ORIGINAL RESEARCH

A 12-month prospective study of intra-abdominal hypertension and abdominal compartment syndrome incidence and outcomes at a tertiary hospital in Nigeria

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Abstract

Background

Peritoneal sepsis is a life-threatening emergency, more so in the low- and middle-income countries (LMICs) where immediate hospital presentation for much needed urgent surgical care is the exception rather than the norm. Continued research into the multifactorial aetiopathogenesis responsible for the high level of morbidity and mortality is necessary. We aimed to determine the incidence of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) in patients presenting with generalized peritonitis in a tertiary hospital in Nigeria.

Methods

We conducted a prospective study involving recruitment of consenting patients managed for generalized peritonitis over 12 months.

Results

Fifty-seven consenting and appropriate patients were recruited over the course of the study and managed as per study protocol. The duration of symptoms ranged between 11 hours and 7 days. All patients had varying degrees of IAH and ACS at presentation with generalized peritonitis. A laparotomy with definitive surgery was done in 51 patients (89%), with 6 patients (11%) having only a bedside peritoneal drain inserted for decompression.

Significant improvement of the respiratory rate ($P<0.001$), oxygen saturation ($P=0.041$), and urinary output ($P=0.021$) only occurred after decompression by laparotomy or tube drainage. The consecutive mean ± standard deviation (SD) intra-abdominal pressures measured, respectively, at presentation, immediately postsurgery, then postoperatively at 6 h, 24 h, and 72 h reflected significant improvement at each point (respectively in cmH_2O: 11.4 ± 6.03, 6.58 ± 5.58, 5.78 ± 3.29, 4.73 ± 2.86, 6.72 ± 5.18; $P<0.001$).

Conclusions

IAH and ACS are not uncommon in our setting, and ACS at presentation is a significant predictor of mortality in patients with peritoneal sepsis. Surgical decompression invariably leads to an improvement in all clinical variables investigated.

Keywords: intra-abdominal hypertension, abdominal compartment syndrome, peritonitis, laparotomy, percutaneous catheter decompression, Nigeria

Introduction

The morbidity associated with secondary peritonitis often leads to prolonged hospital admission, sepsis, and multi-organ failure; death occurs in 20% to 60% of cases.[1–5] Intra-abdominal hypertension (IAH) is a sustained or repeated pathologic elevation of intra-abdominal pressure (IAP) greater than 12 mmHg. Abdominal compartment syndrome (ACS), so named by Kron et al.[6] is sustained IAP greater than 20 mmHg, with or without an abdominal perfusion pressure (APP) less than 60 mmHg, that is associated with new organ dysfunction or failure.[7,8] The effect of IAH and ACS on cardiovascular, renal, pulmonary, splanchnic, abdominal wall, and nervous tissues have been elucidated over the last century and a half.[9–14] IAH and ACS refer to multisystem

