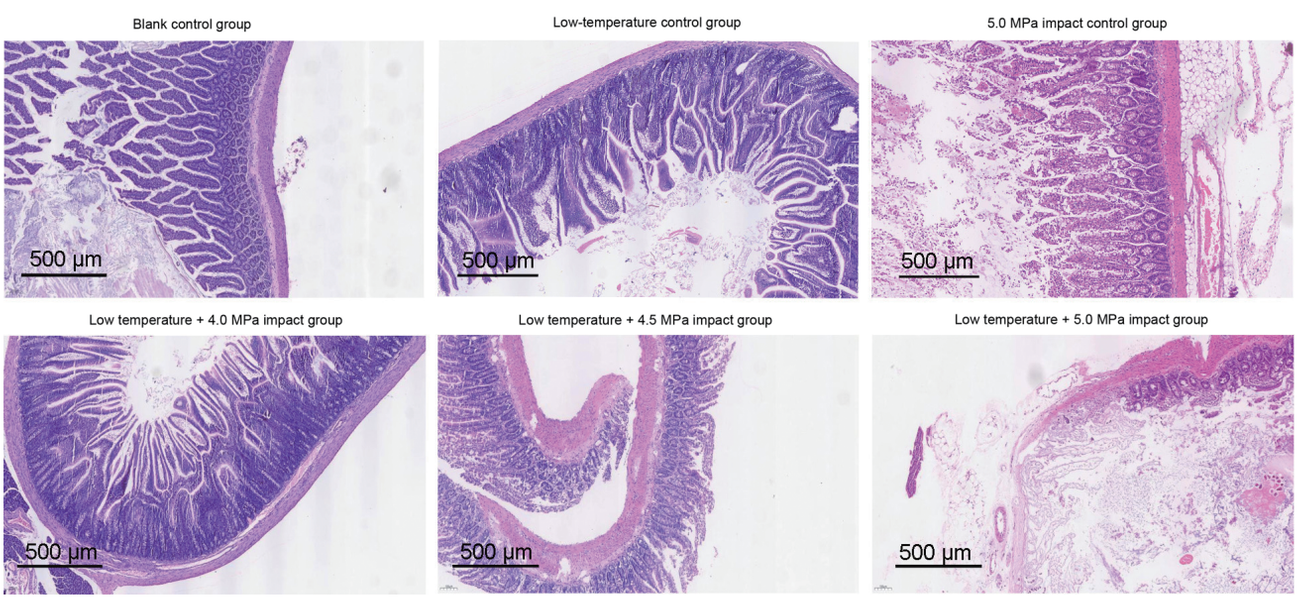


图3 采用HE染色检测各组大鼠肠黏膜损伤的病理图

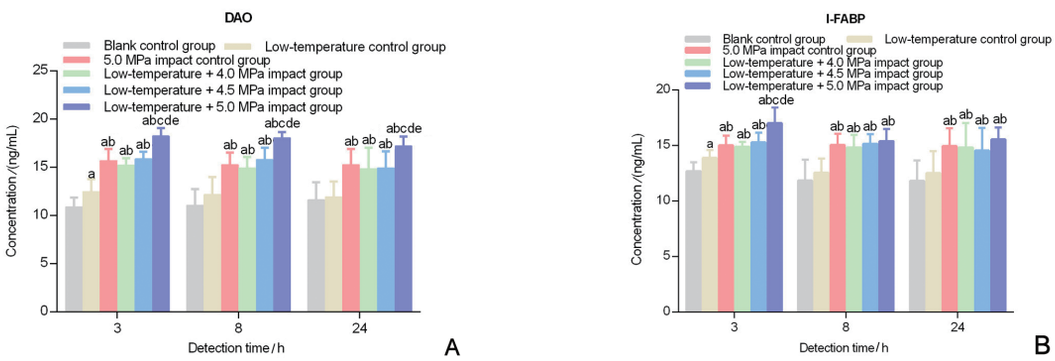
Figure 3 HE staining displaying the pathological images of intestinal mucosal damage in different groups of rats

表2 采用Chiu法病理学评分测量各组大鼠肠黏膜损伤评分表(n=10)

Table 2 Chiu's pathological scores of intestinal mucosal injury in each group of rats (n=10)

Group	Pathological Scores						P
	0	1	2	3	4	5	
Blank control group	10	0	0	0	0	0	–
Low-temperature control group	6	4	0	0	0	0	
5.0 MPa impact control group	0	1	5	3	1	0	<0.05[ab]
Low-temperature + 4.0 MPa impact group	0	5	5	0	0	0	<0.05[ab]
Low-temperature + 4.5 MPa impact group	0	2	4	3	1	0	<0.05[ab]
Low-temperature + 5.0 MPa impact group	0	0	2	2	3	3	<0.05[abc]