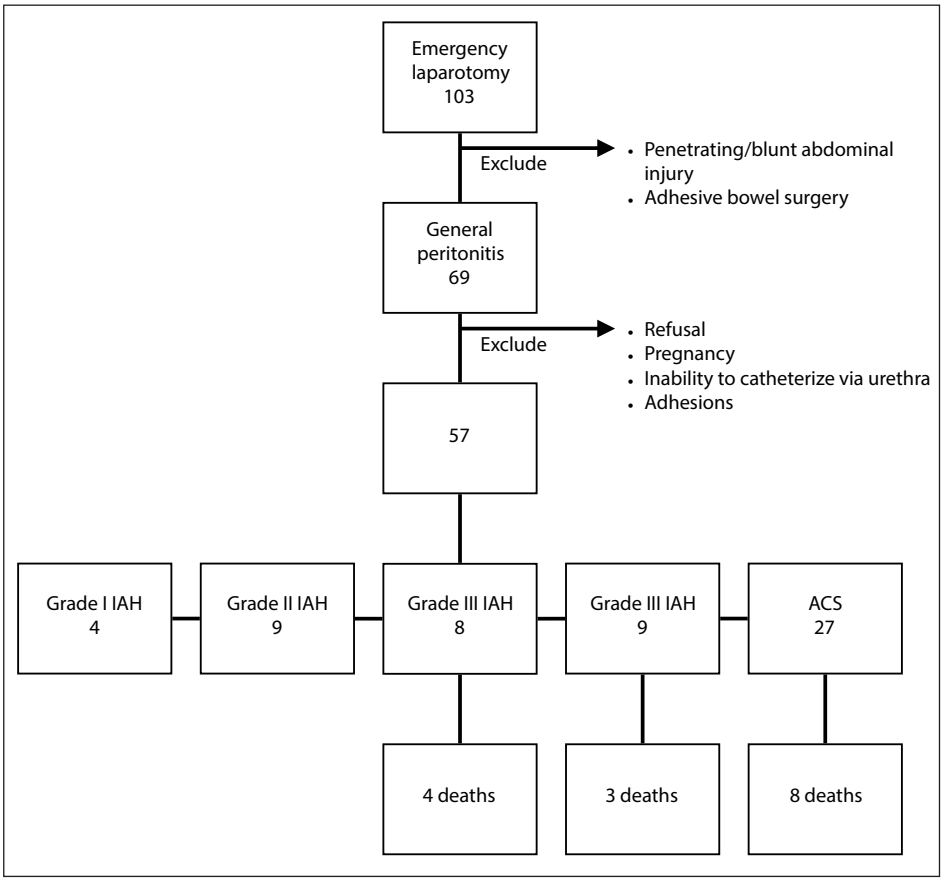

Figure 1. Study recruitment and findings summary
IAH = intra-abdominal hypertension
ACS = abdominal compartment syndrome

pathological entities resulting from IAP elevation regardless of aetiology.[15] The clinical spectrum should not be allowed to run its course, and while many of the potential early management options for IAH are medical interventions (haemodialysis, neuromuscular blocking agents, diuretics, prokinetic agents) that vary depending on aetiology, early surgical decompressive laparotomy is the gold standard for ACS.[16–21] The transient, though physiologically mediated, IAP elevations that occur during cough, heavy object lifting, and Valsalva manoeuvre cannot be sustained or tolerated for long periods.[22,23]

Peritoneal sepsis from a gastrointestinal pathology serves as the first pathologic insult, while the resulting acute increase in IAP greater than 15 mmHg causes an impairment of intestinal oxygenation and, at IAPs greater than 40 mmHg, mesenteric blood flow actually reduces by 60% to 70%,[24] resulting in impaired gut mucosal barrier function and leading to bacterial endotoxin translocation and sepsis as an escalation of the first insult.[25] The multisystemic effect of the worsening IAP on the adjoining organ systems (thoracic, kidney, venous) serves as the second pathologic insult in patients with secondary peritonitis.[6] The occurrence of IAH and ACS has been thoroughly investigated in mostly homogeneous patients groups—mainly trauma, vascular, and intensive care unit (ICU) patients[26,27] with less attention given to secondary peritonitis either at presentation or postoperatively. More important is the absence of any review in sub-Saharan Africa on this self-perpetuating, multisystem, and multifactorial syndrome. The clinical applications of relevant diagnostic and therapeutic advancements are well documented in relation to the developed world, while research and publications from sub-Saharan Africa are lacking.[26,27] This study aimed to determine: (1) the incidence of IAH and ACS, (2) if IAH/ACS is an independent predictor of morbidity and mortality, and (3) the outcomes of management of secondary peritonitis patients with IAH or ACS.

Methods

We conducted a prospective study in the Division of Gastrointestinal Surgery, Department of Surgery, University College Hospital (UCH), Ibadan, Nigeria. Patients with a clinical diagnosis of generalized peritonitis secondary to a gastrointestinal organ pathology were enrolled and followed over a 12-month period from April 2015 to March 2016. Consenting patients over the age of 18 undergoing emergency laparotomy for peritoneal sepsis were eligible. We excluded patients who refused to provide consent, and those who were obese, pregnant, diagnosed with adhesive intestinal obstruction, or for whom urethral catheterization failed. The protocol involved recruitment at presentation. Ethical approval was obtained from the state institutional review board under the Ministry of Health (MOH).

Sociodemographic characteristics, along with vital signs; clinical diagnosis; biochemical, haematological and radiological workup results; and intraoperative and postoperative details were recorded. The surgical intervention, presence or absence of high dependency unit (HDU) or ICU care, and outcomes (morbidity and mortality) were all recorded. We also recorded IAPs at presentation, immediately postoperatively (0 h), and then at 6 h, 24 h, and 72 h postsurgery.

Intra-abdominal pressure measurement

With the patient in the supine position, IAP was indirectly measured, as described by Kron et al.,[6] by passing a 16 Fr Foley catheter into the urinary bladder; further to emptying the bladder, we instilled 50 mL of sterile normal saline and clamped the collecting bag, after which a saline manometer was connected. The pubic symphysis served as the ref-

Table 1. Patient demographic and clinical characteristics

Characterstic	n (%)
Age (years)	
18–40	30 (53)
41–65	17 (30)
≥ 66	10 (18)
Gender	
Male	38 (67)
Female	19 (33)
Clinical diagnosis (intra-abdominal site of origin)	
Gastroduodenal	11 (19)
Small bowel	15 (26)
Appendix	18 (32)
Colon	9 (16)
Biliary	4 (7)
Comorbidities	
Hypertension	14 (25)
Diabetes mellitus	11 (19)
Asthma	2 (4)
Anaesthesia	
General anaesthesia	44 (77)
Local anaesthesia	13 (23)
Subarachnoid block	0
Surgeon	
Senior registrar	22 (39)
Consultant	35 (61)
Surgical Intervention	
Laparotomy and definitive surgery	51 (89)
Peritoneal (tube) decompression only	6 (11)
Postoperative care	
High-dependency unit	28 (49)
Intensive care unit	16 (28)
Ward	13 (23)
Outcome	
Dead	15 (26)
Alive	42 (74)
Postoperative morbidity	
Anastomotic leak/enterocutaneous fistula	3 (5)
Acute respiratory distress syndrome	6 (11)
Burst abdomen	6 (11)
Wound infection	41 (72)
Abscess – organ space	6 (11)

erence point, while pressure was measured initially in cmH_2O at the end of expiration before conversion to mmHg. We used the World Society of the Abdominal Compartment Syndrome (WSACS) grading system for IAH,[8] as follows: Grade I: 12–15 mmHg, Grade II: 16–20 mmHg, Grade III: 21–25 mmHg, and Grade IV: ≥25 mmHg. ACS was deemed to have occurred when IAH and 1 newly diagnosed organ system dysfunction occurred. Statistical analysis was performed using SPSS version 21 (IBM Inc., Armonk, NY, USA). Categorical and continuous variables are presented as numbers and percentages and mean ± standard deviation (SD), respectively. Significance testing for variables obtained before and after operative intervention was done using the paired sample t-test, and trends in IAP were compared using analysis of variance (ANOVA). Statistical significance was set at $P \leq 0.05$.

Results

Over the 1-year study period, 103 emergency laparotomies were performed in the gastrointestinal surgery division at UCH, Ibadan. Of these 69 (67%) were for peritoneal sepsis and 12 (12%) were excluded due to reasons of refusal, pregnancy, adhesive intestinal obstruction, and inability to catheterize via the urethra. A total of 57 patients (55%) were consecutively recruited. The mean age ± SD was 48 ± 12 years. Figure 1 shows the findings of the study, and Table 1 shows the sociodemographic and clinical characteristics of the patients. Comorbidities noted were hypertension,

Table 2. Incidence and outcomes of intra-abdmonial hypertension (IAH) and abdominal compartment syndrome (ACS) among patients with peritonitis

Severity	n (%)	Deaths n
Grade 1 IAH	4 (7)	0
Grade 2 IAH	9 (16)	0
Grade 3 IAH	8 (14)	4
Grade 4 IAH	9 (16)	3
ACS	27 (47)	8

diabetes mellitus, and asthma in, respectively, 14 ± 25%, 11 ± 19%, and 2 ± 4% of patients. All patients had signs of peritonitis (guarding, rebound tenderness, and board-like rigidity, along with at least 2 systemic inflammatory response syndrome (SIRS) criteria. The duration of symptoms ranged between 11 hours and 7 days. Preoperative resuscitation before surgical intervention was administered for between 1 and 8 days. Fifty-one patients (89%) underwent laparotomy with definitive surgery, and 6 patients (11%) had a bed-side peritoneal drain inserted for decompression instead of laparotomy. A mean of 1400 mL (range 900–5300 mL) of pus was drained from the patients. The diagnoses and interventions are listed in Table 1. The majority of patients (75%) were nursed in the HDU or ICU because of the need for vasoactive support or elective ventilation.

All patients recruited had at least Grade I IAH at presentation. Table 2 highlights the incidence and outcomes of IAH and ACS in this patient group. Postoperative morbidity occurred in 46 patients (81%) (Table 1). Nine patients (16%) had to be re-explored on account of burst abdomen and organ space infection (abscess). Blood pressures, pulse rates, pulse volumes, and body temperatures improved following resuscitation, while the respiratory rates ($P < 0.001$), oxygen saturations ($P < 0.041$), and urine outputs ($P < 0.021$) improved significantly only after surgical decompression by laparotomy or percutaneous tube drainage. The mean duration of hospital stay was 11 days (range 8–25 days). The mean ± SD IAPs measured, respectively, at presentation, immediately postsurgery, then postoperatively at 6 h, 24 h, and 72 h reflected significant improvement with each consecutive measurement: 11.4 ± 6.03 cmH_2O, 6.58 ± 5.58 cmH_2O, 5.78 ± 3.29 cmH_2O, 4.73 ± 2.86 cmH_2O, 6.72 ± 5.18 cmH_2O; $P < 0.001$).