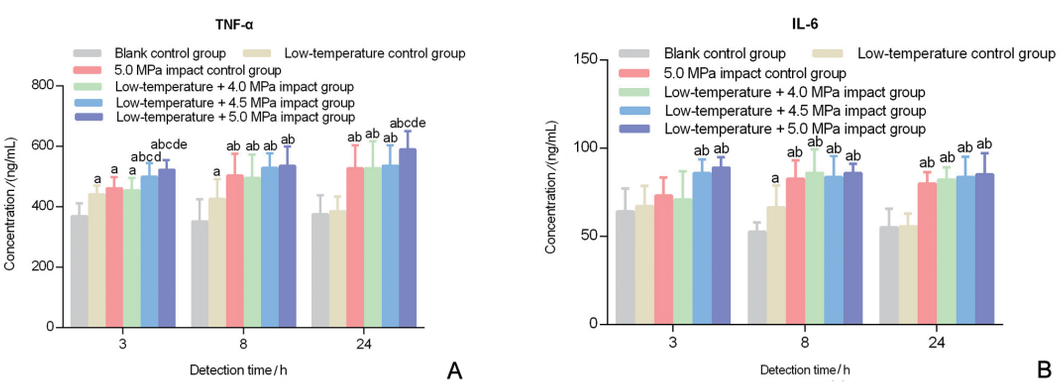
a: P<0.05, compared with the blank control group; b: P<0.05, compared with the low-temperature control group; c: P<0.05, compared with the 5.0 MPa impact control group



a: P<0.05, in comparison with the blank control group; b: P<0.05, in comparison with the low-temperature control group; c: P<0.05, in comparison with the 5.0 MPa impact control group; d: P<0.05, in comparison with the low-temperature+4.0 MPa impact group; e: P<0.05, in comparison with the low-temperature+4.5 MPa impact group.

图4 血清学方法检测各组大鼠3、8和24 h血清DAO(A)及I-FABP(B)浓度变化(n=10)

Figure 4 Serological detection showing the changes in serum DAO(A) and I-FABP(B) concentrations in each group of rats at 3, 8 and 24 h after injury (n=10)



a: P<0.05, in comparison with the blank control group; b: P<0.05, in comparison with the low-temperature control group; c: P<0.05, in comparison with the 5.0 MPa impact control group; d: P<0.05, in comparison with the low-temperature+4.0 MPa impact group; e: P<0.05, in comparison with the low-temperature+4.5 MPa impact group.

图5 血清学方法检测各组大鼠3、8和24 h血清TNF-α(A)与IL-6(B)浓度变化(n=10)

Figure 5 Serological detection showing the changes in serum TNF-α(A) and IL-6(B) concentrations in each group of rats at 3, 8 and 24 h after injury (n=10)

3 讨论

冲击伤由爆炸产生的冲击波超压、动压及负压引起,主要造成肺脏、听器、胃肠道等含气器官损害。我国高寒地区总面积占国土面积的30%~35%,平时的工农业生产及军事活动中,都可能有低温冲击伤的发生。目前,对冲击伤的损伤机制、病理生理等方面的研究已有大量文献报道[12-16],但对高寒环境暴露后冲击伤的研究较少。

由于冲击伤致伤原因的特殊性,临床中很少出现大量高寒肠道冲击伤患者,加之冲击伤具有外轻内重、伤情变化快、诊断和救治困难等特点,故而临床上难以对其进行深入细致的研究[17-19]。因此,有必要在实验室建立稳定可靠的、不同伤情程度的低温环境肠道冲击伤动物模型,用于深入研究肠道冲击伤的病理生理机制、组织修复和康复方法。

本课题组利用冷柜模拟寒冷环境,利用BST-Ⅰ型生物激波管选用3种不同强度驱动压,在肺防护条件下(排除肺损伤原因导致死亡)造成大鼠肠道冲击伤,在实验室条件下对高寒环境暴露后肠道冲击伤特点进行研究。通过实验发现,低温暴露后,大鼠很快出现皮肤苍白、安静,抱团,活动减少、排尿增加等生理反应,叠加腹部冲击波损伤后,大鼠死亡率明显增加,且较常温冲击伤,损伤程度更重、死亡率更高。其肠道伤情特点除肠黏膜血肿、肠系膜出血、撕脱伤等,更易出现肠道水肿,病理检测可见肠绒毛下间隙扩大,上皮抬高与固有膜脱离、毛细血管充血,黏膜固有层崩解、溃疡形成等。肠黏膜损伤及缺血指标血清DAO、I-FABP,以及血清炎症因子TNF-α及IL-6均在损伤后短时间内明显升高。

动物暴露于冲击波后会出现即刻的心动过缓、低血压和呼吸暂停三联征,此三联征相互作用可导致恶性循环,引起组织缺氧[20]。而高寒环境下肠道微循环抑制,肠黏膜血流减少,叠加冲击伤更易导致肠黏膜出现机械性损伤及缺血再灌注损伤,加剧组织缺氧[21],导致炎症级联反应增强,促进炎症因子的释放,进一步加重肠黏膜损伤,表现出肠黏膜水肿及血肿。

综上所述,本研究对高寒环境不同强度大鼠肠道冲击伤伤情进行了比较,对损伤机制进行了初步探讨,实验结果提示,对高寒环境下冲击伤伤员的救治中要特别注意肠道屏障功能的监测和保护,血清I-FABP、DAO、TNF-α等血清学指标有助于对损伤严重程度的判断。这些实验结果对下一步的研究及高寒环境肠道冲击伤伤员的救治具有一定参考意义。

利益冲突 本文所有作者声明不存在利益冲突

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