Discussion

Our study was on a homogeneous cohort with IAH or ACS occurring secondary to pathology in the abdominopelvic region and requiring surgical or interventional radiological management.[28] Notably, most studies on IAH and ACS analyse either trauma or ICU patients,[29] and the presence of IAH is associated with an 11-fold rise in mortality.[30]

The male-to-female ratio in our study closely mirrored studies from other parts of the world,[29,31,32] while the mean age of 48 ± 12 years was similar to the earlier report by Cheatham et al.[33] but distinctly higher than values reported by others.[29,31,32] Peritonitis as a consequence of (blunt) trauma represented only 9% of patients recruited, which contradicted the 17–68% reported in other reviews.[29,33] The mechanism of peritonitis in these cases progressed from bowel devascularization, to indolent ischaemia, and subsequent necrosis or perforation; this differs from the classical characterization of haematoperitoneum and shock seen in the study by Meldrum et al.[32]

The prevalence of IAH in our sample was 100% at admission, which is higher than the value reported by Khan et al.,[29] while the 47% incidence of ACS is also higher than other reports by various authors.[8] Subtle differences in the population of patients used, including trauma, ICU uptake, peritoneal sepsis presence, and cutoff values have all been implicated in the wide range of values reported.[8] It is, however, clear when using the consensus definition of ACS that incidence will be higher in the context of peritoneal sepsis because organ dysfunction may not be solely attributed to an increasing peritoneal pressure but also from the sepsis to multiple organ dysfunction syndrome (MODS) pathway. The organ dysfunction can be explained by either sepsis or MODS potentiating IAH or vice versa to cause the progression to ACS. The increase in IAP seen in peritonitis is a result of an accumulation of gas, pus, faeces, or peritoneal and bowel oedema. The mean ± SD IAP at presentation of 11.4 ± 6.03 is lower than quoted values from other studies, while the post-surgical decompression value closely matches other reported values.[29,30,32] The lower preintervention IAH values seen in our cohort support our argument that the organ dysfunction noted in our patient group (which necessitated classifying those patients as ACS) may not be mainly due to the peritoneal pressure increase but rather to sepsis. Of note, however, is the large volumes of pus, faeces, or air drained from the peritoneal cavity at decompressive laparotomy.

The resolution of oliguria, uraemia, and raised creatinine after resuscitation and decompression suggest that baseline data can be assumed to have been normal before onset of illness. Abdominal wall closure was done primarily. Despite the seeming lack of abdominal wall tension, as evidenced by the tight-looking sutures, there was no IAH observed in the immediate postoperative period. This may be attributed to the perioperative manoeuvres of bowel decompression using the nasogastric tube, to urethral catheterization, or to the use of the Savage decompressor, along with the previously mentioned medical options and nonreversal of neuromuscular function when postoperative elective ventilation was required. Reported cases of immediate postoperative IAH are more common in trauma, where damage control and pressure packing are employed with or without the use of (modified) Bogota bags. Although no IAH was noted postsurgery, some patients developed organ system failure; we believe that this is a representative group of patients in whom the aetiopathogenesis of organ dysfunction is more sepsis-based than IAH-based. They formed a high proportion of patients who died before reaching the 72 h mark after surgical intervention.

Our analysis showed that decompressive laparotomy in our patients resulted in improved renal, respiratory, cardio-

vascular indices. The respiratory system—represented clinically by the respiratory rate, oxygen saturation, and the need for ventilator support (or lack thereof)—showed statistically significant improvement. The renal and cardiovascular systems showed improvement, though this was not statistically significant. This finding was contrary to the findings reported by Khan et al.[29] and Ma et al.,[34] who demonstrated a statistically significant improvement in renal function. Our mean pre- and post-decompression urine outputs were similar to those reported by Sugrue et al.[30]

Although found predominantly in a mixed ICU patient population, the most common risk factors found in a systemic review and meta-analysis included large-volume crystalloid resuscitation, patient respiratory state, sepsis, and the presence of shock or hypotension (systolic blood pressure < 86 mmHg).[26] This was the norm rather than the exception in our cohort, as they were all present to varying degrees in our patients. Surgical intervention within 75 minutes of presentation has also been reported as a key predictor[35]; but due to the resuscitation requirement in established cases of peritoneal sepsis, our mean time from presentation to surgery was 3 days (range 1–8). The extended duration of resuscitation was due to the need to optimize the patients that presented with septic shock, acute kidney injury, or other states of severe decompensation.

Competing interests

All authors declare that they have no competing interests related to this work.

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